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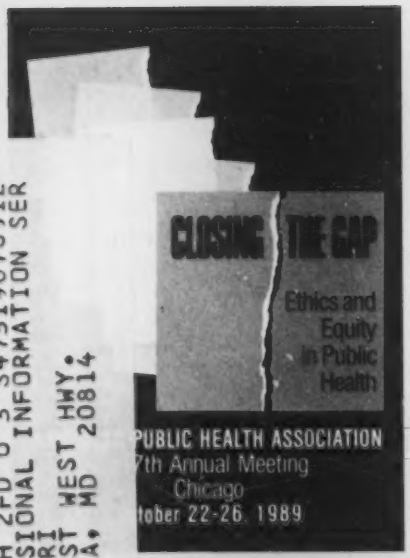

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Quality of Death Certificates in Valencia, Spain

FERNANDO G. BENAVIDES, MD, PHD, FRANCISCO BOLUMAR, MD, PHD, AND RAFAEL PERIS, MD, PHD

Abstract: Certificates of 1,454 deaths occurring over 11 months were retrieved from the Civil Register in Valencia, Spain. Relevant medical information was systematically gathered from hospital records, questionnaires, and Coroner reports. We compared the underlying cause of death (UCD) from the original death certificate, and a reference cause of death (RCD) determined by a panel of experts based on all available information. Overall, 80.2 percent of the certificates were concordant for disease category, but there was a great disparity among some specific disease categories. (*Am J Public Health* 1989; 79:1352–1354.)

Introduction

The quality of mortality statistics has been studied frequently.[1–6]

While mortality statistics are commonly used both for research and administrative purposes in Spain, their reliability has not yet been studied in full detail. This is an important omission; the first studies in this field were carried out in other countries as early as the 1940s.[7–9]

To date, studies of the quality of the registered causes of death have been based on death certificates selected either because stated causes were specific diseases or because of the availability of information required to test the concordance of the death certificate.[10–14] In order to minimize such selection biases, the present investigation was population-based.

Methods

All death certificates for the 255,866 residents in the catchment area of the Teaching Hospital in the region of Valencia, Spain from February–December 1984 were obtained from the Civil Register. For the resulting 1,454 certifications, we systematically reviewed autopsy findings, questionnaires addressed to the certifiers, and hospital records.

The monthly search for this information started in the Teaching Hospital. If hospital records corresponding to each death were available in the first week of the search, and the last inpatient summary had been completed within the three months prior to death, the search for clinical information was concluded.

When results of first step were negative, a questionnaire (based on work by Moriyama, *et al.*[15]) requesting clinical information about the death was sent to the certifier in the first week of the month following the death, and a list of all such deaths was sent to the Hospital Records departments of all the other hospitals in the city.

Finally, records were obtained from the Coroner's Office for those deaths which had been certified by a Coroner.

All the available information was reviewed separately by two specialists in Internal Medicine, who were not conversant with the information recorded in the original death certificate. Kappa index of agreement between the two reviewers was 0.78 (confidence intervals 0.76, 0.81). When internists opinions did not coincide, a third specialist was called in to review the information and thus provide a conclusive decision. All three specialists had been trained previously in certifying procedures by experienced physicians from the Office of Mortality Statistics.

Applying the rules of the ICD-9th revision,[16] professional coders selected the underlying cause of death (UCD) from each original certificate and the reference cause of death (RCD) from each standard certificate produced by the internists.

Using two by two tables for each cause and for each group of causes, the Detection Rate (DR),* and the Confirmation Rate (CR),** were computed working at a three-digit level of ICD-9th codes.

From the initial 1,454 certificates, clinical information was obtained for 1,176 deaths; in the remaining 278 deaths neither clinical records were available nor were questionnaires answered. Out of the 1,176 deaths, 108 (9.2 percent) were not included in the study because the medical reviewers considered the clinical information inadequate to test the concordance. Therefore, the final number of deaths studied was 1,068 (73.5 percent of the initial sample).

Results

In 50.9 percent of the 1,068 deaths, clinical information was obtained only from hospital records, in 27.7 percent only from questionnaires, and in 11.9 percent from both sources. Coroners' reports provided the remaining 9.4 percent of the information, of which 1.9 percent had also hospital records. The total number of deaths with hospital records amounted to 692. In 40 cases, autopsies were carried out by hospital pathologists. Of the 692 deaths with information obtained through hospital records, 562 (81.2 percent) took place in hospitals.

According to the completed questionnaires, the duration of the professional relationship between the certifier and the deceased had lasted less than 24 hours in 29.2 percent of the cases. When deaths occurred in hospitals, this percentage increased to 53.7 percent; when deaths occurred at home, it decreased to 17.9 percent.

Agreement between Detection and Confirmation Rates did not vary by duration of professional relationship, by place

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Editor's Note: See also related editorial p 1349 this issue.

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*Detection Rate equals the number of concordant deaths according to a particular disease category divided by the number of standard death certificates from the same disease category.

**Confirmation Rate equals the number of concordant deaths according to a particular disease category divided by the number of original death certificates from the same disease category.

TABLE 1—Percentage Agreement (with 95% Confidence Intervals) of Detection and Confirmation Rates by Place of Death, Duration of Professional Relationship and Sources of Information

Place of death	N	Agreement Indexes (%)	95% CI
Home	426	80.5	76.7, 84.2
Hospital	642	80.1	77.0, 83.1
Duration of professional relationship			
>3 months	215	81.8	76.7, 86.9
<3 months	85	82.4	74.3, 95.5
<24 hours	124	81.5	74.6, 88.3
Sources of information			
Clinical records	692	79.1	76.1, 82.1
Questionnaires (only)	296	82.1	77.7, 86.5

of death, and by sources of information (Table 1).

The agreement indexes for all causes were 80.2 percent both for the detection and confirmation rates, as shown in Table 2.

The agreement indexes, DR and CR, for the major categories of causes of death can also be found in Table 2. They are highest for tumors (DR = 89.9 percent and CR = 95.3 percent) and circulatory diseases (DR = 87.6 percent and CR = 80.5 percent).

Agreement indexes for the rest of groups of causes were questionable, due to the small number of deaths on which they were based. Table 3 displays the matrix plotting underlying cause against reference cause.

As regards specific tumor sites, the highest indexes of agreement were those for lung cancer (DR = 84.6 percent and CR = 91.7 percent) and breast cancer (DR = 88 percent and CR = 95.7 percent). Conversely, liver cancer showed very low rates of agreement (DR = 53.8 percent and CR = 37.9 percent), and colon cancer showed indexes below 80 percent but of a very similar nature (DR = 75 percent and CR = 71.4 percent).*

*Data available on request to author.

Agreement indexes for circulatory diseases varied greatly. Hypertensive disease produced low indexes of agreement. Arteriosclerosis gave quite similar results, with even lower indexes (DR = 19.2 percent and CR = 21.7 percent). Acute myocardial infarction showed a DR of 75.5 percent and a CR of 53.7 percent and the intra craneal haemorrhage a DR of 84.2 percent and a CR of 55.2 percent. However, the overall agreement in this group was over 80 percent.*

Discussion

In these data, mortality statistics as a whole appear to be reasonably reliable: 80.2 percent of the certificates showed good concordance. This figure lies somewhere within the range of results obtained by Alderson[4] in the Oxford Record Linkage Study Area (61 percent of agreement) and by Faire[5] among Swedish twins born in 1901–25 (DR = 94.9 percent and CR = 95.6 percent) (our estimate from their figures).

When comparing our population based results with those of Kircher in Connecticut,[14] based on autopsies, we found his confirmation rates to be lower than ours for neoplasms, circulatory diseases, and respiratory diseases (85 percent versus 95.3 percent; 75 percent versus 85.5 percent and 31 percent versus 60.3 percent, respectively). These differences might be explained, at least to some extent, by the fact that when only autopsies are used to validate death certificates, a higher level of disagreement is to be expected as autopsies are carried out mainly on diagnoses that appear uncertain.

It is interesting to note that in our study 13 deaths initially certified as due to cancer were reclassified after reviewing the available clinical information, and 30 deaths initially assigned to groups other than tumors were then reclassified as due to cancer. This finding suggests that mortality from cancer was underreported by 6.1 percent, a figure that lies between the value of 5 percent found by Griffith in England and Wales[17] and the value of 10 percent observed by Engel in Georgia.[18]

Percy, *et al*,[6] estimate a DR and CR of 82.7 percent for tumors in the USA, a value close to ours as regards DR but lower in respect to CR. Our results are relatively close to those obtained by Faire[5] in Sweden with a DR of 98.2 percent and a CR of 99.5 percent.

TABLE 2—Agreement Indexes for the Underlying Causes of Death, ICD-9A Main Category

Disease Category	UCD(*) N	RCD(**) N	Agreements N	Detection Rate %	Detection Rate SD	Confirmation Rate %	Confirmation Rate SD
Infectious/parasitic	6	7	2	28.6	17.1	33.3	19.2
Neoplasm	279	298	268	89.9	1.8	95.3	1.3
Endocrine/metabolic	43	51	30	58.8	6.9	68.8	7.0
Blood organs	4	1	1	100.0	0.0	25.0	21.7
Mental disorders	7	3	2	66.7	27.2	28.6	17.1
Nervous system	13	8	6	75.0	15.3	46.2	13.8
Circulatory	430	395	346	87.6	1.7	80.5	1.9
Respiratory	78	85	47	55.3	5.4	60.3	5.5
Digestive	83	99	73	73.7	4.4	87.5	3.6
Genitourinary	29	28	14	50.0	9.5	48.3	9.3
Pregnancy	1	1	1	100.0	0.0	100.0	0.0
Skin	0	2	0	0.0	0.0	0.0	0.0
Musculoskeletal	1	2	1	50.0	35.4	100.0	0.0
Congenital anomalies	7	6	6	100.0	0.0	85.7	13.2
Perinatal	3	4	3	75.0	21.7	100.0	0.0
Ill-defined	14	1	0	0.0	0.0	0.0	0.0
External causes	70	79	59	74.7	4.9	84.3	4.4
Total	1068	1068	857	80.2	1.2	80.2	1.2

(*)UCD: Underlying Cause of Death
(**)RCD: Reference Cause of death.

TABLE 3—Agreement Matrix between Underlying Cause of Death and Reference Cause of Death (RCD)

Underlying Cause of Death	ICD-9[a]	Reference Cause of Death																	
		001 −139	140 −239	240 −279	280 −289	290 −319	320 −389	390 −459	460 −519	520 −579	580 −629	630 −676	680 −709	710 −739	740 −759	760 −779	780 −799	E800 −E999	Total U.C.D.
Infectious/parasitic	(001–139)	2	2							2									6
Neoplasm	(140–239)	1	266	1				3		5	2			1					279
Endocrine-metabolic	(240–279)		2	30				8		3									43
Blood organs	(280–289)		1		1			1		1									4
Mental disorders	(290–319)					2		4			1								7
Nervous system	(320–389)			1			6	4			1							1	13
Circulatory	(390–459)	1	14	15		1	1	346	26	9	7					1		9	430
Respiratory	(460–519)	1	4				1	12	47	4	1		1				1.	6	78
Digestive	(520–579)		4	2					3	73			1						83
Genitourinary	(580–629)		3	2				8			14							2	29
Pregnancy	(630–676)											1							1
Skin	(680–709)																		0
Musculoskeletal	(710–739)													1					1
Congenital anomalies	(740–759)							1							6				7
Perinatal	(760–779)															3			3
Ill-defined	(780–799)							4	7		1							2	14
External causes	(E800–E999)	2						4	2	2	1							59	70
Total RCD		7	296	51	1	3	8	395	85	99	28	1	2	2	6	4	1	79	1068

With respect to mortality due to cardiovascular diseases, our indexes lie between those obtained by Engel[18] (DR = 69.0; CR = 75.6) and those found by Faire[5] in Sweden (DR = 92.4; CR = 88.8).

It has been argued that concordance indexes might depend on the precision and completeness of the information used by the reviewers to establish the standard cause of death. In our case, the influence of what has been called reviewer bias does not seem to play an important role, as additional information obtained after the death usually comes from autopsies and the number of these in our area was dramatically low.

It has also been argued that reviewers might have access to clinical records to which medical certifiers did not have access. But this does not accord with the fact that in our study 81.2 percent of the deaths with clinical records available took place and were certified in hospitals. However, even if this were the case, it would lead to a real discrepancy since the reviewers would have been able to establish the cause of death more accurately.

On the contrary, if the medical certifiers had access, the discrepancies would be spurious. Nevertheless, 72.3 percent of the information used to test the degree of concordance came from hospital records and forensic reports and thus was equally available for both certifiers and reviewers.

Some information was obtained only from questionnaires. This presented the problem of the reviewing doctors not having access to any more information than that which was transcribed. However for 70.5 percent of the completed questionnaires, corresponding to deaths at home, there was presumably no more information than that which appeared on the questionnaires, as the systematic search for the clinical records of these cases was negative. However, the possibility that a crucial piece of information which might have been known to the medical certifier did not appear in the records, although remote, cannot be ruled out in this sort of study.

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Risk Reduction among Intravenous Drug Users in Amsterdam under the Influence of AIDS

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Abstract: We evaluated the impact of a needle/syringe exchange program among 263 drug users in the period December 1985 to April 1988. Participants in this study were asked about their risk-behavior at three different visits. We found no increase in the proportion injecting drugs or in the frequency of intravenous drug use. A strong decrease in borrowing and lending of used needles/syringes was found and this behavioral change was not dependent on learning human immunodeficiency virus (HIV) serostatus. Although use of the exchange program increased over time, reduction in needle sharing was mainly an effect of the study with only a limited impact of the exchange program. We conclude that ample provision of needles and syringes is an important starting point but in itself not enough to produce the necessary drastic change in risk behavior. Intensive counseling of the drug user is also needed. (*Am J Public Health* 1989; 79:1355–1357.)

Introduction

In the absence of an effective treatment or vaccine, control of infections with the human immunodeficiency virus (HIV) relies on efforts to reduce risk behavior. To control the spread of HIV among intravenous drug users (IVDUs) a prevention campaign was launched in Amsterdam, The Netherlands.[1]

Leaflets with information on HIV and its transmission are distributed and meetings are held for workers in the drug field and for drug users. As sharing of needles and syringes is considered to be the main transmission route among intravenous drug users,[2] a needle/syringe exchange program was organized as part of this campaign. The exchange program started on a small scale in 1984; by 1987 700,000 syringes and needles were provided through existing social agencies and treatment facilities.[3] To evaluate the impact of the campaign, a longitudinal study was undertaken. The objectives of this study are to determine whether drug users are able to change their risk behavior and if risk reduction occurred, whether this can be ascribed to the prevention campaign or to the effect of serological testing and counseling during the study.

Methods

Participants were recruited at six methadone outposts and the weekly sexually transmitted disease clinic for drug-using prostitutes. None of these facilities are drug free. As one of the aims of the study was to find out whether the syringe exchange program could lead to an increase in intravenous drug use, ex-intravenous drug users and non-intravenous drug users were also invited to participate.

Participation was voluntary and, after extensive information had been given, informed consent was obtained. A blood sample was taken and participants interviewed using a standard questionnaire which included questions concerning clinical symptoms, medical history, lifestyle, use of oral and intravenous drugs (methadone included), and prostitution. Participants were asked to return for a follow-up visit every four months. At each occasion, the same interview and a blood sample were taken. To encourage continued participation 25 Dutch guilders ($12 US) were paid per follow-up visit.

From December 1985 to March 1988, 569 drug users without HIV-related disease entered the study, approximately one-seventh of the drug users visiting the methadone posts and the special STD-clinic in that period; 263 (46 percent) returned for at least one follow-up visit before July 1988. No difference was found in age, gender, nationality, and history of prostitution between the 263 drug users who returned and the 306 who did not return for a follow-up visit. However, among those who returned, a higher percentage was infected with HIV (33.1 percent vs 19.0 percent, 95% CI of difference = 6.9, 21.3), had been staying in Amsterdam longer than one year (88.2 percent vs 78.4 percent, 95% CI of difference = 3.8, 15.8), and had a history of current IV drug use (71.9 percent vs 53.9 percent, 95% CI of difference = 10.2, 25.8).

We compared results of the interview regarding the intravenous drug use behavior in the six-month period preceding the intake and during the follow-up period.

Since participants did not return for follow-up regularly, only those with an interval of three to six months between the visits were selected for study. As a result, 233 participants remained whose second visit data could be analyzed. The mean period between the first and second visit was 127 days (SD ± 20). A sub-group of 165 participants made a third visit, enabling us to study the risk behavior over a longer period of time. The mean duration between the second and third visit was 123 days (SD ± 20). There was no difference in age, gender, duration of drug use, nationality, and HIV-prevalence between the first-, second- and third-visit groups.

To determine the effect of infection status on risk behavior, three different groups were distinguished among 140 current intravenous drug users: those participants who did not know their serostatus (17); those who knew that they were infected with HIV (39); and those who knew that they were uninfected (84). Forty-nine other current users were excluded because knowledge of serostatus was too close to their second visit or no information was available about the date of disclosure.

The average time between intake and disclosure of the serostatus was 32 days (SD ± 7), and 97 days (SD ± 19) between disclosure and second visit. Only data of the first and second visit were studied; at the third visit the number of persons available was too small.

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Recruitment into the study took place over a period of 27 months. Thus we could also study the risk behavior of current intravenous drug users at intake over time. We divided the 27-month intake period into four periods of equal length (207 days). The HIV antibody seroprevalence of the four intake-groups defined their risk category.

χ^2 test with Yates' correction and χ^2 for trend in proportions were used for testing group differences and behavioral changes. Log-linear modeling[4] was employed for testing differences in behavioral changes between subgroups and for differentiating between effects on behavior caused by the prevention campaign (time effect) or by the study itself (study effect). Serological tests used for HIV-ab testing were ELISA (enzyme-linked immunosorbent assay) and confirmation of positive specimens was performed by immunoblotting and competitive ELISA.[5]

Results

Of the 263 study participants, 140 were male and 123 female. The mean age was 29.1 years (SD 5.4). Seventy-one percent were Dutch, 20 percent German, and 9 percent another nationality. Two hundred and thirty-three of the 263 participants had a history of intravenous drug use at entry into the study; 189 of them used drugs intravenously at least once during the six months preceding intake (current intravenous drug users). Heroin in combination with cocaine was injected by 58 percent of the current users, 21 percent used heroin alone, 7 percent cocaine alone, and 5 percent amphetamine alone. At entry into the study, 87 (33 percent) of the 263 participants had antibodies to HIV (36 percent of the men and 30 percent of the women). None of those 87 HIV-infected had severe HIV-related disease (acquired immunodeficiency syndrome or AIDS-related complex).

The proportion of current intravenous drug users who obtained their needles and syringes exclusively through the exchange program increased from 31.2 percent at the first visit to 52.3 percent at the third visit (Table 1), although the proportion of all drug users reporting using drugs intravenously and the frequency of injecting did not differ over time. Borrowing and lending needles and syringes among current users decreased sharply over time. The behavioral change occurred in both men and women, and in all nationality groups.* Decrease in needle sharing occurred to the same extent regardless of knowledge of serostatus (Figure 1).

The borrowing behavior at intake of current intravenous drug users was comparable over time although those who entered the study later lent their needles/syringes less often to others than those who entered in the beginning (Table 2). The use of the exchange system and single syringe use increased over time. The HIV-prevalence of those entering the study decreased over time.

To investigate the relative importance of a study-related (follow-up) effect versus a time-related (prevention campaign) effect, the effects were log linearly modeled and tested.* The time variable was dichotomized at the midpoint of the intake period.

The reduction in borrowing was merely a study effect, being unrelated to time. The likelihood ratio statistics of lending were significant for both study and time effects indicating a strong study-related reduction in lending behavior independent from the mere time effect. The increase in the use of the exchange system appeared mainly a time-related effect. The increase of single needle/syringe use appeared to be both study-related and time-related. These data are also suggested by inspection of Tables 1 and 2.

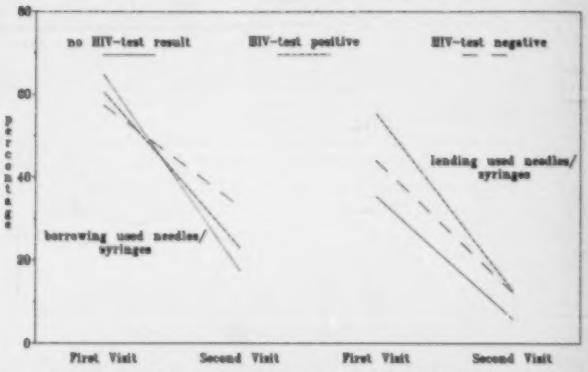

FIGURE 1—Current Intravenous Drug Users Engaged in Needle/Syringe Sharing by HIV-ab Test Result in Amsterdam, December 1985–July 1988

Discussion

The results of this follow-up study clearly show that IV drug users are able to reduce their sharing of needles/syringes, the main risk factor for contracting HIV in this group. Risk reduction among IV drug users has also been observed in New York City,[6] and in various retrospective studies.[7] A drawback of retrospective studies is that participants may tend to make the past look worse and the present better.

There was a sharp increase in the proportion of intravenous drug users in Amsterdam making exclusive use of the exchange program to obtain clean needles and syringes, apparently reflecting the effect of the prevention campaign.

We found no evidence that the non-intravenous drug users in our study group started intravenous use in spite of the availability of clean needles and syringes. Apparently the way in which drugs are used is rather stable.

Although an increasing number of intravenous drug users in Amsterdam obtain their needles/syringes through the exchange program, the impact of this prevention campaign on

TABLE 1—Current Intravenous Drug Users* Engaged in Specific Risk Behavior during Follow-up, Amsterdam, December 1985–July 1988

% of Current IVDU	First Visit n = 189	2nd Visit n = 156	3rd Visit n = 107	p-value**
Shooting daily	55	48	54	0.72
Shooting at home	79	80	80	0.75
Shooting alone	28	31	26	0.94
100% use of needle/syringe exchange program	31	40	52	0.0004
Single needle/syringe use	43	54	65	0.0003
Borrowing used needles/syringes ≥1×	56	26	16	10^{-6}
Lending used needles/syringes ≥1×	44	10	8	10^{-6}

*At the first visit 189/263 (72%) drug users were current intravenous drug users, second visit 156/233 (67%), third visit 107/165 (65%).
**χ^2 test for trend.

Data available on request to author.

TABLE 2—Risk Behavior of 189 Current Intravenous Drug Users at Intake (December 1985–March 1988) Divided by Intake Period

IVDU Behavior	Intake Period* I (n = 54)	II (n = 46)	III (n = 56)	IV (n = 33)	p-value**
	%	%	%	%	
Exclusive use of exchange system	13.0	10.9	44.6	66.7	10^{-6}
Borrowing	55.6	58.7	56.4	46.9	0.51
Lending	50.9	53.3	34.5	34.4	0.04
Single syringe use	38.9	34.8	42.9	60.6	0.06
HIV-ab seropositive	50.0	37.0	37.5	24.2	0.02

*Each intake-period covers 207 days: I. 12/04/85–06/28/86
II. 06/29/86–01/21/87
III. 01/22/87–08/16/87
IV. 08/17/87–03/11/88

**χ^2 test for trend.

risk behavior still leaves something to be desired. Borrowing used needles and syringes—at least once in the prior six months—was as often reported by those who entered our study at the end in 1986 as it was in 1988, even though this last intake group had a lower HIV-prevalence indicating they had been less at risk for HIV-infection. As we asked only whether the participants borrowed at least once, it may be that the frequency of borrowing diminished over time.

However, to be effective, risk reduction has to be total. Such a strong behavioral change was found only among the intravenous drug users who participated in the follow-up study.

The extent of behavioral change that we found during follow-up did not depend on learning one's infection status. In contrast, studies of homosexual men have found that seropositives changed their behavior more than seronegatives and untested comparison groups.[8] Apparently, among drug users counseling has a more important influence. From our results it seems that the needle/syringe exchange program and a testing program on a voluntary basis can be expected to have strong influences on risk behavior only when such programs are combined with intensive counseling.

We conclude that the Amsterdam AIDS prevention campaign did not increase the intravenous use of drugs, as is often feared, but did not automatically lead to complete change of risk behavior among intravenous drug users. To obtain such a maximum effect, intensive counseling of drug users seems indicated. Proposals have now been formulated to start with two special teams of counselors, one for drug users who participate in the methadone programs and one for those drug users who are not in contact with treatment programs.

We recognize that we have studied risk reduction among a selected group that may not represent all drug users in Amsterdam, but this is inevitable in all studies of drug users. In addition, the results are based on self-reporting. An objective measure of self-reported risk reduction is a reduced attack rate of HIV infection over time. As of July 1988, we have observed 10 seroconversions among 148 seronegative drug users with a history of IV drug use in the previous five years. The attack-rate (Kaplan-Meier) after a follow-up of one year was 7.3 percent (95% CI: 2.6, 12.0). The number of seroconversions in the follow-up period is still too small to enable us to do a reliable comparative study of the attack rate over time in relation to this process of risk reduction.

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Impact of the AIDS Epidemic on Morbidity and Mortality among Intravenous Drug Users in a New York City Methadone Maintenance Program

PETER A. SELWYN, MD, MPH, DIANA HARTEL, MPH, WILLIAM WASSERMAN, MPH, AND ERNEST DRUCKER, PHD

Abstract: To examine the impact of the AIDS epidemic on morbidity and mortality in a defined population of intravenous drug users, we analyzed overall and cause-specific death rates, AIDS incidence, and acute medical hospitalizations among patients in a long-term methadone maintenance program in New York City for the years 1984 through 1987 (midyear population for each year 828 to 891; demographic characteristics did not differ). The number of deaths while in treatment increased from 11 (13.3/1000) in 1984 to 39 (44.2/1000) in 1987. Deaths from AIDS increased from 3.6/1000 to 14.7/1000, deaths due to bacterial pneumonia/sepsis from 3.6/1000 to 13.6/1000; deaths from cirrhosis, drug overdose, trauma, and other causes remained relatively stable. AIDS incidence rose from six cases/1000 in 1984 to 20.4.1000 in 1987. Hospitalizations for AIDS, pneumonia, tuberculosis, and endocarditis/sepsis increased from 84.9/1000 in 1986 to 144.8/1000 in 1987.

These data suggest that the AIDS epidemic has had a profound effect on patterns of morbidity and mortality among intravenous drug users in this methadone program population. Drug treatment programs may be important sites for targeting clinical services for drug users with AIDS, although the increasing burden of AIDS-related disease will require expansion of existing funding and treatment resources. (*Am J Public Health* 1989; 79:1358–1362.)

Introduction

Intravenous drug users have historically been known to be at increased risk of death from drug overdose, alcoholism, trauma, and infectious causes.[1–4] Since the early 1980s, the acquired immunodeficiency syndrome (AIDS) has become a new and increasingly important cause of death among drug users, especially in New York City.[5–7] In addition, New York City surveillance data demonstrate a concurrent increase in pneumonia hospitalizations and deaths among narcotics addicts,[5,8,9] which may reflect additional consequences of human immunodeficiency virus (HIV) infection in this population.[5,7–11]

It has been estimated that there are between 1.1 and 1.3 million intravenous drug users in the United States, with over 200,000 in the New York City metropolitan area alone.[6,12] In New York State, there are currently over 35,000 patients enrolled in methadone maintenance treatment programs.* Historical patterns of morbidity and mortality among patients in methadone maintenance treatment are well described[13–19]; however, most of this literature predates the AIDS era and does not address the impact of the current epidemic of HIV infection and AIDS on such populations.

We examined AIDS incidence rates, overall and cause-specific mortality rates, and medical hospitalization patterns among a defined population of intravenous drug users in a long-term methadone maintenance treatment program in the Bronx, NY, from 1984 through 1987.

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*New York State Division of Substance Abuse Services: Census, Capacity, Utilization and Waiting List Report, November 1988.

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Methods

This study was conducted at the Drug Abuse Treatment Program of Montefiore Medical Center, Bronx, NY, which operates three outpatient methadone maintenance clinics for chronic opiate addicts. The treatment program, which serves a population of approximately 900 current and former intravenous drug users, provides on-site primary medical care for all patients, as well as methadone maintenance and support services.

In late 1983, centralized registries were established and maintained by program medical staff for recording all AIDS diagnoses and deaths among program patients; in late 1985, a log was established and reviewed weekly to monitor all hospitalizations occurring in the patient population. AIDS cases were identified both through ongoing patient contact with on-site medical services at the methadone program and through review of hospital records for suspected cases. Causes of death were identified and verified (by the first author) for all patients dying while in treatment, through a review of hospital records, death certificates, and medical examiners' reports for cases in which postmortem examinations were performed. Surveillance for hospitalizations was facilitated by inpatient follow-up of patients referred by medical staff for hospital admission, and by the requirement that hospital staff must contact clinic staff in order to verify methadone dosages for hospitalized patients.

Overall and cause-specific mortality rates per 1000 for methadone program patients dying while in treatment were calculated for each calendar year from 1984 through 1987, using the midyear population census as the denominator for each year. Person-time death rates were also calculated based on time spent enrolled in the methadone program and are expressed as deaths per 1000 person-years. Deaths were classified as due to AIDS in cases with either an ante- or postmortem AIDS diagnosis, in which AIDS was listed as a cause of death; deaths were ascribed to other causes only after AIDS diagnoses had been excluded through review of all available information. In order to evaluate the possible relation between HIV infection and causes of death other than AIDS itself (e.g., deaths from bacterial pneumonia or sepsis), we examined the HIV serologic status of the subgroup of deceased patients who had also been enrolled in a

seroprevalence and longitudinal study of HIV infection initiated in 1985 at the same clinic site.[20] This ongoing study, aspects of which have been previously described,[20–22] involved voluntary, confidential HIV antibody testing and clinical/research follow-up for a cumulative cohort of over 600 patients in the Montefiore program. The overall HIV seroprevalence level in the cohort has been approximately 40 percent.[20–22] As described elsewhere, seroprevalence study participants and non-participants did not differ with respect to standard demographic variables or the length of time in methadone treatment.[20–22] While the aim of the seroprevalence and longitudinal study has been to examine the natural history of HIV infection, that of the current analysis was to evaluate the impact of the epidemic of AIDS and HIV infection over time on a defined population in drug abuse treatment.

The incidence of AIDS in the entire study population was calculated for each year in the four-year period, with the occurrence of incident cases of AIDS expressed both as a proportion per 1000 persons and as a rate per 1000 person-years in treatment. In addition to incident AIDS cases arising out of the clinic population, yearly trends in the number of patients already diagnosed with AIDS and referred to the program for drug abuse treatment were monitored. To ensure uniformity of definitions and reporting, the original[23] and not the revised[24] Centers for Disease Control (CDC) AIDS surveillance definition was used for all four years of the study period as the standard for AIDS diagnoses.

Medical hospitalization logs, which were available only for the latter two full years of the study period, were reviewed for the years 1986 and 1987. Hospitalizations were classified by primary diagnosis into the most frequently occurring categories, and the frequencies of hospitalization for different diagnostic categories were compared for the two 12-month periods. Diagnoses were confirmed through review of hospital records. Hospitalizations for all AIDS-specific opportunistic infections were classified as due to AIDS. Elective admissions for inpatient alcohol or drug detoxification were excluded from this analysis. Hospitalization events by year were calculated for each year with the total number of patients enrolled in the program each year as the denominator.

As noted above for the determination of AIDS incidence and death rates, only those events are included which occurred while patients were enrolled in the methadone program; the analysis does not reflect any additional outcomes which may have occurred among patients leaving the program during the study period. Estimates should therefore be considered minimum estimates. The midyear patient population was chosen as the denominator for incidence calculations for each year since there was movement in and out of the program but the number of persons entering and leaving treatment was balanced for each year of the study period. An examination of time spent by patients in the program each year indicated that person-time did not differ between years, with individuals remaining in the program for a mean of approximately 300 days each year. Analyses of changes in incidence rates over the four-year study period were based on point and interval estimates of rate differences.[25] In addition, trends over the four years in AIDS incidence and overall and cause-specific mortality rates were examined.

Results

From January 1, 1984 through December 31, 1987, a total of 1,598 patients were treated: 59 percent male, 20 percent White, 27 percent Black, 52 percent Hispanic (primarily Puerto Rican), and 1 percent classified as "other." Racial and gender distributions did not differ by year for any of the four years. The median age of the entire patient population as of January 1, 1984, was 32 years, with the central three-fourths of all patients each year between the ages of 30 and 45. The overall age distribution (by year of birth) was fairly constant from year to year. The midyear patient census for each of the four years ranged from 828 to 891 (Table 1). Of the 1,598 patients treated in the program during the study period, 844 (53 percent) were enrolled prior to 1984, of whom 416 (49 percent) remained continuously enrolled through the end of 1987. Seven hundred and six patients were newly admitted during the four years, and 48 patients were readmitted after having left treatment before 1984. The number of patients discharged from treatment each year ranged from 162 to 197; discharged patients did not differ by year with respect to race, gender, or age, nor was there a statistically significant trend in drop-out rates during the study period.

Death Rates

A total of 94 patients died while in treatment during the study period (Table 1). The yearly number of deaths increased steadily from 1984 through 1987, even though the midyear population each year remained stable. The proportion dying (based on the mid-year population size) increased from 13.3/1000 in 1984 to 44.2/1000 in 1987. By person-time analysis, there were 12.9 deaths/1000 person-years in 1984, compared with 44.0/1000 person-years in 1987.

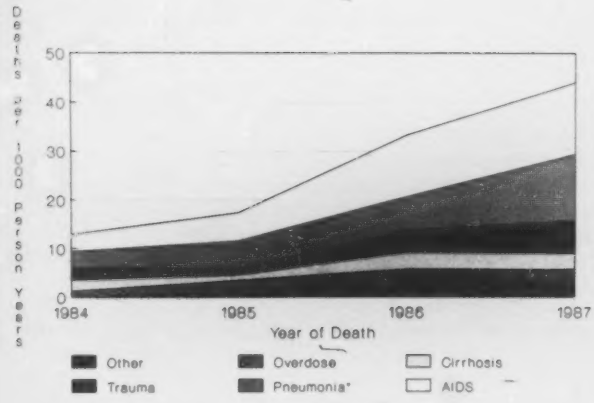
Causes of Death

The crude mortality rate from AIDS in 1984 was 3.6 per 1000 patients. This rate increased steadily through the study period to 14.7 per 1000 in 1987. Deaths from pneumonia, endocarditis, and bacterial sepsis combined—in patients without any diagnosis of AIDS—also rose from 3.6 per 1000 in 1984 to 13.6 per 1000 in 1987. Smaller or minimal increases were seen for deaths from cirrhosis, drug overdose, trauma, and other causes, which were less frequent causes of death. Cause-specific death rates (person-time) among methadone program patients are graphically displayed in Figure 1. The number of deaths due to AIDS rose from 3.5/1000 person-years in 1984 to 14.7/1000 person-years in 1987 (difference 11.2/1000, 95% CI: 2.2, 20.0). Similarly, there was an increase in death rates from pneumonia, endocarditis, and bacterial sepsis, which together rose from 3.5 to 13.5/1000 person-years from the first to the fourth year of the study period (difference = 10.0, 95% CI: 1.4, 18.6). Deaths due to alcoholism/cirrhosis and drug overdose remained relatively constant between 1984 and 1987 (3.5 to 5.7/1000 person-years); death rates from trauma, including homicide, suicide, and vehicular trauma, showed a slight increase during the

TABLE 1—Deaths by Year among Montefiore Methadone Program Patients, New York City, 1984–87

	1984	1985	1986	1987
Midyear Census	828	875	891	882
Person-years	850	864	873	886
Mean Days Observed	296	300	294	300
Number of Deaths	11	15	29	39
Deaths per 1000	13.3	17.1	32.5	44.2
Deaths/1000 person-yrs	12.9	17.4	33.2	44.0*

*Rate difference 1987–1984 = 31.1 (95% confidence interval: 15.3, 46.9).



* Includes endocarditis and sepsis

FIGURE 1—Causes of Death in Montefiore Methadone Program Patients, 1984–87

study period (2.4 to 6.8/1000 person-years). The final category, which also showed a slight increase (0 to 3.4/1000 person-years), was defined as "other," and included deaths from renal failure, pulmonary embolism, bleeding diatheses, stroke, and neoplasms.

Analysis of the HIV serologic status of the subgroup of deceased patients who had been enrolled in the HIV seroprevalence/longitudinal study, as described above, indicated that of nine patients with known serologic status dying from bacterial pneumonia, endocarditis, or sepsis, all were HIV seropositive. In contrast, of 12 patients with known HIV serologic status dying from cirrhosis, overdose, or trauma, 4 (33 percent) were seropositive and 8 (67 percent) seronegative. Of two patients with known serologic status dying of causes classified as "other," both were HIV seropositive. Decedents enrolled in the seroprevalence/longitudinal study did not differ from those in the larger clinic population demographically or by the length of time in methadone treatment.

Incidence of AIDS

The number of incident cases of AIDS newly diagnosed each year increased from five in 1984 to 18 in 1987 (Table 2). The crude annual incidence of AIDS rose from 6.0/1000 in 1984 to 20.4/1000 in 1987. The rate by person-time increased from 5.9/1000 person-years to 20.5/1000 person-years in 1987. In addition to incident cases of AIDS occurring in the treatment program population, 16 additional patients already diagnosed with AIDS were enrolled for treatment during the study period, with a steady increase in such referrals from the first to the fourth year. These patients are excluded from the calculation of AIDS incidence but included in the calculations of hospitalizations and deaths over the four years.

TABLE 2—AIDS Cases among Montefiore Methadone Program Patients, 1984–87

	1984	1985	1986	1987
Midyear Census	828	875	891	882
Person-years	848	860	868	877
Incident cases of AIDS	5	8	11	18
AIDS cases per 1000	6.0	9.1	12.3	20.4
AIDS cases/1000 person-yrs	5.9	9.3	12.7	20.5*
Referred to methadone program after AIDS diagnosis	1	3	5	7

*Incidence rate difference for 1987–1984 = 14.6 (95% confidence interval: 3.8, 25.4).

Hospitalizations

Table 3 presents the frequency of hospitalizations occurring among the Montefiore program patients for different indications during the successive 12-month periods of 1986 and 1987. In 1986, there were 219 hospitalizations, or 202 per 1000 patients; in 1987 there were 269 hospitalizations, or 250 per 1000. A net increase of 71 percent in the frequency of hospitalizations from 1986 to 1987 was observed for AIDS, pneumonia, tuberculosis, and endocarditis/sepsis, which collectively increased from 85 to 145 hospitalizations per 1000 patients over the two years. All other categories showed either minimal increase, no increase, or decrease between the two years.

Discussion

These data document a dramatic threefold increase in overall mortality rates and AIDS incidence and mortality rates from 1984 through 1987, among intravenous drug users in a New York City methadone maintenance treatment program. The parallel increase in deaths from bacterial infections, in patients not diagnosed with AIDS, is consistent with New York City mortality data from the same time period which suggest that such deaths are indeed HIV-related and must be considered in any assessment of the impact of the AIDS epidemic among drug users.[5,7–9] It is noteworthy that among the subgroup of deceased patients with known HIV serologic status, all the bacterial infection deaths occurred in HIV seropositives, compared with only one-third of the deaths due to cirrhosis, overdose, or trauma. These findings are supported by additional data from our prospective studies of HIV infection in the same methadone program population, in which we have observed both a fivefold increased incidence of bacterial pneumonia and an increased death rate from bacterial pneumonia and sepsis in HIV seropositive

TABLE 3—Medical Hospitalizations among Montefiore Methadone Program Patients, 1986–87

Primary Diagnosis	1986 Hospitalizations Number n	1986 Hospitalizations per 1000*	1987 Hospitalizations Number n	1987 Hospitalizations per 1000*	Percent Change +/−
AIDS	31	28.6	43	39.9	+40
Pneumonia	50	46.1	87	80.8	+75
Tuberculosis	2	1.9	8	7.4	+302
Endocarditis/sepsis	9	8.3	18	16.7	+101
Suspected endocarditis	6	5.5	6	5.6	+1
Cellulitis/abscess	19	17.5	24	22.3	+27
Trauma	26	24.0	17	15.8	−34
Cirrhosis	8	7.4	7	6.5	−12
Drug overdose	18	16.6	9	8.4	−50
Seizures/stroke	11	10.1	12	11.1	+10
Gastrointestinal bleeding	5	4.6	4	3.7	−20
Cardiovascular/circulatory	10	9.2	6	5.6	−40
Pancreatitis	4	3.7	6	5.6	+51
Other**	20	14.5	22	20.4	+11
Total hospitalizations	219	202.0/1000	269	249.8/1000	+24%

*Total number treated in methadone program in 1986 = 1084 patients, in 1987 = 1077 patients.

**All categories with fewer than five admissions for both years, including: osteomyelitis, urinary tract infection, leg ulcers, diabetes mellitus, cancer, biliary tract disease, asthma, gastroenteritis.

versus seronegative intravenous drug users within the cohort.[21,26]

Previous studies of mortality patterns and rates among intravenous drug users consistently suggested both that death rates among addicts were higher than among the general population and that two-thirds or more of drug user deaths were specifically related to drug overdose, violence or trauma, or alcoholism.[1–4,15–19,27–33] Observed annual death rates in such studies, most of which predated the AIDS era, have ranged between approximately 8 and 16 per 1000.[4,17,18,27,30] Several studies of mortality rates among patients in methadone maintenance programs in the 1970s indicated similar yearly rates, all under 16 per 1000.[16–19] Of note is the fact that the pattern of deaths for the year 1984 in our population was comparable to these historical observations from the pre-AIDS era: the overall level in 1984 was 13.3 per 1000, and close to half of the deaths were due to cirrhosis, overdose, or trauma.

However, by the end of 1987, deaths had increased to 44.2 per 1000, with only 28 percent of deaths due to these causes, and 64 percent of deaths due to AIDS or bacterial infections. Thus, the advent of AIDS has introduced a new risk of alarming magnitude. Also of note regarding causes of death in our study was the small increase in deaths in the "other" category, which included deaths from renal failure and bleeding diatheses, among other causes; in at least two of these cases, the patients were suspected of having HIV-related nephropathy or HIV-related thrombocytopenia, respectively, suggesting that HIV infection may have been implicated in these deaths as well. Further study of larger populations is required to elucidate the relation between HIV infection and less common modes of death such as these; however, it is of interest that New York City vital statistics data indicate a similar increase in such miscellaneous causes of death among drug users over the same interval in which death rates from AIDS and bacterial infections were found to rise more dramatically.[5,8,9]

In addition to the striking increases in mortality among patients in our methadone program population, the increasing incidence of AIDS has generated significant morbidity as well. The rate of 20.4 new cases per 1000 persons in 1987 is an order of magnitude higher than an estimate derived for intravenous drug users in New York in 1983–84.[6] It must also be recognized that our reported incidence is an underestimate of the total number of cases which could now be classified as AIDS, since we intentionally used the more restrictive original CDC AIDS case-definition[23] in order to maintain uniform reporting criteria throughout the study period. While we do not therefore have systematic data regarding the increased number of AIDS cases which would be expected under the revised case-definition criteria,[24] clinical experience involving our patient population has suggested that use of the revised criteria would expand the group of AIDS-diagnosed patients by approximately 30–40 percent (unpublished data). This estimate would be in the same range as what was recently calculated by the CDC concerning the percentage of AIDS cases among intravenous drug users in 1988 meeting only revised case-definition criteria.[34]

For more than 2 percent of all patients in the methadone program population per year to be newly diagnosed with AIDS has had important implications for patient care and clinical practice. HIV infection and AIDS have clearly become the preeminent health problems of our patient population, affecting all areas of treatment, including medicine, nursing, counseling, and administration. Few, if any, other areas of the health care system are now being faced with such a high concentration of patients with or at risk for HIV infection; indeed, drug treatment programs may be among the only clinical settings in which the entire patient population is defined by a history of high-risk behavior for HIV infection. Observations that 50 percent or more of intravenous drug users in New York are already infected with HIV,[35,36] and data from a 1985 seroprevalence study in our own population indicating close to a 40 percent prevalence of HIV antibody,[20–22] suggest that any such treatment program for drug users cannot help but be transformed by the consequent impact of AIDS on its patient population. This trend has been amplified by the acceptance into drug treatment of new patients from hospital AIDS units, where fewer than 30 percent of drug users newly diagnosed with AIDS have been found to be enrolled in drug treatment programs.[37]

The need to expand the availability of drug treatment in response to the AIDS epidemic has been stressed repeatedly.[7,12,38–42] However, our data suggest that in order to deal effectively with the AIDS crisis, treatment programs will need to receive much-expanded support for on-site medical services and ancillary care to address the steadily increasing burden of HIV-related morbidity and mortality among their patient populations.

Treatment programs are indeed well-situated to provide comprehensive care to patients with HIV infection, including counseling, family supportive services, and, increasingly, primary medical care interventions such as antituberculous chemotherapy,[22] zidovudine, aerosolized pentamidine, and reproductive health care services for at-risk women. However, the growing volume of methadone treatment program patients with AIDS and HIV-related conditions threatens to overwhelm existing drug treatment resources unless structural changes are made to address the new dimensions of these patients' need for care. Such changes should begin with a thorough re-examination of the character and mission of drug treatment programs, including funding, reimbursement mechanisms, staff training and development, need for interdisciplinary collaboration, and the relationship between drug abuse treatment agencies and local and state health departments. Failure to act now may result in a missed opportunity to use a key point of access to intravenous drug users at the center of the expanding AIDS epidemic, and (because of the great stresses which AIDS places on drug treatment program staff) may also lead to the deterioration of existing services.

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ANA Issues Call for Abstracts of Computer Demonstrations in Nursing

The American Nurses' Association (ANA) Council on Computer Applications in Nursing will sponsor a computer demonstration theater and software exchange at the ANA Biennial Convention in Boston, June 17–19, 1990. The council is now issuing a call for abstracts of demonstrations in computer applications in nursing. All types of nursing software and related hardware and peripherals are being solicited.

The deadline is November 15, 1989 for submission of written abstracts of demonstration requests. Individuals submitting abstracts will be notified of acceptance by December 15, 1989. Attendees are urged to bring software they would like to exchange; diskettes should be formatted using MS-DOS. Several public domain software packages will be available for review and copying. A limited number of blank diskettes will be available on-site for purchase

To obtain special rules on abstract submission as well as information on hardware to be used, contact: Catherine Taylor Foster, PhD, RN, ANA, 2420 Pershing Road, Kansas City, MO 64108. Tel: (816) 474-5720, ext. 326.

Suicide Risk among Women with Alcohol Problems

EDITH S. LISANSKY GOMBERG, PHD

Abstract: The current study compares 301 alcoholic women, ages 20 to 50, interviewed in 21 treatment facilities, and a control group of nonalcoholic women matched for age and socioeconomic status of family of origin. A significantly higher percentage of alcoholic women (40.0% vs 8.8%) reported having made suicide attempts, a difference of 31.2 percentage points (95% CI = 23.9, 38.5). Age comparisons within the alcoholic sample show the percentage of younger alcoholic women attempting suicide to be twice as great as the percentage of the alcoholic women suicide attempters in their forties. Such age differences were not found among the nonalcoholic control group. The findings suggest that youthfulness and alcohol/drug abuse are the critical combination for high risk. Awareness of the suicide attempt risk potential is necessary both for emergency room personnel and for substance abuse facility workers. (*Am J Public Health* 1989; 79:1363–1365.)

Introduction

Almost a century ago, Durkheim's classic work on suicide pointed out that more men than women committed suicide, but that suicide attempts were more frequent among women.[1] There have been attempts to explain this gender difference in terms of sex roles and cultural attitudes.[2–4] Durkheim also noted the relation between suicide and alcoholism and, indeed, suicide is one of the more frequent causes of death among alcoholics.[5–8] This relationship between alcoholism and suicide has been related to depression and other psychiatric disorders, to erosion of social networks, and to disrupted interpersonal relationships.[9–12]

Similar male:female patterns of completed suicide and suicide attempts seem to prevail among alcoholic persons.[13–16] Goodwin concluded that there was an association between alcoholism and suicide among White male alcoholics during the middle years of life, whereas, ". . . attempters generally are younger and are more likely to be women . . ."[17]

A review of the epidemiology of suicide attempts[17] shows the risk years to be the twenties and the same review showed more female than male attempters in the countries and time periods studied. A recent analysis of male and female suicide rates[18] found completed suicide rates are significantly higher for men.

Suicide rates are high among both male and female alcoholics. A recent study of predictors of mortality among women alcoholics found significantly higher mortality for alcoholic women than for the general population of women; "accidents and violence" accounted for 26 percent of the deaths, ranking second as the cause of death in the alcoholic sample.[19] There are few studies of suicide attempts by alcoholics, by women alcoholics, or by women alcoholics of different ages. We studied suicide attempts in alcoholic and nonalcoholic women by age group.

Methods

Three hundred and one White women, admitted to 21 alcoholism treatment facilities in Michigan, completed a one-to-two-week period of detoxification and were then interviewed by trained female interviewers of the Field Office, Institute for Social Research, University of Michigan.

The 21 facilities included inpatient, outpatient, small town, and urban facilities, and the women were referred to the facility by self, physician, family member, the court, friends, etc. All consecutive admissions between the ages of 20 and 50 were asked to participate; refusal rate was 7 percent. All respondents were White because available evidence indicates differences in age, drinking patterns, and alcohol-related problems when White and Black alcoholic women are compared.[20] Age at onset was, inevitably, related to current chronological age, i.e., alcoholic women in their twenties had a mean age at onset of 19.58 years (standard deviation = 3.18); alcoholic women in their thirties reported their mean age at onset as 28.34 years (standard deviation = 5.35); and the alcoholic women in their forties reported mean age at onset as 36.52 years (standard deviation = 5.93).

When 200 of the 301 women in the alcoholic sample had been interviewed, the demographic information was evaluated in order to obtain a nonalcoholic sample of women approximately matched on socioeconomic status. The control women were White, between 20 and 50 years of age, and approximately of the same social class as the alcoholic women. They were selected by nomination; the alcoholic women who remained to be interviewed (100) and women in the local chapters of Alcoholics Anonymous and Women in Sobriety were asked to give the name of an acquaintance or neighbor who was *not* alcoholic. Refusal rate of the nominated women was the same as that of women in treatment; when funding was terminated, interviews had been completed with 34 nonalcoholic women in their twenties, 53 in their thirties, and 50 in their forties. Of these 137 control women, none had ever needed a drink before breakfast and none have ever sought help because of their drinking, three had gotten into trouble at school, one had been arrested for driving under the influence, and one reported that she had "neglected obligations" for two days. Two said that they had once "wanted to stop drinking but couldn't." As a group, however, they were nonalcoholic.

The economic status of family of origin and the white/blue collar status of respondents' parents were similar in the alcoholic and nonalcoholic groups.[21] The control group was also compared with a random sample of women interviewed in an earlier, national survey.[22] When women between the ages of 20 and 50 from the national sample were compared with the present control group on items dealing with psychiatric symptomatology, no significant differences were apparent.*

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*Data available on request to author.

The standardized interview schedule include items dealing with demographics; early family experience; drinking history; marital, parenting and employment experience; health; social supports; pregnancies; drug usage; and psychiatric symptomatology. The interview was composed of items culled from several alcoholism study interviews in the Connors Collection of the Rutgers Center of Alcohol Studies, from the Diagnostic Interview Schedule (DIS) of the National Institute on Mental Health, from the Center on Epidemiological Studies Depression Scale (CES-D), and from several earlier surveys conducted at the Institute for Social Research. After field-testing and revising, the interview schedule was reviewed by a panel of three clinicians and a panel of three recovered alcoholic women.[21]

Although alcoholic and nonalcoholic women were matched for age and for socioeconomic status of family of origin, the alcoholic women reported more behavioral, emotional and impulse control problems in childhood and adolescence.[21,23] There are significant differences between the two groups of women in educational achievement, marital history and marital status, and occupational level.[21]

Results

The difference between alcoholic and nonalcoholic women in the proportion reporting any suicide attempt is striking: 40.0% versus 8.8%, a difference of 31.2 percentage points (95% CI = 23.7, 38.5) (Table 1).

Among alcoholic women, suicide attempts were twice as common at ages 20–29 as at ages 40–49. The same age differences, however, did not appear in the nonalcoholic sample. These results do not support the idea that younger women in general are at more risk for suicide attempts than women of other ages.

When the women were interviewed, they were asked about the use of substances other than alcohol, and the alcoholic women reported significantly more often that they had used or were currently using nicotine, prescribed drugs, and banned substances (Table 2). Those who had made suicide attempts were also more likely to have used any of the drug substances asked about. There were too few nonalcoholic women who reported a suicide attempt (N = 12) for meaningful comparisons; it is of interest that those who have made a suicide attempt are also more likely to be users of Valium and sedatives.

TABLE 1—Proportions of Alcoholic and Nonalcoholic Women Reporting Suicide Attempts, by Age

Age (years)	Alcoholic N/Total (%)	Nonalcoholic N/Total (%)	% Difference	95% Confidence Interval
20–29	50/99 (50.5)	3/34 (8.8)	41.7	27.8, 55.5
30–39	46/108(43.0)	5/53 (9.4)	33.6	21.2, 45.9
40–49	24/94 (25.5)	4/50 (8.0)	17.5	5.9, 29.2
All Women	120/301(40.0)	12/137(8.8)	31.2	23.9, 38.5

Among alcoholic women, there was a clear difference in the reported psychiatric symptoms of suicide attempters and nonattempters. Those alcoholic women who have made suicide attempts also reported significantly more tension, indecisiveness, anxiety and nervousness (Table 2). The report on psychosomatic symptoms was mixed: attempters report more dizziness, hand trembling, and headaches than nonattempters but there is little difference between the two groups in loss of appetite, upset stomach, shortness of breath, hives, or sweating hands.

There was a positive but not strong relation between paternal alcoholism and suicide attempts. Of the alcoholic women who made at least one suicide attempt, 63 percent reported a problem-drinking father; among the nonattempters it was 48 percent. Among the nonalcoholic women, 73 percent of attempters reported having had a problem-drinking father; among nonattempters, it was 28 percent. Among the alcoholic women having an older sibling who was a heavy or problem drinker while the respondent was growing up differentiated between those who made at least one suicide attempt and those who did not: 66 percent and 52 percent. It is of interest that maternal alcoholism did not differentiate

TABLE 2—Ever or Current Drug Use and Psychiatric Symptoms Reported by Alcoholic Women, Suicide Attempters and Nonattempters

Drug/Symptoms	Attempters N/Total (%)	Nonattempters N/Total (%)	Difference	95% Confidence Intervals
Drug				
Nicotine	107/120 (89.2)	140/180 (77.8)	11.4	3.1, 19.7
Valium	93/120 (77.5)	106/180 (58.9)	18.6	8.2, 29.2
Stimulants	86/119 (72.3)	91/180 (50.6)	21.7	10.8, 32.6
Sedatives	79/120 (65.8)	74/180 (41.1)	24.7	13.5, 35.9
Marijuana	82/120 (68.3)	77/180 (42.8)	25.5	14.5, 36.6
Psychodelics	54/120 (45.0)	45/180 (25.0)	20.0	9.0, 31.0
Cocaine	51/120 (42.5)	37/180 (20.6)	21.9	11.3, 32.6
Heroin	17/120 (14.2)	7/180 (3.9)	10.3	3.4, 17.2
Symptom				
Feel explosive	59/119 (49.6)	40/178 (22.5)	27.1	16.2, 38.0
Indecisive	51/120 (42.5)	37/179 (20.7)	21.8	11.1, 32.5
Tense and nervous	43/120 (35.8)	31/180 (17.2)	18.6	8.4, 28.8
Fearful, anxious	31/120 (25.8)	22/179 (12.3)	13.5	4.3, 22.8
Difficulty concentrating	38/119 (31.9)	24/179 (13.4)	18.5	8.7, 28.3
Difficulty getting up in the morning	34/120 (28.3)	26/178 (14.6)	13.7	4.1, 23.4
Thoughts of others harming you	34/120 (28.3)	27/180 (15.0)	13.3	3.7, 23.0
Thoughts of harming other people	37/120 (30.8)	26/180 (14.4)	16.4	6.6, 26.1

between suicide attempters and nonattempters, alcoholic or controls.

Discussion

The results suggest that it may not be youthfulness per se which is critical in the relatively high percentage of young female suicide attempters seen in hospital emergency rooms but the combination of youth and substance abuse. Among the nonalcoholic women in this study, there was little difference in the proportion of suicide attempters among younger and older women.

An attempt was made to deal with the question of bias in the selection of clinical subjects by interviewing the patients of 21 different treatment facilities. While the facilities were limited to southeastern Michigan, they represented a wide range of treatment orientations and methods. One may question whether alcoholic and control women are equally likely to admit to a suicide attempt, to remember such an attempt, or to report a suicide attempt. There is no unequivocal answer to this question, but if experimental procedures are reasonably well planned, and the interviewers skilled so as to establish excellent rapport, one can assume that the groups of women were reasonably comparable on this score.

Why the age differential among alcoholic women? One could speculate about the role of depression, impulsivity, hostility, hysterical trends, risk-taking behaviors, and combinations thereof—and the linkage between such patterns of behavior and heavy alcohol intake among different age groups of alcoholic women. Analyses of other data obtained in this study of alcoholic women[21] show nonsignificant age differences in depression among alcoholic women, but significant age differences in early history of impulse control.[23]

Emergency room personnel are acutely aware of the problems of drug overdose and drug abuse. It is worth reiterating: while it is true that young women are a high-risk group for suicide attempts,[24] it is also true that such attempters are likely to have a history of alcohol or other drug abuse. Whether this is due to "... social stigmatization and disapprobation"[25] is beside the point. A person presenting with a suicide attempt is in need of assessment and appropriate referral from the emergency room. As for substance abuse treatment facilities, it is important that they be aware of the suicide attempt potential of young women drug abusers; the abuse of a given substance or substances may not be preventable at the point of entry into treatment but suicide attempts should be investigated with the goal of prevention of such attempts.

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Prevention '90 Set for April 18–22, 1990 in Atlanta

"Assuring the Public's Health" will be the overall theme of the Prevention 90 meeting, to be held April 18–22, 1990 in Atlanta. This will be the 7th annual national meeting on disease prevention and health promotion sponsored by the American College of Preventive Medicine, the Association of Teachers of Preventive Medicine, and co-sponsored by a number of other health organizations, including the American Public Health Association.

To receive further information and obtain the standard abstract forms, contact: Joanne Angle, Association of Teachers of Preventive Medicine, 1030 15th Street, NW, Suite 410, Washington, DC 20005. Telephone: (202) 789-2928.

The deadline for abstract submission is October 6.

Business Recession, Alcohol Consumption, Drinking and Driving Laws: Impact on Oklahoma Motor Vehicle Fatalities and Fatal Crashes

ANDREAS MULLER, PHD

Abstract: In 1982, Oklahoma enacted a series of drinking and driving laws. In the ensuing years, motor vehicle fatalities and fatal crashes were reduced by one-third. Factors contributing to this reduction were examined using interrupted time series analysis of monthly rates of motor vehicle deaths and fatal crashes for the period January 1980 to December 1986. Decreasing per capita alcohol consumption and increased unemployment apparently account for most of the fatality and fatal crash reduction in Oklahoma. The enactment of two traffic safety laws—one specifying the illegal blood alcohol concentration level (BAC law) and the other facilitating license withdrawal from suspected drunk drivers (administrative per se law)—together reduced Oklahoma traffic deaths and fatal crashes by about 9 percent. The effectiveness of the laws appeared to be greatest in the first two years following their enactment. (*Am J Public Health* 1989; 79:1366–1370.)

Introduction

In 1983, Oklahoma motor vehicle fatalities decreased by more than one-fifth and have continued to decrease since then.[1] The oil shortage and the reduction of the national speed limit in 1973 had less impact on motor vehicle fatalities. The only comparable decrease in such deaths occurred in 1942, reflecting the American entry into World War II.

In July 1982, the Oklahoma state legislature established a blood alcohol concentration level of .1 g/dl (22 mmol/L) as "evidence per se" of driving while intoxicated (BAC law). In April 1983, this law was reinforced by an administrative per se law (ADM law) permitting automatic license revocation, if a police officer was refused the right to administer a blood alcohol test to a suspected drunk driver. In September of the same year, Oklahoma raised the minimum age for purchase, possession, and consumption of beverages in excess of 3.2 percent alcohol concentration from 18 to 21 years.[2] An interrupted time series analysis of monthly motor vehicle fatalities and fatal crashes from January 1975 to March 1986 suggested that the law reduced Oklahoma fatalities and fatal crashes by about one-third.[3] In addition, the effectiveness of the administrative per se law appeared to increase over time; this finding departed from previous studies showing the deterrent effect of drunken-driving laws and related enforcement campaigns to be short-lived, reviewed elsewhere.[4]

The findings of an effect of the Oklahoma laws contrast with studies of similar legislation enacted in California, Maine, and Massachusetts conducted by Hilton[5] and Hingson, *et al*,[6] which have found the trends in monthly single-vehicle night time fatal crashes comparable to those in adjacent states without the legislation and to trends in non-alcohol-related fatal crashes.

A recent nationwide study by the Insurance Institute for Highway Safety (IIHS) for the period 1978–85 found that administrative per se laws and BAC laws reduced fatal crashes by 4.6 percent and 2.4 percent, respectively. State-specific variation in effectiveness may occur and can only be determined by additional analyses.

Unlike other parts of the United States, Oklahoma experienced an oil-related economic boon during the period 1980–82 followed by a severe recession beginning with Penn-Square Bank failure in July 1982. The recession was prolonged by a sharp drop in crude oil prices in January 1986. The unemployment rate for the civilian labor force in Oklahoma increased from a 4–5 percent level in 1980–81 to over 10 percent in early 1983, receded to 7–8 percent in 1984–85, and increased again in 1986 (Figure 1). Business conditions affect the motor vehicle fatality trend since unemployment reduces work-related travel and disposable income, making alcohol consumption and leisure travel less likely.[8]

Apart from the effects of business cycles, alcohol consumption patterns have changed over recent years, with decreased consumption of distilled liquor and increased consumption of beer and wine having the overall effect of reducing the per capita amount of pure alcohol (ethanol) consumption since 1982.[9] Correspondingly, between 1982 and 1986, the proportion of fatal crashes involving intoxicated drivers has decreased by 11 percent.[10] Per capita pure alcohol consumption by Oklahomans (Figure 2) parallels the national trend showing a gradually decreasing trend since 1982.

Since alcohol consumption contributes to motor vehicle crashes and deaths, the gradual reduction in alcohol consumption in Oklahoma should have resulted in fewer alcohol-related crashes regardless of the imposition of stricter alcohol-related traffic safety laws. The effect of such legislation would be overestimated if the alcohol consumption trend and the effect of the business recession were not controlled.

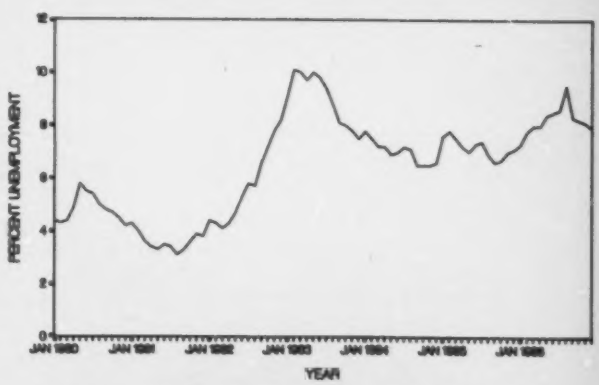

FIGURE 1—Monthly Unemployment Rate, Oklahoma 1980–86
SOURCE: Oklahoma Employment Security Division

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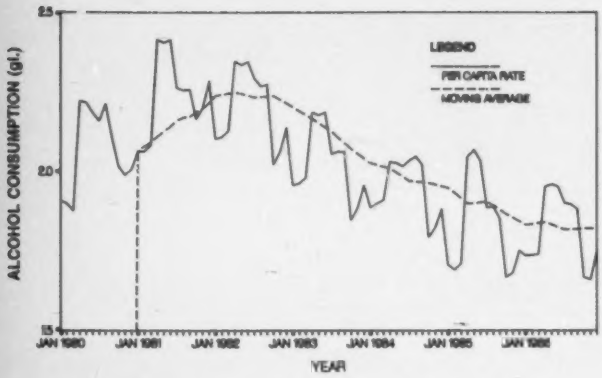

FIGURE 2—Monthly per capita Alcohol Consumption and Moving Averages, Oklahoma 1980–86
SOURCE: Appendix A

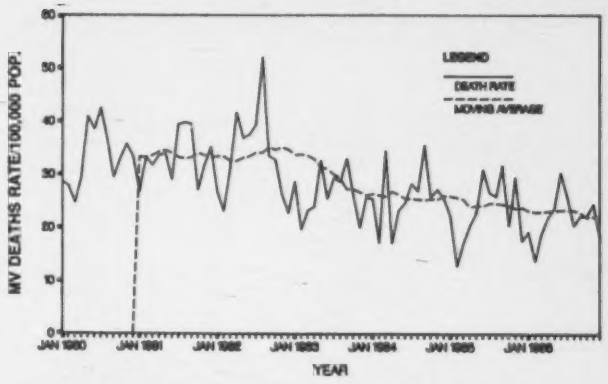

FIGURE 3—Monthly Motor Vehicle Death Rates and Moving Averages, Oklahoma 1980–86
SOURCE: Department of Transportation, Oklahoma Highway Safety Office

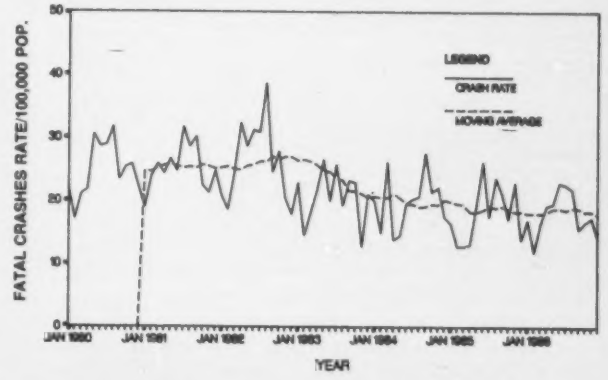

FIGURE 4—Monthly Fatal Motor Vehicle Crash Rates and Moving Averages, Oklahoma 1980–86
SOURCE: Department of Transportation, Oklahoma Highway Safety Office

Methods

The time series analysis was based on three sets of data pertaining to motor vehicle fatalities and fatal crashes for the period January 1980 to December 1986 (n = 84). The first data set included all deaths whose cause was coded E810-E825, ICDA-9 for Oklahoma residents.[11] About 3 percent of the fatalities occurred beyond state boundaries.

The second data set pertains to the number of crashes involving at least one fatally injured person. Fatal crashes involving heavy trucks and buses (7 percent) were deleted, since operators of those vehicles are likely to be out-of-state residents unfamiliar with Oklahoma's laws regarding drinking and driving.

The third data set is a subset of the second, removing fatal crashes involving drivers under 21 years of age. This deletion controls for the potentially confounding effect of the change in the minimum drinking age law. Due to the small number of fatal crashes involving persons under 21 years of age, no reliable analysis of the effect of the minimum drinking age law could be performed.

To control for changes in Oklahoma's population, we divided the time series by the appropriate population estimates.[12] The time series of fatal crashes involving drivers over 20 years of age was divided by the corresponding age-specific population estimates; the two remaining series were divided by estimates of the total population. The procedure resulted in three sets of rates:

- monthly motor vehicle deaths per 100,000 population,
- monthly fatal crashes per 100,000 population, and
- monthly fatal crashes involving adult drivers per 100,000 population over 20 years of age.

The data pertaining to fatal crashes were extracted from Department of Transportation files and provided by staff of the Oklahoma Highway Safety Office. The unemployment data including estimates for 1986 were obtained from staff of the Oklahoma Employment Security Division.

The Oklahoma Department of Public Safety released license revocation data pertaining to BAC testing. Inspection of the time series and discussion with the administrator of the records division showed that reporting lags of one to two months occurred occasionally. To even out such lags, the data were averaged by quarter.

Oklahoma per capita alcohol consumption was estimated from data provided by the alcoholic beverage industry (Appendix A). The time series were analyzed by ordinary least squares multiple regression using SPSS-X Trends computer software[13] (Appendix B).

Results

Monthly time series of motor vehicle death rates and fatal crash rates in Oklahoma (Figures 3, 4) showed typical seasonality with the highest rates in the summer months and the lowest rates in February. The time series for the subgroup of fatal crashes with adult drivers (not shown) was very similar to those shown. All time series have one exceptionally high rate, August 1982, an outlier removed in subsequent analyses (see Appendix B). The trend began to decrease late in 1982, reached a new plateau by the last quarter of 1983, and decreased more slowly thereafter.

Table 1 shows the best fitting regression models using dummy variables and counters for the three monthly time series. The models indicate that with the enactment of the BAC law (July 1982) the level of the time series dropped between 5.9 and 12.2 percent. With the enactment of the administrative per se law (April 1983), the levels of the time series further dropped between 9.7 and 13.2 percent when compared with levels before the enactment of the law. The

TABLE 1—Effects of Blood Alcohol Concentration and Administrative per se Laws on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
BAC Law	b	−4.05	−2.34	−1.87
	s.e.	1.49	1.32	1.68
	p	.01	.08	.27
ADM Law	b	−3.78	−2.44	−4.21
	s.e.	1.77	1.57	1.99
	p	.04	.13	.04
ADM Law counter	b	−.099	−.078	−.093
	s.e.	.044	.039	.05
	p	.03	.05	.07
F		14.4	8.8	8.9
Adj. R^2		.69	.57	.57
Durbin-Watson		1.87	2.09	1.86
Mean rate before laws		33.2	25.3	32.0
Estimated % Reduction Due to Laws				
BAC Law		−12.2	−9.3	−5.9
ADM Law		−11.4	−9.7	−13.2
ADM Law counter		−13.4	−13.8	−13.1
Total		−37.0	−32.8	−32.2

The models omit coefficients for intercept and 11 dummy variables representing seasonality.
p = probability of two-tailed test; b = 0.
n = 84

TABLE 2—Effects of Administrative per se Law, Per Capita Alcohol Consumption and Unemployment Rate on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
Alcohol consumption	b	11.42	10.74	17.52
	s.e.	5.46	4.64	6.25
	p	.04	.02	.01
Unemployment rate	b	−.78	−.31	−.25
	s.e.	.33	.28	.38
	p	.02	.28	.52
ADM law	b	−3.53	−2.58	−2.27
	s.e.	1.88	1.60	2.15
	p	.07	.11	.29
F		10.9	9.0	7.4
Adj. R^2		.63	.57	.52
Durbin-Watson		2.00	2.14	2.05
Mean rate before laws		33.2	25.3	32.0
Estimated % Reduction due to Law				
ADM Law		−10.6	−10.2	−7.1

The models omit coefficients for intercept and 11 dummy variables representing seasonality.
p = probability of two-tailed test; b = 0.
n = 84

negative coefficients for the ADM time counter indicate that the rates continued to decrease. By December 1986, the fatality and fatal crash rates were about one-third lower than before the enactment of the laws.

Table 2 shows the decrease in motor vehicle deaths and fatal crashes associated with increased unemployment, reduced alcohol consumption, and the administrative per se law. Depending upon the time series, the law appeared to have reduced fatality and fatal crash rates between 7.1 and 10.6 percent. However, the coefficients for the BAC law were inconsistent in sign and statistically insignificant. Therefore, the BAC law dummy variable was deleted from the final mode specification.

Another analysis, shown in Table 3, includes fatal crashes in Oklahoma which police determined not to be alcohol related. This series is a proxy for changes in extraneous factors, such as travel or weather. In addition, the dummy variables and counters representing BAC and administrative per se laws are replaced by a measure of enforcement of drunken driving: the number of licenses revoked involving BAC tests.

The models fit the time series very well, mainly because the series is regressed on the time series of nonalcohol-related fatal crashes. The multiple regression estimates show that per capita alcohol consumption increases fatality and fatal crash rates, while increased law enforcement has the opposite effect. Increased enforcement reduced motor vehicle death rates by 9.6 percent after the enactment of the administrative per se law, and by 5.0 percent during the nine-month period (7/82 to 3/83) during which only the BAC law was in effect. The estimates are similar for the time series of fatal crash rates.

In Table 3 the unemployment rate showed a weak direct effect, because part of the overall unemployment effect was shared by the trend of nonalcohol-related crashes.

Police enforcement varied over the study period (Figure 5), with a gradual increase in license revocations following enactment of the BAC law. Enforcement was highest during the first quarter after the enactment of the administrative per se law. Thereafter, enforcement activity declined until early 1985 despite some seasonal variation. Thus, the effect of the two laws should have been most pronounced during the first year after enactment of the administrative per se law. It is estimated that fatal crash rates of adult drivers were reduced by 12.3 percent during the first year of enactment of the administrative per se law, 10.8 percent during the second year of enactment, and 7.8 percent during the remaining 21 months (4/85–12/86). Since both laws were in effect from April 1983 on, the estimates reflect the combined effective-

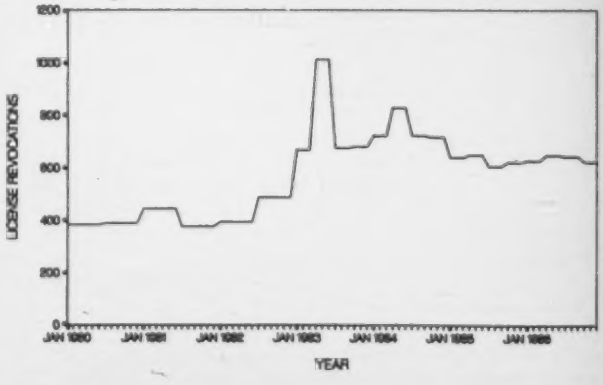

FIGURE 5—License Revocations due to BAC Tests, Oklahoma 1980–86
NOTE: Data represent quarterly averages
SOURCE: Oklahoma Department of Public Safety

TABLE 3—Effects of License Revocation, Per Capita Alcohol Consumption, Unemployment Rate and Nonalcohol-related Crashes on Motor Vehicle Deaths and Fatal Crashes, Oklahoma 1980–86

Independent Variables		MV Death Rate (100,000 Pop)	MV Fatal Crash Rate (100,000 Pop)	MV Fatal Crash Rate Adult Drivers (100,000 Pop >20 yrs)
Non-alcohol-related fatal crash rate	b	1.23	1.20	1.19
	s.e.	.16	.09	.09
	p	<.001	<.001	<.001
Alcohol consumption	b	10.57	7.28	10.97
	s.e.	2.91	1.77	2.23
	p	<.001	<.001	<.001
Unemployment rate	b	−.24	.14	.18
	s.e.	.32	.20	.26
	p	.47	.49	.50
License revocations	b	−.0107	−.0091	−.0104
	s.e.	.004	.002	.003
	p	007	<.001	.001
F		23.0	38.4	35.8
Adj. R^2		.80	.87	.86
Durbin-Watson		1.87	1.98	2.03
Mean rate before laws		33.2	25.3	32.0
Estimated % reduction due to Increased Law Enforcement				
BAC Law (7/82–3/83)		−5.0	−5.6	−5.1
ADM + BAC Law				
(4/83–3/84)		−12.1	−13.6	−12.3
(4/84–3/85)		−10.6	−11.9	−10.8
(4/85–12/86)		−7.6	−8.5	−7.8
(4/83–12/86)		−9.6	−10.8	−9.8

The models omit coefficients for intercept and 11 dummy variables representing seasonality.
p = probability of two-tailed test; b = 0.
n = 84

ness of the laws. The effectiveness of the BAC law for the nine-month period preceding the introduction of the administrative per se law is estimated at about 5 percent.

Discussion

The enactment of a BAC law and an administrative per se law in Oklahoma reduced fatality rates and fatal crash rates by about 9 percent in the four years following their enactment. This estimate is less than one-third of the size found in a previous study of the effectiveness of the Oklahoma administrative per se law.[3] The difference in estimates is due mainly to the control of alcohol and to unemployment trends which were not considered in the earlier study.

The effect of the BAC law was not statistically significant when other variables were controlled and deleted from the analysis (Table 2). The most plausible reason is that the enactment of the BAC law nearly coincided with rapidly rising unemployment making the BAC regression estimate unreliable. However, when license revocations were used to determine the effectiveness of the laws, the BAC law was estimated to have reduced fatality and fatal crash rates by about 5 percent. This estimate pertains to the initial nine-month period separating the two laws; the long-term effectiveness of this legislation may be different. A recent nationwide study estimated an average (long-term) effect of 2.4 percent for BAC laws and 4.6 percent for administrative per se laws.[7] These estimates approximate the combined long-term effect (7.2 percent), past two years, found in this study.

Deleting fatal crashes involving drivers under 21 years of age did not markedly affect the estimated effectiveness of the laws (Table 3). This finding suggests that the BAC and the administrative per se law are responsible for the decrease in fatal crash rates rather than the raising of the minimum drinking age law.

The accuracy of police-reported alcohol-impaired driving is a concern in this study. Oklahoma police could not determine alcohol involvement for 11 percent of drivers involved in fatal crashes. The percentage was about the same from 1980 to 1986 suggesting no increased effort by police to report alcohol involvement in fatal crashes.

In addition, an increase in the reporting of nonalcohol-related fatal crashes coinciding with the enactment of the laws could not be found. Such a change would be expected, if police officers became more lenient when faced with administering harsher legal sanctions. Errors in the reporting of alcohol involvement would not bias the regression estimates unless the error rate changed over time; no evidence supporting this conclusion could be found.

The gradual reduction in per capita alcohol consumption has had a substantial effect on Oklahoma's fatal crash trend. The annual per capita consumption dropped by about .4 gallons pure alcohol between 1981 and 1986. The corresponding decrease in motor vehicle death and fatal crash rates is estimated at 12–14 percent, controlling for changes in unemployment and drinking and driving laws.

The total effect of the business recession, measured by the unemployment rate, is less clear. Since the unemployment rate was on the average 4 percent higher after the enactment of the drinking and driving laws, about 10 percent of the reduction in death rates should be due to increased unemployment.

However, increased unemployment appears to have had little direct effect on fatal crash rates, although indirect effects via the other variables are consistent with the findings. For instance, unemployment had a significant negative effect on nonalcohol-related fatal crashes. A 4 percent increase in the unemployment rate reduced fatal crashes by 2/100,000 controlling for seasonal variations. Thus, increased unemployment, by reducing nonalcohol-related fatal crashes, could have contributed to a 7 percent reduction in the fatal crash rates.

APPENDIX A

Per Capita Alcohol Consumption Index

Data for estimating monthly per capita consumption of pure alcohol (ethanol) in Oklahoma are provided by the Distilled Spirits Council of the United States, the Beer Institute and the Wine Institute. The data pertain to shipment and sales records regularly compiled by the alcoholic beverage industry. Beer and wine data were provided by month, while distilled spirits data could only be obtained by year. To estimate monthly pure alcohol consumption, the data were converted into gallons and the annual amount of distilled spirits was divided by 12. The following formula was used to derive the estimated monthly per capita alcohol consumption.

P.C. ethanol consumption = (.414 * DSP + .033 * Beer + .129 * Wine) * 12/POP;(1)

where: DSP = distilled spirits (gl.);
POP = Oklahoma resident population over age 20;

The coefficients for distilled spirits and wine refer to the average alcohol concentration estimated by the Alcohol Epidemiological Data System.[14] The average alcohol concentration of beer is less in Oklahoma than in other states,

due to the mandated 3.2 percent limit for beer sold in grocery and convenience stores. According to Oklahoma tax receipt data, only 5 percent of beer has higher alcohol concentration. The estimated average alcohol concentration of beer sold in Oklahoma is estimated to be 3.3 percent.

APPENDIX B

Statistical Model and Method of Analysis

The time series are analyzed by a form of multiple regression which uses dummy variables and dummy counters to represent changes in the level (mean) and trend (slope) of the time series.[15,16] Another set of dummy variables is used to represent seasonality.[17] The basic model for this study is as follows:

$$Y_t = a + b_1X_1 + B_2X_2 + B_3X_3 + B_4X_4 + \Sigma B_iX_i + e_t; \quad (2)$$

where: Y_t = monthly motor vehicle death rate (fatal crash rate);
a = reference category;
b_i = regression coefficients;
X_1 = linear trend counter, coded 1 to 84;
X_2 = dummy variable, coded '0' before BAC law went into effect and '1' thereafter;
X_3 = dummy variable, coded '0' before administrative per se law went into effect and '1' thereafter;
X_4 = dummy variable counter, coded '0' before administrative per se law went into effect and '1' to n thereafter'
ΣX_i = set of 11 dummy variables identifying months (January to November) representing seasonality;
e_t = error term;

The set of monthly dummy variables controls for seasonal fluctuations in the time series, while the linear trend counter X_1 controls for any existing secular, linear trend which is independent of the effects of traffic safety laws. The effects of the two laws are modeled by variables X_2 to X_4. The laws may be effective, if one or more of the coefficients associated with variables X_2 to X_4 are negative and statistically significant. To prevent multicollinearity, a dummy variable counter for the BAC law was excluded from the model.

In order to control for unemployment and alcohol consumption trends, these variables were added to equation (2). The best-fitting model was determined by deleting statistically insignificant terms and by inspecting residual plots.

First, extreme outliers ($z = \pm 2.57$ standard deviations) were replaced by linear interpolation using data pertaining to adjacent months. Data for August 1982 and March 1984 (observations 32 and 51) were replaced by this method normalizing the distributions of residual plots.

Second, the distribution of residuals for the best-fitting models did not significantly depart from homoscedasticity, although the variances of residuals were somewhat smaller for the period after the laws went into effect. Converting the dependent variables to their natural logarithms further stabilized the variance of residuals, but did not change the regression estimates substantially. The time series were also analyzed by weighted least squares regression without substantial change in findings.

Third, besides Durbin-Watson tests of the first order autocorrelation, the residuals were tested by the Box-Ljung statistic. The latter tests showed that higher order autocorrelations were small and consistent with a random process. The autocorrelation plots indicated no significant spikes at critical lags for models including the control variables.

ACKNOWLEDGMENTS

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An earlier version of this paper was presented at the 115th annual meeting of the American Public Health Association, New Orleans, LA, October 1987.

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6th National Cancer Communications Conference Set for January 1990

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Six-Year Follow-up of the First Waterloo School Smoking Prevention Trial

BRIAN R. FLAY, DPHIL, DAVID KOEPKE, PHD, SHIRLEY J. THOMSON, MA, SUSANNE SANTI, MSC, J. ALLAN BEST, PHD, AND K. STEPHEN BROWN, PHD

Abstract: This paper reports six-year follow-up data from the first large-scale randomized trial of the social influences approach to smoking prevention. In 1979, 22 schools were randomly assigned to program or control conditions. Students in program schools received a social influences curriculum in six core and two maintenance sessions in grade 6, two booster sessions in grade 7, and one booster session in grade 8. All students were assessed at pretest (T1), immediate posttest (T2), end of grade 6 (T3), beginning and end of grade 7 (T4 and T5), end of grade 8 (T6), and grades 11 and 12 (T7 and T8). Ninety percent of study students were relocated and data obtained from over 80 percent of them at T8. Program effects on experimental smoking observed in grades 7 and 8 had completely decayed by T8, six years after the beginning of the program. Grade 6 smoking experience and social risk were each strong predictors of T8 smoking behavior. Subjects who had left school were smoking at more than twice the rate of subjects still in high school (grade 12) at T8. We discuss implications of the results. (*Am J Public Health* 1989; 79:1371–1376.)

Introduction

The social influences approach to smoking prevention has proven efficacious in a large number of studies.[1,2] The approach concentrates on making students aware of the social pressures to smoke—from peers, parents and other adults, and the mass media—and on teaching them behavioral skills with which to resist such pressures. Most studies have reported about 50 percent reduced levels of smoking onset by students exposed to social influences programs when compared to control students, at least in the short term. Ultimately, however, the effectiveness of the tested programs, and of the social influences approach generally, must be assessed from long-term results. We must be able to reduce the prevalence of smoking in high school and beyond.[3]

Long-term Effects

To date, only a few investigators have reported results of social influence interventions into high school or beyond two years after the intervention (Appendix A).[4–10] Reported effects become smaller at longer follow-ups, until they disappear altogether at six-year follow-up.[10] Results in all cases are difficult to interpret because of alternative methodological explanations.[1,2] These methodological problems were overcome in "third generation" studies,[1,2] of which the Waterloo study was one. The data reported here are the first long-term follow-up data to be reported from a "third generation" study.

The Waterloo Study

The first large-scale randomized trial of the social influences approach to smoking prevention was initiated in Waterloo, Canada in 1979. At that time, 22 schools with grade 6 students were randomly assigned to receive or not receive a social influences curriculum. Grade 6 students in the 11 program schools received a 6-session curriculum from research staff. Two maintainence sessions were provided at the end of grade 6, two booster sessions in grade 7, and one booster session in grade 8. Appendix B provides a summary of curriculum content, and further details are provided in previous publications.[11–13]

Students in both program and control schools were assessed by questionnaire at pretest (T1), immediately after the core program (T2), at the end of grade 6 (T3), at the beginning and end of grade 7 (T4 and T5), and at the end of grade 8 (T6). Potential underreporting of self-reported smoking behavior was minimized by assurances of confidentiality, and collection of a sample of saliva as a modified "bogus pipeline."[14] Results indicated that although the program did not reduce levels of regular smoking or significantly increase the probability of remaining a nonsmoker, it was successful at preventing the onset of experimental smoking up to the end of grade 8.[12,13] Results were especially encouraging for high-risk students—those at risk to become smokers because they had already tried smoking at grade 6 or because their parents, siblings, and friends were smokers.[11]

Methods

Subjects

Participants in the first Waterloo smoking prevention trial were reassessed five and six years after the beginning of the study, in grades 11 (T7) and 12 (T8). Extensive tracking procedures resulted in the location of a large percentage of the original subjects. For the 11th grade (T7) assessment, 79 percent (223) of the original 281 subjects from one school board were located and 68 percent were tested. However, failure to obtain cooperation from one school board in time resulted in a failure to assess many subjects in grade 11, and only 40 percent (166) of the 412 subjects from the second school board were located, and 35 percent (143) tested. Overall 56 percent (389) of the subjects were located and 48 percent (335) tested.

The following year we obtained increased cooperation from the second school board. This enabled us to locate 623 (90 percent) of all study students and to obtain questionnaire data from 532 (77 percent) of them and telephone information from an additional 28 (4 percent). Therefore, all outcome results reported below are limited to data from T8 when we obtained responses from most subjects.

Measures

Measures used at T8 were essentially the same as those previously used in grades 6 through 8. In addition to self-reported smoking behavior, students and school leavers were asked for demographic information; reports of smoking habits of parents, siblings, and friends; and information on a

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set of mediating variables including knowledge and beliefs regarding smoking, environmental change (from grade school to high school), other substance use, and academic aspirations. Students were also asked for reports of smoking habits of teachers, and information on school environment (including smoking regulations and enforcement).

Smoking behavior categories—On the basis of self-reported smoking behavior, subjects were classified as being in one of five behavioral categories at pretest and at the 12th grade follow-up:

- Never smoker (never smoked even one puff of cigarette);
- Tried once (has smoked, but only once);
- Quitter (has smoked more than once, but has "quit for good");
- Experimenter (currently smokes, but less than once a week);
- Regular (currently smokes at least once a week or more).

Some analyses reported below compare regular smokers against all others. For some analyses, we also classified subjects as to whether or not they were currently smoking at all.

Pretest social environment risk—Risk levels were based on pretest responses to questions about the smoking habits of parents, siblings, and friends. Levels were defined as follows:

- Low risk (no smoking parents, siblings, or friends);
- Intermediate risk (smokers in only one of the three social model groups);
- High risk (smokers in two or all three social model groups).

Both the behavior smoking category and the social environment classifications depend on multiple questionnaire responses. If needed items were missing, the subjects were unclassifiable and excluded from the analyses. Over 93.5 percent had smoking behavioral categories for both pretest and 12th grade and 91 percent had complete data for all variables used in the analysis.

Procedure

Most subjects were still in school, and in the high schools they expected to be in when they last participated in the study. However, a significant number had changed schools or dropped out. Extensive tracking procedures were initiated to find and assess as many of these students as possible. Those students who had left school or had moved to another area were surveyed by mail (see reference 15 for details).

Those students located in schools in the vicinity of the University of Waterloo were surveyed in classrooms. Participation in measurement was by full informed consent by both the students and their parents. Confidential ID numbers enabled us to match student responses across waves of data collection.

Results

Attrition Analyses

Measures taken at T1 were used in a logistic model[16,17] to determine whether subjects lost at T8 differed systematically from those who were retained. Effects analyses could be biased to the extent that variables related to outcome behavior also relate to the process of attrition. There was no significant relation between treatment condition and attrition. Neither was there a relation between pretest social environment risk and attrition. However, there was a relation between pretest smoking behavioral category and attrition, with greater attrition of pretest triers compared to never smokers (odds ratio = 1.84, 95% CI = 1.04, 3.28). Age was also related to attrition. The study retained more subjects who were ages 9–11 at pretest compared to those who were age 12 or older (OR = 2.53, 95% CI = 1.45, 4.39). (These older subjects were more likely to have left school and were thus more difficult to track.) As both students smoking at pretest and older students were more likely to be smoking at the time of the follow-up, these differences tend to bias downward observed smoking rates across all conditions.

Smoking Levels

Approximately 33 percent of all subjects at T8 were regular smokers (95% CI = 27.95, 38.72) and another 10 percent were experimental smokers (95% CI = 6.63, 13.49). By this time only 15 percent had never tried a cigarette (95% CI = 11.07, 19.26).

Students and school leavers—Students and school leavers reported very different patterns of smoking behavior. Approximately 68 percent of school leavers were regular smokers compared to 28 percent of students still in school at grade 12 (OR = 5.54, 95% CI = 3.14, 9.79). Nearly all of the school leavers (98 percent) reported having tried smoking compared to only 84 percent of the students (OR = 12.50, 95% CI = 1.71, 91.8). Figure 1 presents smoking behavioral categories for students in grade 12 and for school leavers.

Measures taken at T1 were used in a logistic model to predict school leaving six years later at T8. There were no significant differences between students and school leavers by original school board, treatment condition, or pretest social environment risk. There were, however, differences by age and by pretest behavioral smoking category. Most of the students were 11 years old at the time of the intervention, but a few were as young as 9 years or as old as 14. Students who were age 12 or older at the time of the pretest were more likely to have left school than those who had been ages 9–11 (OR = 5.05, 95% CI = 2.53, 10.08). Those who had tried smoking once at the time of the pretest were more likely to leave school than those who had never smoked (OR = 2.95, 95% CI = 1.38, 6.29), as were those who had quit (OR = 3.21, 95% CI = 1.30, 7.91) and those who had been regular smokers at the pretest (OR = 7.49, 95% CI = 2.15, 26.09).

However, age and pretest smoking rate differences cannot account for all differences between smoking rates of students and school leavers at the T8 follow-up. When age,

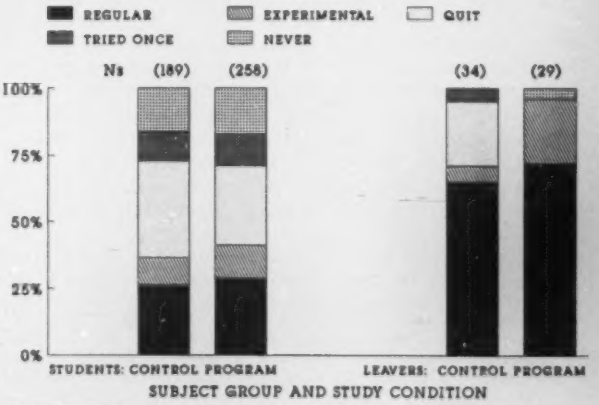

FIGURE 1—Percentage of Grade 12 Students and School Leavers in Smoking Behavior Categories

TABLE 1—Logit Model Summary Table for 12th Grade (T8) Smoking

T1 Predictor of Smoking at T8	Odds Ratio	(95% CI)
Intercept	0.26	(0.15, 0.46)
Smoking Category		
Tried vs Never	1.97	(1.27, 3.06)
Quit vs Never	2.70	(1.46, 4.99)
Experiment vs Never	4.53	(2.19, 9.37)
Regular vs Never	14.35	(3.09, 66.68)
Social Environment Risk		
Intermediate vs Low	1.71	(0.97, 3.02)
High vs Low	2.23	(1.22, 4.07)
Original School Board		
Waterloo vs Oxford	0.69	(0.47, 1.01)
Treatment Condition		
Treatment vs Control	1.22	(0.83, 1.80)

TABLE 2—Logit Model Summary Table for 12th Grade (T8) Regular Smoking

T1 Predictor of Regular Smoking	Odds Ratio	(95% CI)
Intercept		
Smoking Category		
Tried vs Never	1.67	(1.0, 2.70)
Quit vs Never	3.69	(1.96, 6.93)
Experiment vs Never	4.86	(2.3, 9.92)
Regular vs Never	7.09	(2.2, 22.38)
Social Environment Risk		
Intermediate vs Low	1.12	(0.61, 2.06)
High vs Low	1.75	(0.93, 3.29)
Original School Board		
Waterloo vs Oxford	1.56	(1.03, 2.35)
Treatment Condition		
Treatment vs Control	1.24	(0.83, 1.86)

pretest behavioral smoking category, social environment risk, original school board and treatment condition are included with school leaving in a logistic model to predict regular smoking at T8, there is still more regular smoking by school leavers than by students (OR = 2.18, 95% CI = 1.58, 3.01).

Program Effects

Overall program effects also were assessed using a multiple logistic model. The dependent variable is smoking at T8. The primary predictor is treatment condition. The model includes smoking behavioral category at the 6th grade pretest (T1) and pretest social environment risk as additional predictors. These were previously shown to be effective predictors of smoking at the end of 8th grade (T6).[11,13] Original school board is included as a blocking factor for possible differences between the two populations. Table 1 summarizes the results of this analysis. Interactions of treatment condition with each of these two variables then were added to assess possible differential program effects for students at differing degrees of risk. These interactions were not significant.

By T8 there is no longer a significant overall effect for the treatment program. Both pretest social environment risk ($p < .05$) and pretest smoking behavior ($p < .0001$) predict whether students are smoking more than six years later. The odds ratio indicate that those at low social environment risk in grade 6 are least likely to be smoking six years later. There is no significant difference in smoking rates between the two original school districts. Since the interactions are insignificant, there do not appear to be any differential effects of the treatment program for students at different initial levels of risk for smoking. The overall likelihood-ratio test (*Chi-square* = 30.86, $df = 38$, $p > .75$) indicates that the terms in the model are adequate to account for the observed patterns of responses.

A parallel analysis was done using regular smoking as the outcome measure. Table 2 summarizes these results. A similar pattern of effects is observed for regular smoking (at least once a week) as for smoking at all. There are no overall program effects nor differential program effects for students at differing initial levels of risk. Pretest smoking behavior category ($p < .0001$) predicts whether a student is a regular smoker at T8. Pretest social environment risk is a less strong predictor ($.05 < p < .10$) but the odds ratio are in the same direction for regular smoking as they were for smoking. The likelihood-ratio test (*Chi-square* = 43.42, $df = 38$, $p > .25$) indicates that the terms in the model are adequate to account for the observed patterns of responses.

Thus, six years after the beginning of the intervention, there were no overall differences between the program and control groups when all students are considered together. Figure 2 displays the proportion of subjects smoking regularly and experimentally at each wave by experimental condition. It demonstrates decay of the significant difference between conditions in level of experimental smoking that was observed at grades 7 and 8.[13]

Considering pretest smoking behavior and social risk categories,[11] Figures 3 and 4 show how pretest risk is a strong predictor of subsequent smoking level. The probability of regular smoking in high school increases as grade 6 risk increases; the probability of experimental smoking in high school is lowest for the students at lowest risk in grade 6, and the probability of never smoking by grade 12 decreases as grade 6 risk increases. At grades 7 and 8, we observed significant program effects for the high- and middle-risk students—we did not observe effects for the low-risk students because only a few of them had tried smoking by grades 7 and 8. We observed no significant differential program effects by 12th grade for any risk group.

Discussion

Observed effects of the Waterloo program on the onset

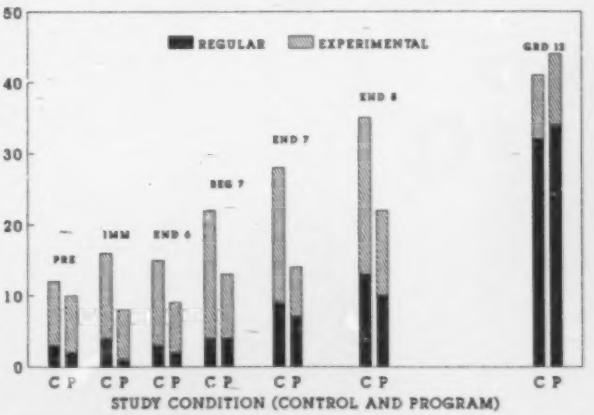

FIGURE 2—Proportion of Subjects Smoking Regularly and Experimentally at Each Wave of the Study

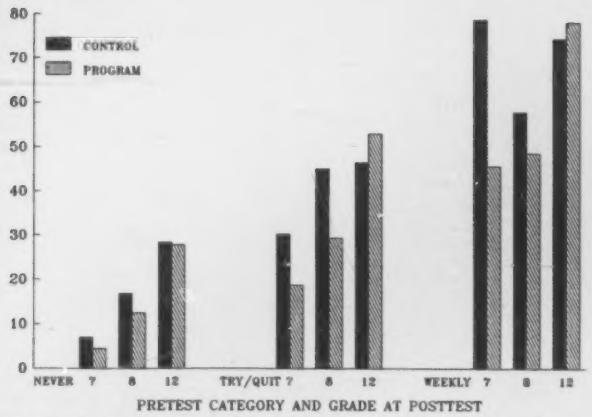

FIGURE 3—Percentage of T8 Subjects Smoking by Pretest Smoking Category and Experimental Condition

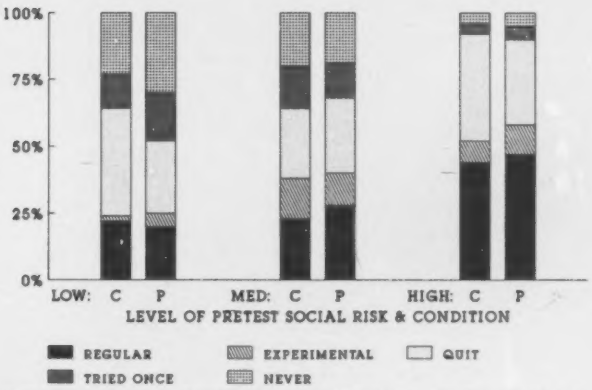

FIGURE 4—Percentage of T8 Subjects in Each Behavioral Category by Pretest Social Risk Level and Experimental Condition

of experimental smoking in grades 7 and 8 were not maintained through grade 12. Expected effects on grade 12 smoking (regular or experimental) did not occur. This finding is consistent with that of the only other study of long-term effects of adolescent smoking prevention programs.[9,10]

The lack of significant preventive effects by grade 12 raises the question of the value of the social influences approach for smoking prevention. In retrospective non-intervention studies, delayed onset is associated with improved prognosis for quitting and lower incidence of total morbidity and mortality.[18,19] Prevention program-induced delays in onset may reduce total lifetime exposure and may be an important outcome.[10] This is particularly true if delayed onset increases the probability of earlier quitting. However, the extent to which an intervention-induced delay in onset is associated with earlier or more successful quitting, or even with decreased exposure to toxins, is not established. That would require following the subjects of this study, and others like it, into middle adulthood. We might expect to find earlier quitting by subjects who are currently smokers but who delayed onset until grade 9 or 10, partly because of the delayed onset and partly because of reduced peer influences.

The value of the social influences approach to preventing the onset of regular smoking by the end of high school needs further study. Results from both the Minnesota and Waterloo studies suggest that program effects obtained in junior high school decay gradually during the following years to totally disappear by grade 12. Such a pattern suggests that booster sessions might be necessary. However, the Waterloo results suggests that boosters in grade 8 are insufficient. These results, together with findings of increased risk at the transition from one level of school to another[20] (or from school to college or working), and the importance of school policy variables[21] suggest the need for boosters in high school. Results of reported high school interventions[6,22,23] suggest that social influence curricula can be effective with high school students, although effects were small in all cases. Booster sessions were also recommended by an expert advisory panel convened recently by the National Cancer Institute.[24]

The apparent lack of effects of social influence programs on smoking prevention by grade 12 should not be over-interpreted. First, as noted above, boosters in early high school years may help to maintain early substantial effects. Second, there is a much better understanding today than 10 years ago of the essential components of effective prevention programs.[24] These improvements may well mean that current versions of social influence programs may produce more durable effects. Third, the broader social norms are now more supportive of nonsmoking, so that students in control schools may, in fact, have been exposed to many of the elements of the social influence approach during the last five years.

One alarming result of this study is the large difference in smoking behaviors between students and school leavers. These differences cannot be explained by differences in age or by pretest differences in smoking risk or experience, and they also replicate results from the Minnesota group.[25] Indeed, they may be underestimated in both the Minnesota and this study because school leavers were followed up less successfully than subjects still in school. The high rates of smoking by early school leavers or dropouts warrant special attention by future research. Prevention programs for youth who will leave or drop out of school will need to go beyond the school setting. Early cessation programs also need to be developed for this group, and these will require components designed to motivate young adults to consider quitting, something which most current smoking cessation programs do not include.

Additional studies of the long-term effects of social influence prevention interventions are necessary. We cannot draw strong inferences from only the two long-term follow-up studies conducted to date. The short-term effects of the social influences approach appeared very promising, but interpretation of their long-term value must await further long-term follow-up studies.

APPENDIX A

Results and Characteristics of Long-term or High School Follow-up Studies on Smoking Prevention

Authors/ Date/Reference	Length of Follow-up (Years)	School Grade of Program	Program Effect (Yes or No)	Methodological Problems (See below)
Evans, *et al*, 1981[4]	3	7	Y	a,b
Telch, *et al*, 1982[5]	2	7	Y	c,d
Johnson, *et al*, 1986[6]	1	10	Y	a,e
Hansen, *et al*, 1988[7]	3	7	Y	a,e
Luepker, *et al*, 1983[8]	2	7	Y	f
Murray, *et al*, 1988[9]	4–5	7	Y	f
Murray, *et al*, in press[10]	5–6	7	N	f

Methodological problems:
a) Serious attrition.
b) Reliance on successive cross-sectional analyses of different subjects.
c) Pretest differences.
d) Unable to follow same subjects over time.
e) Marginal significance.
f) Pretest differences in direction of expected effects.

APPENDIX B

Summary of Content of the First Waterloo Smoking Prevention Program

Session	Components	Purpose	Methods
Core program (Six one-hour sessions)	Informational	Provide factual basis for the rest of the program.	Film Classroom discussion Poster-making Videotapes Categorization
	Skills Development	Develop and practice skills to resist social influences to smoke.	Videotapes Modeling Role playing Poster-making
	Decision-making	Integrate other components and express intentions.	Decision sheet Public commitment to class
Maintenance (same grade)		Reestablish contact Review	Poster-making Classroom discussion "Talking" videotape Categorization
Booster sessions (grades 7 & 8)		Reestablish contact Review Update information and skills	Classroom discussion Small group work Decision sheet Role playing

ACKNOWLEDGMENTS

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Portions of these data were previously presented at the 6th World Conference on Smoking and Health, Tokyo, Japan, October 1987.

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RWJ Foundation Extends Grants Program for Native American Health Projects

The Robert Wood Johnson Foundation recently awarded nearly $2 million to fund 13 community health care projects run by and for American Indians and Alaska Natives, and in a further commitment has allocated additional funds to extend the program through 1990.

Believed to represent the first commitment by a major foundation to work directly with tribal governments, the 1989 grants program will target projects designed to prevent alcohol and drug abuse, control diabetes, reduce domestic violence, and improve maternal and infant health among tribal populations in eight states (AZ, ID, MN, MT, NM, OK, SD, and WI). The tribes plan to approach their health problems with imagination and creativity, using traditional Indian methods of care as well as modern medical technology, according to the foundation.

When the call for proposals was issued last fall for the program, "Improving the Health of Native Americans," the foundation received applications from Indian-related groups in 31 states. The high quality and magnitude of that response prompted the foundation to commit additional funds of up to $2 million to continue the program in 1990.

Tribes and related community organizations interested in applying for funds under the program's second phase should contact Timothy L. Taylor, PhD, Program Director, Improving the Health of Native Americans, College of Public Health, University of Oklahoma Health Sciences Center, PO Box 26901, Oklahoma City, OK 73190. Tel: (405) 271-3221. Dr. Taylor is also a member of the Kiowa Tribe.

Two workshops for potential applicants will be held in November in Nashville, TN and Denver, CO, to answer questions about the program and the application process. For more information about the workshops, contact Dr. Taylor at the address above.

Grants under the program's second phase will be announced in August 1990; proposals must be received by February 1, 1990 to be considered for funding.

The RWJ Foundation, based in Princeton, NJ, is the nation's largest health care philanthropy, established in 1972. Since then, it has awarded more than $996 million in grants to improve health care in the United States.

Elevated Blood Levels of Carcinogens in Passive Smokers

MALCOLM MACLURE, ScD, RONIT BEN-ABRAHAM KATZ, MD, MATTHEW S. BRYANT, PhD, PAUL L. SKIPPER, PhD, AND STEVEN R. TANNENBAUM, PhD

Abstract: The hypothesis that involuntary exposure to tobacco smoke—passive smoking—results in greater risk of cancer was assessed by measuring the levels of two known carcinogens in the blood of 57 nonsmokers with varying degrees of involuntary exposure, including six heavily exposed bartenders. The concentrations of hemoglobin adducts of 4-aminobiphenyl, a bladder carcinogen, were significantly higher in subjects with confirmed involuntary exposure (plasma cotinine concentrations between 2 and 23 ng/ml) compared with subjects with undetectable levels of cotinine. Similarly, adducts of 3-aminobiphenyl were significantly elevated in subjects with confirmed exposure. The odds of 3-aminobiphenyl adduct levels exceeding 2 pg/g of hemoglobin were 6:7 among the confirmed exposed, compared with the odds of 2:42 among subjects with undetectable cotinine (odds ratio = 18; 95 percent confidence interval = 3.3, 94). The validity of the assay was demonstrated by showing striking declines in adduct levels among quitting smokers. (*Am J Public Health* 1989; 79:1381–1384.)

Introduction

Involuntary exposure to tobacco smoke, or "passive smoking," has been found weakly associated with lung cancer in several epidemiologic studies, but several others have found no association.[1–3] Critics argue this evidence is flawed.[4,5] The International Agency for Research on Cancer (IARC) states that: "The observations on nonsmokers that have been made so far are compatible with either an increased risk from 'passive' smoking or an absence of risk. Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during 'passive' smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens . . . leads to the conclusion that passive smoking gives rise to some risk of cancer."[3] Mantel[6] concludes that the relative risk of passive smoking is too low to be detected by epidemiologic studies.

If Mantel is right, assessment of the risk of passive smoking depends on measurement of the extent of absorption of carcinogens from tobacco smoke. No evidence has been reported to date of increased blood levels of a known carcinogen in passive smokers. We collected blood samples from nonsmokers with various degrees of passive exposure, classified them by plasma cotinine detectability, and measured levels of 4-aminobiphenyl and 3-aminobiphenyl covalently bound to hemoglobin. The adducts of these aromatic amines with hemoglobin (4ABP-Hb; 3ABP-Hb) are formed over the 120-day lifespan of the erythrocyte and therefore may serve as dosimeters of average exposure over the previous four months.[7] 4ABP is a known human carcinogen[8] and 3ABP, although not proven to be carcinogenic in humans, is a marker of 4ABP formation by pyrolysis[9] and has been shown carcinogenic in rats.[10]

Methods

Subjects

Nonsmokers were recruited by a variety of methods to cover a range of passive exposures. To represent chronic passive exposure, six heavily exposed nonsmoking bartenders were recruited by a telephone survey of local bars. To represent more normal but quantitatively uncertain levels of passive smoking, 21 nonsmokers who reported exposure to at least one pack of cigarettes per day were recruited by asking colleagues to survey their friends. To represent lack of exposure, 14 nonsmokers who reported negligible passive exposure in the past two months were recruited from our research staffs. Added to this was a group of blood donors in Italy who served as nonsmoker controls for another study.[11] The Italians were not selected according to their degree of passive exposure and therefore were presumed to be more representative of a cross-section of nonsmokers.

Exposure

Local volunteers, after completing a consent form, donated 10–20cc of blood, which was drawn into a vacuum tube containing EDTA (ethylenedinitrilotetraacetate) as anticoagulant and immediately placed in a cooler. Using a short standard questionnaire, subjects were interviewed about their exposure to tobacco smoke during the past two months. Questions included numbers of smokers in the work and home environment, and estimated numbers of packs of cigarettes per day smoked in their presence. Information on involuntary exposure to smoke was not available from of the Italian blood donors.

Plasma cotinine was measured by the laboratory of N.J. Haley of the American Health Foundation, Valhalla, NY. Four subjects were excluded for having cotinine levels that were suspicious of active smoking[12]: One local volunteer who claimed only passive exposure had 240 ng/ml of cotinine, and three Italians who had reported being nonsmokers had 61, 97, and 101 ng/ml of cotinine in their plasmas. The remaining subjects were divided into two groups: 1) undetectable cotinine, and 2) detectable cotinine. (The limit of detection of the cotinine assay was 2 ng/ml.) These two groups were then subdivided according to the level of exposure they reported in their interviews, as follows.

Group 1a (n = 14): Research workers; no cotinine; reported little or no passive exposure.

Group 1b (n = 15): Italian blood donors; no cotinine; no interview about passive exposure.

Group 1c (n = 15): Local volunteers, no cotinine; reported passive exposure to at least one pack of cigarettes per day.

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Group 2a (n = 7): Local volunteers; measurable cotinine; reported passive exposure to at least one pack of cigarettes per day, plus one Italian blood donor with measurable cotinine.

Group 2b (n = 6): Local bartenders; measurable cotinine; reported passive exposure to five or more packs of cigarettes per day.

Group 1b was ranked between Groups 1a and 1c because it presumably comprised a mixture of people who would have been assigned to Groups 1a or 1c if they had been interviewed.

Adduct Assay

The methods of sample preparation and analysis for determining the concentration of 4ABP adducts with hemoglobin have been described in detail.[13,14] In brief, hemoglobin was isolated from red blood cells and purified by dialysis. The dialysate was spiked with a nanogram of an internal standard (4'-fluoro-4-aminobiphenyl) and then hydrolysed with dilute base to release free 4ABP and 3ABP from the adducts. The amines were extracted into purified hexane and then derivatized to form the corresponding pentafluoropropionamides. These were quantified using capillary gas chromatography with detection by negative-ion chemical ionization mass spectrometry. The average limit of detection of the assay for 3ABP was 1.0 pg/g of Hb, calculated as the quantity of adduct necessary to produce a peak intensity three times the root mean square of the individual chromatogram's baseline noise level.

The precision of the assay was assessed by splitting two 100 ml blood samples into 8 and 7 aliquots.[14] The coefficient of variation of the estimates of 4ABP-Hb concentration, expressed in pg/g of Hb, was 10 percent for each sample. In addition, from 15 people with varying levels of 4ABP-Hb, duplicate blood samples were collected 15 or 48 hours apart. The coefficient of variation in 4ABP-Hb adduct concentration among duplicates was 9 percent, entirely attributable to variation in the assay technique. This suggested day-to-day biologic variation in adduct levels was negligible.

Validation Studies

To demonstrate the validity of the level of the hemoglobin adducts as dosimeters of human exposure, we recruited seven quitting smokers and measured adduct levels in blood samples drawn on the day they entered the stop-smoking program, and two months or more later. Serum cotinine was used to confirm that all seven had successfully quit. Further validation data were found in the results of a cross-sectional study conducted in Italy in which the dose-response relationship between intensity of smoking and adduct level was examined.[11]

Data Analysis

Since the distributions of adduct levels among passive smokers and unexposed nonsmokers were found to be skewed, nonparametric tests were used. The median two-sample test was the preferred basis for inference because it is relatively insensitive to outliers.[15] The Wilcoxon two-sample test was also used.[15] One-sided p-values were used because the hypotheses were directional.

Subjects with undetectable levels of 3ABP-Hb were handled in two ways. First, the subjects were assigned the highest level of 3ABP that could have gone undetected, given the noise level of each chromatogram. Second, the subjects were omitted from the analysis.

Results

Validity

We found striking reductions in adduct levels among quitting smokers. The mean level of 4ABP-Hb fell 75 percent from 130.4 pg/g Hb (s.e. = 35.1 pg/g Hb) at baseline to 33.3 pg/g Hb (s.e. = 7.3 pg/g Hb) after 2 months. The mean level of 3ABP-Hb fell at least 80 percent from 16.0 pg/g Hb (s.e. = 7.2 pg/g Hb) to a baseline of approximately 1.7 pg/g Hb (s.e. = 1.3). Only three of the seven subjects contributed to the estimate of the average final level of 3ABP-Hb because concentrations in the other four subjects were below the limits of detection of the assay.

The cross-sectional study of Italian smokers[11] showed dose-response relations existed between number of cigarettes smoked per day and levels of both 4ABP and 3ABP.*

Passive Smokers

The characteristics of each subgroup and the median and mean levels of 4ABP-Hb and 3ABP-Hb are shown in Table 1. The individual levels of 4ABP-Hb and 3ABP-Hb are plotted in Figures 1 and 2.

Levels of 4ABP-Hb were marginally higher in Group 2 than in Group 1 (Figure 1). The difference was of borderline statistical significance: the median two-sample test statistic was 1.64 (p = 0.05). The Wilcoxon two-sample test statistic was only 1.08 (p = 0.14) due to several atypically high values in Group 1. The highest value (152 pg/g) was from a woman who claimed passive exposure to 10 cigarettes per day but had no detectable cotinine in her blood. The next two highest values were from women in Group 1a. One woman with 89 pg/g confirmed on reinterview that she had had absolutely no

*Data available on request to author.

TABLE 1—Levels of 4-Aminobiphenyl and 3-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers with and without Detectable Serum Cotinine, According to Level of Reported Passive Smoking

Group	Detectable Cotinine	Reported Passive Smoking	Number of Subjects	Mean Age	Percent Male	4-Aminobiphenyl*			3-Aminobiphenyl*†		
						Median	Mean	(S.E.)	Median	Mean	(S.E.)
1a	No	None	14	37	43	40	50	24	1.0	1.3	1.5
1b	No	—	15	39	100	37	42	15	1.0	1.2	0.6
1c	No	Yes, Low	15	28	26	38	46	31	1.2	1.2	0.7
2a	Yes	Yes, Low	7	32	86	42	45	14	0.7	1.5	1.3
2b	Yes	Yes, High	6	31	83	48	54	21	2.6	2.4	1.1

*Picograms of amine per gram of hemoglobin.
†Minimum detectable levels were used for subjects with undetected levels.

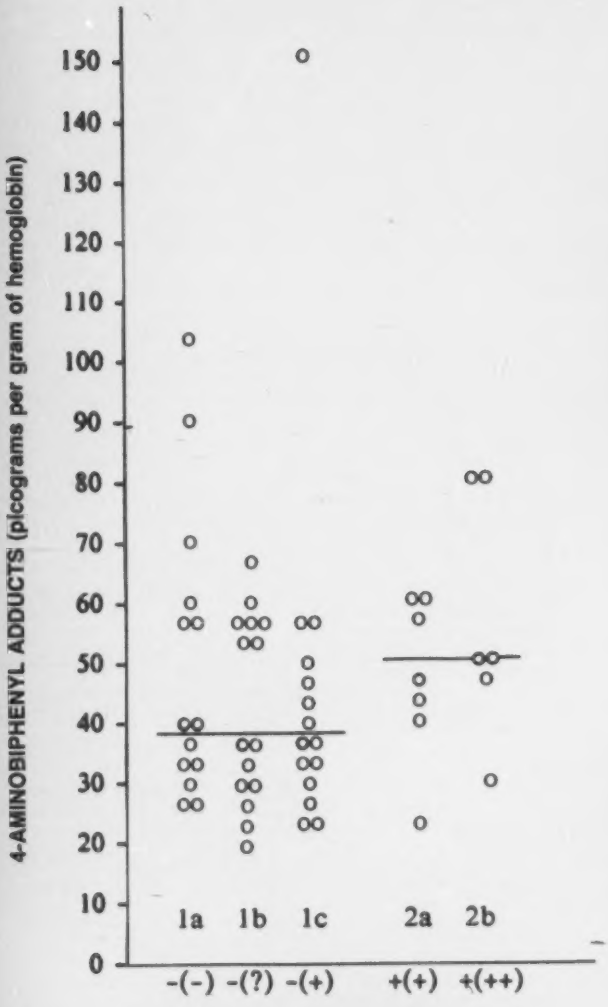

FIGURE 1—Levels of 4-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers without (1a, 1b, 1c) and with (2a, 2b) Detectable Serum Cotinine, Indicated by − and + Signs.
Symbols in parentheses refer to self-reported level of passive exposure to tobacco smoke: (−) none; (?) unknown; (+) low; (++) high. Horizontal lines indicate locations of group medians.

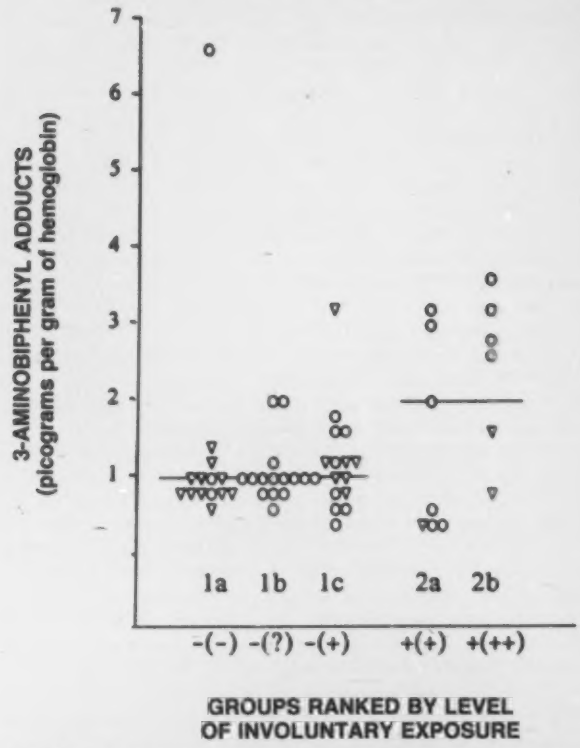

FIGURE 2—Levels of 3-Aminobiphenyl Adducts with Hemoglobin in Groups of Nonsmokers without (1a, 1b, 1c) and with (2a, 2b) Detectable Serum Cotinine, Indicated by − and + Signs.
Symbols in parentheses refer to self-reported level of passive exposure to tobacco smoke: (−) none; (?) unknown; (+) low; (++) high. Horizontal lines indicate locations of group medians. Triangles represent undetectable levels of adduct and are plotted at the highest level the subject could have had without detection.

passive exposure. Another, with 103 pg/g, reported only occasional mild exposure for short periods. Increasing the size of the control group by adding the seven exsmokers from the validation study resulted in a mean two-sample test statistic of 1.54 ($p = 0.06$) and a Wilcoxon two-sample test statistic of 1.41 ($p = 0.08$).

A clearer difference in the hypothesized direction was found for 3ABP-Hb levels (Figure 2). Group 2 had significantly higher levels than Group 1. The median two-sample test statistic was 1.92 ($p = 0.027$) and the Wilcoxon two-sample test statistic was 1.22 ($p = 0.11$), using the lowest detectable level for subjects with undetectable adduct levels. When undetectable levels were omitted, the median two-sample test statistic was 2.10 ($p = 0.017$) and the Wilcoxon two-sample test statistic was 1.65 ($p = 0.05$). The odds of a subject with detectable cotinine having a level of 3ABP-Hb greater than 2 pg/g of Hb were 6:7, compared with 2:42 among subjects with undetectable cotinine. The odds ratio was 18 with a 95 percent confidence interval from 3.3 to 94.

Discussion

Our observations corroborate the hypothesis that involuntary exposure to tobacco smoke results in higher levels of carcinogens in the blood. Subjects with plasma cotinine concentrations between 2 and 23 ng/ml had significantly higher concentrations of 4ABP-Hb and 3ABP-Hb than subjects with undetectable levels of cotinine.

The association with 4ABP-Hb was of only borderline statistical significance. However, it is less likely to be due to chance than the p-values indicate because the significant association with 3ABP-Hb provides additional evidence against the null hypothesis. It is well established that 3ABP forms in the same pyrolysis reactions as 4ABP.[9] Consistent with this, our validation study showed that 4ABP-Hb and 3ABP-Hb both declined among quitting smokers. Therefore, 3ABP-Hb is in part a marker of 4ABP exposure.

Nevertheless, the magnitude of the elevation in adduct levels was small relative to normal background levels. Non-tobacco sources of 4ABP, and perhaps 3ABP, appear to

exist. This was underscored by the fact that one woman with absolutely no passive exposure to tobacco smoke had levels of 4ABP-Hb higher than all the heavily exposed bartenders. Her level of 3ABP-Hb was undetectable, but another woman with minimal passive exposure had equally high 4ABP-Hb and the highest level of 3ABP-Hb in the study.

Since it is well established that 4ABP causes bladder cancer in humans,[8] our data suggest that risk of bladder cancer among heavily exposed passive smokers is slightly elevated. However, the increase in risk may be too small to detect in an epidemiologic study. A relative risk of 1.2 is regarded by many as the lower limit of detection in nonexperimental epidemiology. It is hard to detect a relative risk of 1.1 not just because of sampling variation but because of uncontrolled confounding. A potential confounding factor weakly associated with disease (relative risk = 1.7) could produce a spurious relative risk of 1.1 for passive smoking if passive smokers were twice as likely as unexposed nonsmokers to be exposed to the confounder.[16] A relative risk of 1.1 corresponds to a 10 percent excess in risk. For passive smoking to cause at least 10 percent excess in risk of bladder cancer, when active smoking causes an excess of only 100 percent (i.e., relative risk = 2), the excess concentration of activated carcinogens in the tissues of passive smokers should be approximately 10 percent of the excess in active smokers (assuming a linear relation between *in situ* dose and risk). In our data, the excess of adduct attributable to passive smoking appeared to be lower than this. Whereas the median level of 4ABP-Hb adducts in active smokers was found to be 200 percent to 400 percent in excess of the median level in unexposed nonsmokers,[10,13] the median level among passive smokers was only 10 percent to 20 percent in excess (i.e., the excess in passive smokers was 2.5 percent to 10 percent of the excess in active smokers).

Since our data are few, especially among confirmed passive smokers, and the assumption of linearity between adduct levels and risk is hypothetical, firm conclusions cannot be drawn. However, when compared with data that indicate cotinine levels in passive smokers are generally 1 percent, and at most 2–10 percent, the levels in active smokers,[3,17] our data seem more compatible with an undetectable rather than a detectable relative risk of bladder cancer due to passive smoking. One conclusion that does seem warranted, however, is that 4ABP does not conform to Remmer's hypothesis.[18] Remmer suggested that a larger proportion of inhaled carcinogens are metabolically activated in passive smokers, as compared to active smokers, due to the induction of detoxifying enzymes by substances in mainstream smoke that are not found in sidestream smoke. This does not appear to hold for 4ABP, assuming that the adduct with hemoglobin is a marker of the levels of activated 4ABP. There is evidence to support such an assumption.[13,19]

It is important to emphasize that "undetectable" does not mean "nonexistent." Since involuntary exposure to tobacco smoke is very common, even an undetectable relative risk such as 1.1 would be responsible for a great deal of avoidable cancer. The purpose of distinguishing between detectable and undetectable relative risks is for planning research to quantify the risk of passive smoking. If studies like ours show that post-activation levels of other tobacco carcinogens are much lower in passive smokers than active smokers, it will add weight to the view that quantification of the risk of passive smoking is beyond the capability of questionnaire-based epidemiologic studies, except when the association between active smoking and disease is very strong (e.g., relative risk of 10 for lung cancer). Risk assessment may instead depend on biochemical studies of carcinogen metabolites.

In conclusion, our results corroborate both the statement by the IARC that "passive smoking gives rise to some risk of cancer" and critics' contention that relative risks due to passive smoking may be undetectable.

ACKNOWLEDGMENTS

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Delayed Blood Regeneration in Lead Exposure: An Effect on Reserve Capacity

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Abstract: Twenty-five lead-exposed Danish battery production workers and 25-age-matched controls were examined to evaluate subclinical effects on blood formation. Blood lead levels averaged 2.14 μmol/L and 0.35 μmol/L in the two groups; the lead workers also showed high levels of erythrocyte protoporphyrin, as compared to the controls. Otherwise, the hematological parameters indicated an appropriate iron status and no other deviations.

From all subjects, 0.45 L of blood was bled as part of a normal blood donation. Five and 11 days later, reticulocyte counts were significantly higher in the control group than in the lead-exposed workers. On day 15, the lead workers showed a significant delay in blood regeneration, as evidenced by lower hemoglobin concentration, and erythrocyte and reticulocyte counts.

The lead exposure in the present study was within legal limits, and lead-induced anemia would be expected only at much higher exposure levels. Thus, despite the normal hematological findings in the initial examination, the lead exposure caused a decreased reserve capacity for blood formation, and this effect became evident only after the blood loss. (*Am J Public Health* 1989, 79:1385–1388.)

Introduction

Although lead is one of the most extensively studied pollutants, the health significance of early hematological changes at slightly increased exposures remains unclear. Thus, heavy lead exposures are associated with low hemoglobin or anemia.[1,2] However, when blood-lead levels do not exceed 2.9 μmol/L, hemoglobin concentrations in male lead workers are usually entirely within normal ranges.[1–3] Despite the apparent absence of hematological abnormalities, such lead exposures reduce the lifespan of the erythrocytes and inhibit the heme biosynthesis.[4,5] These biochemical effects in a lead-exposed worker could place an increased strain on the bone marrow to keep the red-cell values within normal ranges.

Therefore, increased demand on the formation of blood following blood loss could result in a delayed blood regeneration in individuals exposed to lead. Only one experimental study has examined this question: dogs exposed to lead in the feed showed no delay in blood regeneration after a controlled bleeding.[6] However, lead exposure caused an unanticipated increase in the pre-hemorrhage hemoglobin as compared to controls in this study, and dogs may well differ from humans in their reactions to lead.

We have examined the blood regeneration following phlebotomy in men with occupational lead exposure and an age-matched control group.

Methods

Sample and Research Design

Based on a review of occupational lead exposures at nearby industrial facilities, two plants were selected for study: a battery factory with high lead exposures, and a tin can factory with minimal lead exposures. The safety committees at the two facilities helped identify possible volunteers among the long-term employees. Detailed information about the study was given to each participant. At each facility, informed consent was obtained from 25 healthy men who had never been blood donors before. The median duration of occupational lead exposure at the battery plant was seven years. The comparison group at the tin can factory was age-matched (within five years) with the participants at the battery plant. The median age in the exposed and the control groups was 39 and 36 years, respectively. The median length of employment in the control group was 10 years. Information on iron intake was not included in the matching criteria.

Phlebotomy (0.45 L) was performed in cooperation with the Blood Bank at the University Hospital. Due to practical constraints, the two groups went through the procedures at two different points of time; strict quality assurance efforts were therefore applied to ensure comparability of the results. Blood lead (B-Pb), erythrocyte Zn-protoporphyrin (ZPP) and indicators of iron status were determined only in samples obtained at the time of phlebotomy. Routine hematological parameters were subsequently assessed at days 5, 11, and 15 after phlebotomy; at day 15, signs of considerable blood regeneration would be expected.[7–10]

The protocol was prepared in accordance with the Helsinki II Declaration and reported to the local ethics committee.

Blood Lead

Blood samples for lead analysis were taken in Vacutainer vials with Na-heparin (dark-blue stoppers) (Becton-Dickinson, Rutherford, NJ) under conditions minimizing the risk of sample contamination. The samples were frozen at −80°C until analysis. B-Pb analysis was carried out by electrothermal atomic absorption spectrometry.[11] We used a Perkin-Elmer model 5000 instrument with Zeeman background correction, graphite furnace HGA-500, and autosampler AS-40 (Perkin-Elmer, Norwalk, CT). All results were read against a blood-based standard curve with standard reference material SRM 955 from the US National Bureau of Standards as primary standard. The total analytical imprecision was estimated to be 8.1, 2.1 and 1.9 percent at blood lead levels of 0.24, 1.63 and 2.96 μmol/L, respectively. Participation in international external quality assurance schemes showed deviations from the overall median between −8 and +11

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percent for 14 blood samples, thus indicating a satisfactory accuracy.

Hematology

Hematological parameters were determined on fresh samples drawn in Venoject vials with Na_2EDTA (Venoject, Terumo Europe, Leuven, Belgium); special quality control procedures were used to minimize day-to-day variation.

ZPP was measured in oxidized blood on an Aviv hematofluorometer (Aviv, Lakewood, NJ).[12]

Erythrocyte count and hemoglobin concentration were determined on a Coulter Counter S using Coulter reagents (Coulter Electronics, Hialeah, FL). The instrument was whole-blood calibrated regularly and prior to each analytical series, and the calibration was controlled using the Coulter 4C Normal calibration material. Results for erythrocyte count and hemoglobin were accepted if within 2.7 percent and 2.6 percent, respectively, of the values assigned. Hematocrit was measured in duplicate by centrifugation (Sigma 2M, Christ, Osterode, Federal Republic of Germany) at 15,000 × G for 5 min using 75 mm borosilicate capillary tubes (Houmsen, Risskov, Denmark); the hematocrits were read with an Adams Micro-Hematocrit reader (Clay Adams, Becton-Dickinson, Parsippany, NJ) according to the NCCLS approved standard.[13] Mean cell volume and mean cell hemoglobin content were calculated from the erythrocyte count, hematocrit, and hemoglobin results.

Reticulocytes were counted in unfixed blood films following supravital staining with brilliant cresyl blue (1.5 g/L). Results are given as number of reticulocytes per 1000 erythrocytes.[14]

Serum iron (Fe^{3+}) was determined on a Prisma multichannel analyzer (New Clinicon, Stockholm, Sweden) at 600 nm after complex formation with 2,4,6-tri(2-pyridyl)-1,3,5-triazine following release from transferrin by hydrochloric acid and reduction by ascorbic acid.[15] We found a total coefficient of variation of 4 percent at an average of 31 μmol/L; the 95 percent reference interval using this method is 9–35 μmol/L.

Serum ferritin was measured by a sandwich radioimmunoassay (Bio-Rad, Richmond, CA) using an NE 1600 gamma-counter (Nuclear Enterprises, Edinburgh, Scotland). The total coefficient of variation was 6.2 percent at 366 pmol/L, 6.1 percent at 64.6 pmol/L and 15.0 percent at 13.5 pmol/L; the lower limit of the 95 percent reference interval is given by the manufacturer as 44 pmol/L for males below 45 years and 100 pmol/L for males above that age.

Serum transferrin and haptoglobins were determined by immunoturbidimetry in a Cobas Fara centrifugal analyzer (Hoffmann-La Roche, Basle, Switzerland).[16] We used antibodies from Dakopatts (Copenhagen, Denmark). For transferrin, the total coefficient of variation was 3.1 and 2.4 percent at mean values of 32.2 and 49.1 μmol/L, respectively; the 95 percent reference interval using this method is 24.5–39.0 μmol/L. For haptoglobins, the total coefficient of variation was 3.4 and 3.9 percent at mean values of 1.16 g/L and 1.80 g/L, respectively; the 95 percent reference interval for healthy individuals is 0.35–2.40 g/L.

Results

Lead Exposure

All B-Pb levels at the battery factory (Table 1) were higher than those seen at the control facility, but only one was above 2.9 μmol/L, a legal limit used in several countries. However, the median was above a proposed health-based limit of 1.9 μmol/L.[4] The ZPP levels overlapped only slightly between the two groups (Table 1), thus confirming the increased lead exposure at the battery factory and the expected interference with the heme biosynthesis.

Hematological Status

The hematological parameters showed minimal differences between the two groups (Table 1). The lead-exposed workers showed a slightly lower average haptoglobin level, and somewhat lower reticulocyte counts than did the controls. A few individuals in both groups showed high ferritin levels. Thus, in general, the hematological parameters indicated essentially normal conditions. Most importantly, no difference was seen in iron status between the two groups.

Effects of Phlebotomy

On days 5 and 11, the hematological parameters reflected the recent blood loss in both groups. In particular, the reticulocyte counts began to increase. However, on both occasions, the lead-exposed workers showed a significantly lower number of reticulocytes, as compared to the controls (Figure 1). Otherwise, no significant differences were seen between the two groups.

On day 15 after blood donation, both groups still showed significantly decreased hemoglobin, hematocrit and erythro-

TABLE 1—Indicators of Lead Exposure and Hematological Parameters in 25 Lead-Exposed Workers and 25 Age-Matched Controls at the Time of Phlebotomy[a]

Indicators	Exposed Workers		Control Workers		
	Median	Range	Median	Range	P value
Blood lead (μmol/L)	2.14	0.93–3.59	0.35	0.13–0.84	0.000
ZPP (μmol/mol Hb)	108	34–429	29	13–51	0.000
Hemoglobin (mmol/L)	9.3	8.5–10.0	9.2	8.5–10.3	NS
Hematocrit ($\times 10^{-2}$)	44.5	41.0–48.0	44.7	41.0–47.5	NS
Erythrocytes ($\times 10^{12}$/L)	4.93	4.27–5.20	4.88	4.42–5.59	NS
Reticulocytes ($\times 10^{-3}$)[b]	7	2–27	10	0–18	NS
Mean cell volume (fl)	91	86–102	92	82–106	NS
Mean cell hemoglobin (fmol)	1.88	1.80–2.13	1.91	1.67–2.07	NS
Iron (μmol/L)	18	13–34	19	8–41	NS
Transferrin (μmol/L)	32.7	28.5–40.1	32.5	17.8–39.4	NS
Ferritin (pmol/L)	75	28–563	83	15–478	NS
Haptoglobins (g/L)	1.10	0.42–2.69	1.47	0.55–2.80	0.11

[a]Probability was calculated by Mann-Whitney U-test. NS denotes not significant.
[b]Number per 10^3 erythrocytes.

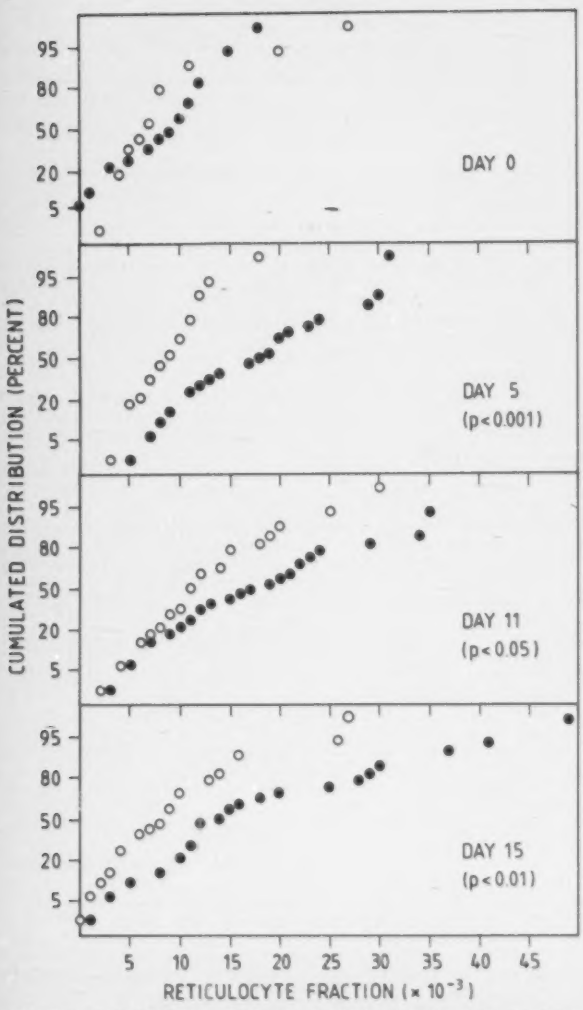

FIGURE 1—Cumulated Distribution (probability scale) of Reticulocyte Counts (per 1000 erythrocytes) at the Time of Phlebotomy (day 0), and 5, 11, and 15 Days Later (open circles, lead-exposed workmen; filled circles, age-matched controls).

cyte count, as compared to the day of phlebotomy. However, the decrease in hemoglobin and erythrocyte count was now significantly larger in the lead workers as compared to the control group ($p < 0.01$). As a further indication of the difference in blood regeneration activity, the increase in reticulocyte counts from day 0 to day 15 was less in the lead workers than in the controls (upper and lower panel of Figure 1).

When comparing the hematological data of the two groups at day 15, the high-lead group had significantly lower hemoglobin, and erythrocyte and reticulocyte counts, as compared to the controls (Table 2 and lower panel of Figure 1). No difference was seen in the hematocrit, mean cell volume, or mean cell hemoglobin.

Discussion

The cross-sectional data at the time of phlebotomy confirmed previous findings[1–3] that hemoglobin levels are within normal limits in male workers exposed to lead below the legal B-Pb limit of 2.9 μmol/L. The slightly decreased haptoglobin levels could be related to a lowered erythrocyte lifespan, as decreases in this parameter most often reflect hemolysis or dyshemopoiesis.[17] No significant tendency toward lower mean cell volume was found in the lead-exposed workers, although anemia in lead poisoning may be of the microcytic type.[4,5]

TABLE 2—Hematological Parameters in 25 Lead-Exposed Workers and 25 Age-Matched Controls 15 Days after Phlebotomy[a]

	Exposed Workers		Control Workers		
	Median	Range	Median	Range	P value
Hemoglobin (mmol/L)	8.4	7.7–9.4	8.7	7.8–9.8	0.006
Hematocrit ($\times 10^{-2}$)	42.0	38.0–46.3	41.8	39.0–46.0	NS
Erythrocytes ($\times 10^{12}$/L)	4.38	4.04–4.77	4.58	4.11–5.23	0.020
Reticulocytes ($\times 10^{-3}$)[b]	9	0–27	14	1–49	0.003
Mean cell volume (fl)	93	82–106	92	84–101	NS
Mean cell hemoglobin (fmol)	1.89	1.82–2.11	1.92	1.72–2.15	NS

[a]Probability was calculated by Mann-Whitney U-test. NS denotes not significant.
[b]Number per 10^3 erythrocytes.

The blood donation appeared to have similar effects on the two groups, except that the lead workers exhibited almost no increase in the reticulocyte count on day 5 and later lagged behind in reticulocyte counts. This deficient response to the blood loss is unexpected, because heavy lead exposure is known to cause an increase in reticulocyte counts, presumably as a result of the continuously increased demand on blood formation.[4,5]

Two weeks after phlebotomy, indications of increased blood formation should be apparent;[7–10] the findings in the control group concur with this pattern. However, the lower hemoglobin and erythrocyte count in the lead-exposed workers suggested delayed blood regeneration. No difference was detected in the hematocrit determination, perhaps due to the increased mean cell volume in the lead workers 15 days after phlebotomy. Although small, the differences in hemoglobin and erythrocyte count are considered valid, as comparability was ensured by vigorous quality control. Also, a delay in blood regeneration is in accordance with the slow increase in reticulocyte numbers seen in the lead workers.

Phlebotomy may be viewed as an artificial situation, but the half-liter blood loss is a reproducible intervention resulting in an increased demand on blood formation similar to, though probably less extensive than, the demand seen in a variety of medical conditions with excess blood loss. Also, this blood loss in blood donors represents an acceptable type of intervention that is known to cause no significant risk.

The present study of blood regeneration indicates that the reserve capacity was reduced in the lead workers despite the fact that minimal hematological changes were apparent before phlebotomy. In general, the level of erythropoiesis will depend on the marrow's capacity to proliferate, on erythropoietin stimulation, and on iron supply.[18,19] In the lead-exposed workers, the most important limiting factor in the rate of blood production after blood loss is likely to be the amount of available iron. As lead interferes with the utilization of iron in the bone marrow,[4,5] insufficient iron may be biochemically available for increased blood formation, despite a normal iron status. None of the subjects used iron supplements, and possible differences in dietary iron intake after blood donation would be unlikely to cause changes in iron status large enough to explain the differences in hemo-

globin and erythrocyte count. The biochemical indications of deranged heme biosynthesis and of decreased red-cell survival seen in lead-exposed workers would therefore appear to have physiological significance.

The decision whether or not a chemically induced change has any health significance is often difficult. However, decrements in reserve capacity would generally be regarded an adverse effect that should be prevented. Wide agreement seems to prevail on this point, despite international differences in preventive strategies, as evidenced, for example, by occupational exposure limits. Thus, according to a report from the US National Academy of Sciences, a nonadverse effect of a chemical exposure must not result in impairment of functional capacity or the ability to compensate for additional stress.[20] Similarly, in the Soviet Union, effects are regarded adverse or abnormal if departures from the generally accepted "normal" values become manifest under functional or biochemical stress.[21–23] A World Health Organization study group also expressed the need to protect against decrements in homeostasis or decreases in functional capacity and therefore initiated an effort to recommend health-based limits for occupational exposures.[4]

Unfortunately, studies on reserve capacity are few, and this lack of information hampers the detailed evaluation of chemical hazards. Thus, the theoretical approaches to prevention should inspire appropriate experimental and epidemiological studies to explore more fully how chemical exposures may interfere with homeostatic and compensatory mechanisms. With the multicausal etiology of many diseases, such subclinical effects of numerous environmental and occupational exposures may well be of importance. The present study has illustrated the possible significance of this concept.

ACKNOWLEDGMENTS

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OSHA awards $1 million in Job Safety and Health Education Grants

Secretary of Labor Elizabeth Dole recently announced that grants totaling $1 million have been awarded to eight labor and four employer nonprofit associations to continue developing job safety and health training and education programs. Since this "New Directions" effort began in 1978, OSHA has awarded approximately $81 million in job safety and health grants to 202 organizations.

Information about the "New Directions" program is available from the Occupational Safety and Health Administration Training Institute, 1555 Times Drive, Des Plaines, IL 60018. Tel: (312) 297-4810.

The Expense of Testing in a Teaching Hospital: The Predominant Role of High-Cost Tests

ROBERT M. HARTLEY, MD, MSc, MIRIAM A. MARKOWITZ, MSc, AND ANTHONY L. KOMAROFF, MD

Abstract: We analyzed all 32,206 tests ordered in a random sample of 1,000 patients admitted to a teaching hospital. The analysis examined costs instead of charges, included professional costs in addition to hospital costs, and considered the effect of combining tests that are ordered as a panel (e.g., cardiac enzymes) or as part of a procedure (e.g., bronchoscopy). High-cost tests (costing more than $100) accounted for only 4 percent of the total number of tests ordered, but for nearly 50 percent of total test costs. The pattern varied by clinical service: high-cost tests accounted for 31, 47, and 51 percents of total test costs on the obstetrics, medicine, and surgery services, respectively. It would seem to be efficient to concentrate on the use of high-cost tests in attempting to foster cost-effective use of diagnostic technologies. (*Am J Public Health* 1989; 79:1389–1391.)

Introduction

Many billions of dollars are spent each year on diagnostic technologies. How this money for diagnostic testing is spent remains unclear. While attention formerly focused on the use of high-cost tests, many have argued recently that the high volume ordering of inexpensive items may constitute the greatest fraction of total diagnostic testing costs.[1,2] Several reports have lent some support to this argument.[3,4] However, these studies have been designed to address other questions and have not attempted to measure directly the actual costs of testing.

Because of the importance of this issue in understanding how large sums of health care dollars are being spent and in choosing the best targets for cost-containment strategies, we have studied the cost of diagnostic testing in a sample of hospitalized patients. Because charges may poorly reflect true costs[5] and professional fees can add substantially to a test's cost, we have differentiated *costs* from *charges* and have examined both *hospital* and *professional* billing in our analysis.

Methods

Patient Sample

The Brigham and Women's Hospital in Boston, Massachusetts is a 720-bed tertiary hospital with an active teaching program. We randomly selected 1,000 patient admissions to the medical, surgical, gynecological and obstetrical services during fiscal year 1982. We included gynecological service patients with surgical patients since, in terms of resource consumption, they were more like surgical than obstetrical patients. Infants born in the hospital were excluded from the sampling frame. All data were obtained from and analyzed by a computerized system (CHASE System) built by one of us (AK), which stores itemized resource utilization data.

Definition of a Test

We defined a "diagnostic test" as any hospital procedure that was requested solely to seek information about a patient's status and which: required some sort of machinery; had been specifically requested in each instance of use; was not performed in a regular operating room; and did not involve continuous monitoring (e.g., central venous pressures) for an undefined time period. Using this definition, we examined all tests ordered in the sample of 1,000 patients: 1,195 different types of tests, ordered on 32,206 occasions.

We arbitrarily defined a high-cost test as one costing $100 or more, and a low-cost test as one costing less than $100.

Hospital Charges and Costs

We chose to estimate costs by two standard methods: ratios of costs-to-charges (RCCs) and relative-value units (RVUs). RVUs were either taken from College of American Pathologists standards, or developed through time-motion studies in our laboratories. RCCs were developed internally through detailed analyses of the laboratory's direct costs: the salaries of all personnel, including the physician laboratory director; supplies and equipment; and allocated expenses from all non-revenue producing cost centers, using a methodology that has been described in detail elsewhere.[6,7]

Analysis revealed no important differences when costs were estimated by the RCC or RVU method. Hereafter, all cost estimates are based upon RCC methodology.

Professional Charges and Costs

Because radiological, medical, and surgical professional groups billed separately for their services, we reviewed their billing data for each of our 1,000 patients. Pathology and cytology services did not bill separately for professional services. However, because professional labor constitutes a major proportion of these services' costs, we identified and separated the professional cost components. Routine hematology and chemistry professional costs were not separated because these professional costs contribute a very small proportion to their respective total test costs.

No reliable measures of true professional costs were available. Of available sources, the 1982 Medicare Part B customary reimbursements appeared to best express the direct costs of physician time and the indirect allocated costs of his or her business. However, because these estimates are likely in some cases to overestimate costs, we have conducted all analyses with and without professional costs

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included. Conclusions about the relative contribution of high-cost items were not substantially affected.

Determination of Unit Cost of Each Type of Test

The unit cost of each type of test included both the hospital cost and, if there was one, the professional cost. In addition we aggregated the costs of several tests when they were ordered simultaneously for the same clinical purpose.

Specifically, this was done in two instances: 1) *Test panels*: whenever several clinically-related tests (e.g., cardiac enzymes or electrolytes) were ordered on the same day; 2) *Procedures*: whenever a diagnostic procedure was performed which of necessity generated a number of different tests (e.g., a lumbar puncture which required measurements of cerebrospinal fluid glucose, protein, cell count, culture, and cytology) in order to complete the diagnostic intervention.

We examined the impact on our conclusions of these aggregations and found that test panels predominantly involved low cost tests which, even when aggregated, remained low-cost items under $100 and mostly under $10. The disaggregation of test panel-related and procedure-related tests did not materially change the ratio of high-cost to low-cost tests. Hence, all results reported involve aggregation of test panel- and procedure-related tests, a process which we believe make most clinical sense.

Results

Patient Sample

Our random sample of 1,000 patients was representative of the more than 25,000 patients (except newborns) admitted to Brigham and Women's Hospital between October 1, 1981 and September 30, 1982; age, sex, distribution by service, length of stay, and mean charges were virtually identical.

Volume and Total Costs

All Tests—For our sample of patients, physicians ordered 32,206 tests, which generated $763,108 in hospital charges, $554,811 in hospital costs, and $121,303 in professional costs. Test volume and costs, per patient-hospitalization, varied greatly across the services, with costs for an average medical admission being nearly 14-fold higher than for an average obstetrical admission (Table 1). The professional fee component of test costs averaged 12–19 percent of total test costs on each hospital service (Table 1).

Low-Cost and High-Cost Tests—Low-cost tests were ordered in high volume, accounting for 96 percent of the number of all tests ordered; 68 percent cost less than $10; 85 percent cost less than $20.

Total hospital test costs had two major peaks which occurred at the opposite ends of the distribution. The low-end peak was accounted for by the high-volume low-cost tests; non-angiographic radiology procedures dominate the mid-range ($100–500 each) and various angiographic investigations primarily account for the high-end peak (greater than $500 each), as shown in Figure 1.

High-cost tests accounted for about 50 percent of total costs; tests costing more than $245 accounted for nearly 30 percent of total costs (Figure 2).

TABLE 1—Test Usage and Costs Per Patient On Each Hospital Service

Hospital Services	Number of Tests	Hospital Test Costs	Professional Test Costs	Total Test Costs
Obstetrics	8.6	$ 85.72	$ 11.70	$ 97.42
Surgery & Gynecology	34.7	636.29	145.33	781.62
Medicine	62.4	1108.97	240.07	1349.04
Average for All Services	32.0	554.81	121.30	676.11

Professional Costs

Professional costs contributed 17.9 percent of total costs. The cumulative distribution of professional costs, plotted according to the total test cost with which it was associated, paralleled that of total costs (Figure 2). In fact, the majority of the professional costs were incurred by tests costing less than $60, indicating that the inclusion of professional costs did not cause high-cost items to become the major contributors to total test costs.

Differences: Medical, Surgical, and Obstetrical Services

Diagnostic testing costs accounted for 14 percent of total hospital costs (professional costs excluded) in the total sample of 1,000 patients: 20 percent on the medical service, 12 percent on the surgical service, and 5 percent on the obstetrical service.

In surgery and medicine, expensive tests made the predominant contribution. High-cost tests accounted for 31, 47, and 51 percent of total test costs on the obstetrical, medical and surgical services, respectively (Figure 3). Tests costing more than $450 accounted for about 20 percent of total test costs for medical and surgical patients but none of the costs for obstetrical patients.

Discussion

Diagnostic testing makes a large contribution to the costs of care for hospitalized patients. Our results indicate that tests costing $100 or more account for one-half of the total diagnostic costs. We made the inherently arbitrary decision to define unit test costs of $100 or greater as "high-cost" because the distribution of total test costs by unit costs (Figure 1) demonstrated a natural break point at this level. The next natural break point occurred at $175, a substantially greater unit cost, and the unit prices for such tests were typically well over $200; it did not seem that most people would use the phrase "low cost" to apply to such tests. It is especially interesting to note the very large contribution to total test costs by tests whose unit costs were greater than $500 (Figure 1).

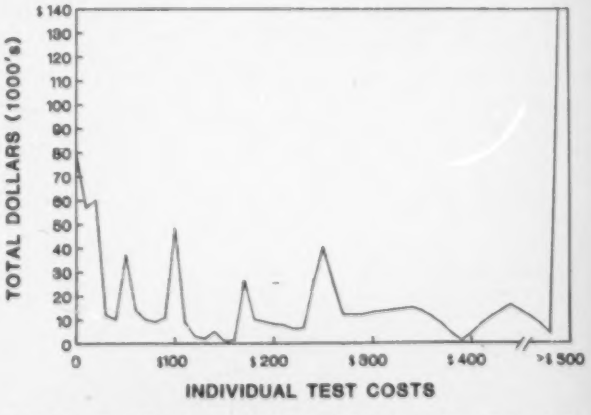

FIGURE 1—Total Test Costs, by Unit Cost of Each Type of Test Ordered, for 1,000 Randomly Selected Patients, 1982.

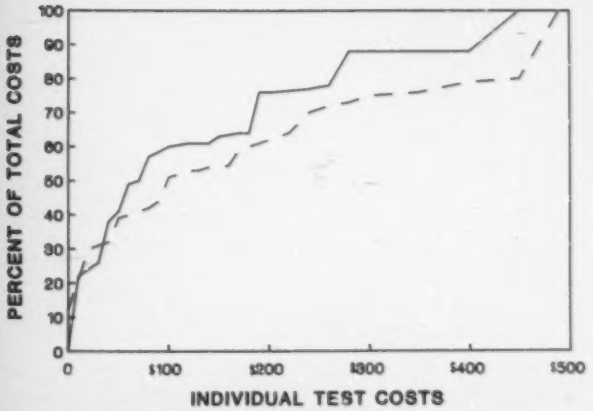

FIGURE 2—Cumulative Distribution of Total Test Costs, and of Professional Costs, by Unit Cost of Each Type of Test Ordered, for all Hospital Resources Utilized, in 1,000 Randomly Selected Patients, 1982. KEY = Solid line is professional cost. Broken line is total test cost.

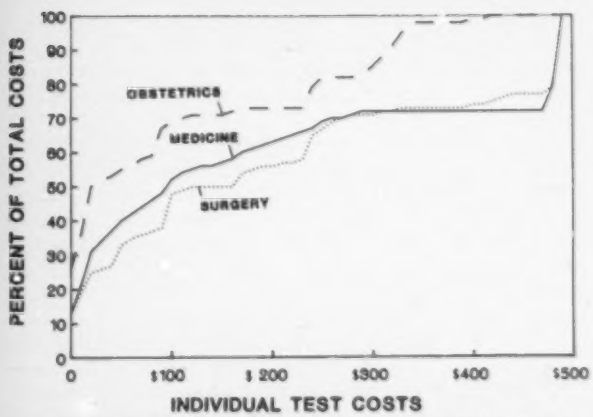

FIGURE 3—Cumulative Distribution of Total Test Costs, by Unit Cost of Each Type of Test Ordered, for each Hospital Resource Utilized, in 1,000 Randomly Selected Patients, 1982.

We believe the distinction we have made between high-cost and low-cost tests is useful for several reasons. First, attempts at control of the use of most high-cost items have traditionally employed regulatory measures directed at the capital expenditures needed for test machinery, an approach of no value for modifying low-cost testing. Second, reported experience suggests that at least one cost containment strategy, organized educational programs, may be less effective in reducing the use of high-cost than low-cost technologies.[4,8] Finally, test-ordering habits vary with test cost. Low-cost tests are often repetitively or reflexly ordered before information is available as to whether they are needed, whereas higher cost tests are usually sequentially ordered depending upon results from preceding investigations.

Our conclusions about the dominant contribution of high-cost items were unaffected by the inclusion or exclusion of professional costs. However, the contribution of professional costs to total test costs is nevertheless substantial. In our study, it represented more than 20 percent of the total for medical and surgical patients. In those situations where the test-ordering physician is the same as the test-performing physician—such as endoscopy or various cardiology procedures—the magnitude of the professional reimbursement represents an incentive for ordering and thus an additional challenge for cost-containment attempts.

Several aspects of our methodology should be noted. Our hospitalized patients were in a teaching center. In such a setting, the test-ordering physician is relatively inexperienced; there is easy access to costly test machinery and procedures; and patients may be more severely ill.[9] For these reasons, our findings may not be generalizable to nonteaching hospitals. On the other hand, we may have underestimated the contribution of high-cost tests, even at a teaching hospital: we did not include as high-cost items several *therapeutic* procedures which have diagnostic components (e.g., laparoscopy, arthroscopy).

While low-cost tests may contribute more in other settings, our results indicate that high-cost tests make an important—in this study, predominant—contribution to the costs of diagnostic testing. Cost containment efforts should target strategies designed to ensure effective use of high-cost tests.

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The Mortality Consequences of Raising the Speed Limit to 65 MPH on Rural Interstates

HERBERT M. BAUM, PHD, ADRIAN K. LUND, PHD, AND JOANN K. WELLS, BA

Abstract: As of April 1987, states were permitted to raise the speed limit on rural interstates to 65 mph without incurring federal sanctions; 38 states elected to do so in 1987. Fatality data for the months when the new limit was in effect in 1987 were compared with fatalities in the same months of 1982–86 on rural interstates and other rural roads. Fatalities on rural interstates in the states with increased speed limits in 1987 were conservatively estimated to be 15 percent higher than they would have been if the states had retained the 55 mph limit (95% CI = 6, 24). Among states that retained the 55 mph limit, fatalities on rural interstates were 6 percent lower than expected (95% CI = −23, 13). (*Am J Public Health* 1989; 79:1392–1395.)

Introduction

Speed limit laws, which date back to 1901, have traditionally been the responsibility of the states. However, in 1974, Congress responded to the oil embargo by directing the US Department of Transportation to withhold highway funding from states that did not adopt the national maximum speed limit of 55 mph (PL 92-239 and 93-643).

The National Research Council (NRC) attributed 4,000 fewer fatalities from 1973 to 1974 to the decreased speeds. Despite some increases in travel speeds since 1974, the NRC concluded that in 1983 the 55 mph speed limit was responsible for approximately 3,000 fewer fatalities, 3,500 fewer serious, severe, and critical injuries, and 47,000 fewer minor and moderate injuries.[1] The NRC estimated that returning the speed limit on rural interstates to their pre-1974 level in all states would result in 500 more fatalities annually, a 20–25 percent increase on these highways.[1]

In 1987, Congress enacted the Surface Transportation and Uniform Relocation Act (PL 100–17), which allowed states to increase the speed limits on rural interstate highways up to 65 mph. By the end of 1987, 38 states had raised their speed limits on rural interstates to 65 mph. Two additional states, Georgia and Virginia, followed suit in 1988. The District of Columbia, Delaware, and Alaska had no eligible miles of highway, leaving eight states with eligible mileage that have not increased speed limits (Figure 1). In December 1987, Congress established a four-year demonstration project that expanded the list of eligible highways to include turnpikes and other highways that meet federal interstate standards. Consequently, Alaska increased the speed limits to 60 mph on some roads, and some other states raised the speed limit on non-interstate rural roads.

Recent observational studies in several states found that, although the previous 55 mph speed limits had been exceeded frequently, the increased speed limits resulted in more motorists traveling at faster speeds.[2] In New Mexico, the percentage of passenger cars exceeding 65 mph went from 37 percent in the first month of implementation to 61 percent in the eighth month; the percentage of passenger cars exceeding 70 mph quadrupled, from 5 percent to 21 percent. In Virginia, the percentage of passenger cars exceeding 65 mph went from 26 percent immediately before the change to 54 percent immediately after, and the percentage exceeding 70 mph went from 7 to 15 percent.[3]

The National Highway Safety Administration (NHTSA) issued a report in January 1989 on the effects of the higher speed limit. NHTSA noted that it was too early to assess the long-term impact of the new speed limit on highway safety. However, the most sophisticated of the report's analyses (a regression model that controlled for fatalities on other roads, and the change in rural interstate mileage, for each year since 1975) indicated that rural interstate fatalities were 16 percent higher than expected.[4]

An American Automobile Association (AAA) sponsored analysis of the effect of the 65 mph limits in Indiana showed no effect on fatalities.[5] However, individual state analyses can often be misleading because the number of crashes on rural interstates are few and the statistical variation associated with these counts can be quite large. In Indiana, for example, there are about 90 fatalities annually on rural interstates. By chance, this count would vary by as much as 20 percent (the standard error of a Poisson distribution with a mean of 90 is ± 9.5). Further, one month after the speed limit was raised, Indiana's mandatory seat belt use law went into effect, and the AAA analysis did not adequately address this change.

This paper presents analyses of fatalities on highways with increased speed limits for the 38 states that raised limits in 1987. By including all these states, the analysis minimizes the problem of statistical unreliability inherent in individual state analyses. In addition, to help control for other contemporaneous changes that might affect fatal crashes on rural interstates, the analysis also examined the fatal crash experience on other roads that might be expected to respond

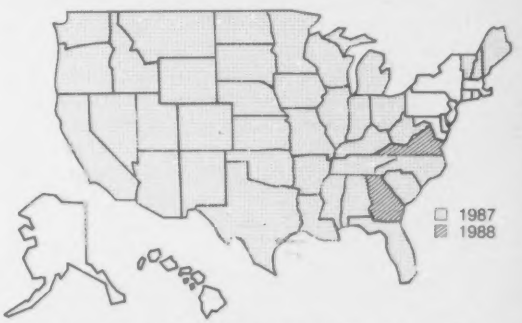

FIGURE 1—States with 65 MPH Speed Limits on Rural Interstates

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similarly to many of these changes. Fatality data for the months of higher limits in 1987 were compared to the average for the same months during the previous five years.

Method

Fatality data are from the Fatal Accident Reporting System (FARS), the federal data base of detailed reports on all fatal motor vehicle crashes on public roads in the United States.[6] States enacted the higher speed limits on various dates throughout 1987 (Table 1). As shown in the table, fatalities on rural interstates in 1987 were counted for each state for all months following the month in which the speed limit was raised to 65 mph. For example, in Alabama where the increased limit became effective on July 20, fatalities were included for August through December. The month in which the change occurred was excluded because of possible delays in posting signs. Rural interstate fatality counts were also obtained for the same calendar months in each of the previous five years, 1982–86. Both overall fatality counts and counts of fatalities among occupants of passenger vehicles (cars, light trucks, and vans) were examined.

Under the 1987 law, a state can post a 65 mph limit on a "highway on the interstate system located outside of an urbanized area of 50,000 or more persons." This definition of eligible highways does not correspond exactly with the highway designations used in FARS; all interstates in FARS that are coded as rural meet the Act's definition, but a small percentage of FARS designated urban highways also meet the definition. However, because 96 percent of the interstate highway miles eligible to be posted at 65 mph are rural interstates as defined by FARS, and 97 percent of these eligible miles were posted at higher limits, these highways were used to represent those with 65 mph limits.[4] An alternative way to count fatalities was to tabulate using the actual posted speed limit recorded in FARS to indicate eligible highways; however, this approach would have precluded comparisons prior to 1987.

Because motor vehicle crash fatalities fluctuate from year to year for a variety of known and unknown reasons, changes observed on rural interstates might be due to other trends in motor vehicle crashes. Therefore, fatality counts on rural interstates were compared to those on all other rural roads.

The odds that a fatality occurred on a rural interstate road (versus all other roads or all other rural roads) were compared to the same odds averaged over the previous five years (1982–86). The 95 percent confidence limits for this odds ratio were computed by methods given in Fleiss.[7]

The fatality experience of states that increased the speed limit was not compared directly to the experience of states that retained the 55 mph limit. As shown in Figure 1, most states that retained the 55 mph limit are in the densely populated northeast and differ on a number of factors that could affect traffic fatalities. In addition, the fatality experience of these states may have been affected by the spillover of higher speeds from states that changed, thereby making a direct comparison inappropriate. Nevertheless, the fatality experience of the states that retained 55 mph limits was examined for any evidence that changes in the 65 mph states were simply part of a larger national trend. Odds ratios, similar to those for the 38-state analysis, were constructed using data from eight states (Connecticut, Georgia, Maryland, Massachusetts, New Jersey, New York, Pennsylvania, and Virginia) that had 55 mph limits and at least 25 miles of rural interstate highway. Fatalities were counted only during the months of May through December to simulate a speed limit change in April of 1987. If there were no larger national trend in rural interstate fatalities in 1987, this odds ratio would be expected to equal unity.

TABLE 1—Effective Dates for 65 mph Speed Limit and Months of Data Included in Analysis, 38 States

State	1987 Effective Date	Analysis Months
Alabama	July 20	August–December
Arizona	April 15	May–December
Arkansas	April 20	May–December
California	May 28	June–December
Colorado	April 6–10	May–December
Florida	April 27	May–December
Idaho	May 2	June–December
Illinois	April 27	May–December
Indiana	June 1	July–December
Iowa	May 12	June–December
Kansas	May 14	June–December
Kentucky	June 8	July–December
Louisiana	April 8	May–December
Maine	June 17	July–December
Michigan	November 29	December
Minnesota	June 17	July–December
Mississippi	April 14	May–December
Missouri	May 1	June–December
Montana	April 16	May–December
Nebraska	April 27	May–December
Nevada	April 13	May–December
New Hampshire	April 16	May–December
New Mexico	April 2	May–December
North Carolina	August 10	September–December
North Dakota	April 16	May–December
Ohio	July 15	August–December
Oklahoma	April 6	May–December
Oregon	September 27	October–December
South Carolina	July 15	August–December
South Dakota	April 15	May–December
Tennessee	May 8	June–December
Texas	May 9	June–December
Utah	May 21	June–December
Vermont	April 21	May–December
Washington	April 20	May–December
West Virginia	April 20	May–December
Wisconsin	July 17	August–December
Wyoming	May 19	June–December

Results

The numbers of fatalities on rural interstates during the months with higher speed limits in 1987 and the same months in 1982–86 for the 38 states with changed limits are shown in Figure 2. This figure shows that fatalities on rural interstates posted with 65 mph limits in 1987 were considerably higher than in any of the previous five years.

Although the number of fatalities on rural interstates increased slightly over the five baseline years, a similar trend was also found for the control series of fatality counts on other rural roads. The yearly odds of fatalities on rural interstates compared to other rural roads remained fairly constant until 1987. Specifically, for 1982 through 1986, the ratios were 0.097, 0.106, 0.109, 0.107, and 0.100, respectively.

There were 19 percent more fatalities on rural interstates in 1987 than the average for the previous five years, while there were only 4 percent more fatalities on other rural roads. For all fatalities, the odds of death on rural interstates versus other rural roads were 15 percent (95% CI = 6, 24) greater in

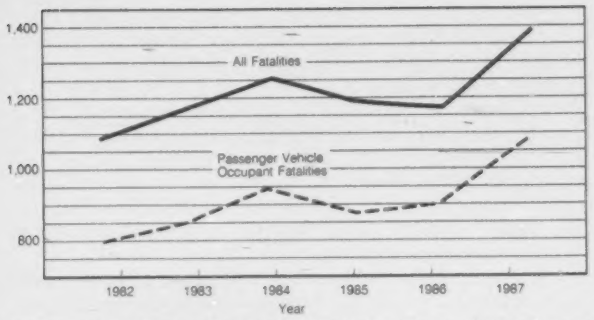

FIGURE 2—Fatalities on Rural Interstates during Months of Higher Speed Limits in 1987 and Same Months in 1982–86

1987 than the average for 1982–86 (Table 2). If *all* other roads are used for the comparison, the ratio yields a 16 percent effect.

The odds ratio for passenger vehicle occupant deaths showed a similar increase—16 percent (95% CI = 6, 28). There were 25 percent more passenger vehicle occupant fatalities on rural interstates in 1987 than the average of the previous five years, while on other rural roads the increase was only 8 percent. When all other roads were used as the comparison group, the odds for passenger vehicle occupant fatalities on rural interstates were 17 percent higher than expected.

A number of other factors that might affect the relation between speed limit changes and fatalities were also examined. These factors included different speed limits for cars and trucks, mandatory seat belt laws, daytime (6 am–9 pm) versus nighttime crashes, and the proportion of single or multiple vehicle fatal crashes. These analyses were done by subdividing the data for rural interstates and other rural roads for all fatalities by the appropriate factor and comparing the odds ratios. The estimated effect of 14 percent for the eight states with different speed limits for cars and trucks (California, Illinois, Michigan, Missouri, Ohio, Oregon, Texas, and Washington) was similar to the 15 percent effect for states with uniform limits. The effects were also similar in states with belt use laws (15 percent) and without belt laws (13 percent). For daytime and nighttime crashes, the effects were also similar (16 percent and 14 percent). The effect for fatalities involving single vehicles was 17 percent, and for fatalities involving multiple vehicles it was 10 percent. A test for homogeneity of the odds ratios was not significant implying no difference between the odds ratios.

The analysis for the eight states that retained 55 mph limits in 1987 indicated no effect during the comparison period. There was a 4 percent increase in all fatalities on rural interstates, but this was offset by an even larger increase on other rural roads (12 percent). The estimated odds ratio was 0.94 (95% CI = 0.77, 1.13).

Discussion

In the 38 states that set higher speed limits in 1987, fatalities on rural interstates were conservatively estimated to be 15 percent greater than they would have been if the states had retained the 55 mph limit on these roads. Increased fatalities were seen in 24 of the 38 states. In human terms, there were nearly 200 additional fatalities in 1987 during the months when the higher speed limits were in effect. This number will be larger for 1988 because two additional states have raised their speed limits, more miles of highways were eligible, and states will have had a whole year of experience.

In a 1988 interim report on the effect of the higher speed limit, NHTSA had expressed concern that the observed 1987 increase in fatalities began in the months prior to initiation of the higher limits.[8] To test this possibility, an alternative analysis examined fatalities for January through March of 1987 versus those for 1982–86. The odds ratio for fatalities on rural interstates versus other rural roads indicated that fatalities on rural interstates were about 6 percent higher than expected during the first quarter of 1987. Although the odds ratios in Table 2 could be adjusted for this first quarter increase, the odds ratio for the first quarter included 1.0 suggesting that the observed difference was due to random fluctuation. In addition, fatalities during winter months tend to fluctuate greatly from year to year because of varying weather patterns, and the current analysis could not control for these patterns. Moreover, there is the possibility that some drivers began increasing their travel speeds even before the speed limits were raised. Such anticipatory speed increases could have been triggered by news reports and publicity regarding possible federal action in early February and again in March.[9,10] For these reasons, it would have been inappropriate to adjust the estimated effects of the higher speed limits for the nonsignificant differences observed in January through March.

The estimated 15 percent increase in these data is somewhat smaller than the 20–25 percent increase predicted by the NRC partly because of the NRC assumption that all states would return to pre-1974 speed limits, often 70 and 75 mph. Because the energy associated with a crash increases exponentially with the speed of the crash, the likelihood of serious injury and fatality increases very rapidly as travel speeds increase in this range.[11] Therefore, even larger effects

TABLE 2—Number of Fatalities by Road Type and Time Period for All Fatalities and Passenger Vehicle Occupants

	Average 1987 Number	Average 1982–86 Number	Chi-Square Statistic	Odds Ratio	(95% Confidence Limit)
All Fatalities					
Rural Interstates	1,393.0	1,169.2			
vs Other Rural Roads	11,713.0	11,259.2	10.40	1.15	(1.06, 1.24)
vs All Other Roads	19,607.0	19,110.2	13.20	1.16	(1.07, 1.26)
Passenger Vehicle Occupants					
Rural Interstates	1,084.0	864.8			
vs Other Rural Roads	9,321.0	8,632.4	9.53	1.16	(1.06, 1.28)
vs All Other Roads	13,906.0	13,031.8	11.50	1.17	(1.07, 1.29)

than estimated here would have been expected if states had returned to these higher limits.

In addition, the 15 percent increase in fatalities found here probably underestimates the true effect of the 65 mph limits. The effect was measured by comparing the odds of a fatality on rural interstates to other roads. If higher travel speeds on rural interstates spill over onto some of these roads, as suggested by research on speed adaptation, then the number of fatalities would have increased on these other roads as well.[12–14] Such a spillover would reduce the estimated effect (but not the true effect) of the higher speed limits as measured in this analysis.

The NHTSA report highlighted two factors that argued against a large increase in fatalities attributable to the 65 mph speed limits: the states that did not increase their speed limits also had an increase in fatalities on rural interstates and six states accounted for 64 percent of the excess fatalities in 1987.[4] The current analysis examined the odds ratio for the unchanged states for 1982–86 versus 1987 and found rural interstate fatalities were marginally fewer than expected for May–December of 1987 in states that retained 55 mph speed limits. Thus, there is no evidence of a larger national trend toward more fatalities on rural interstates in 1987.

When odds ratios were computed for each state, they were greater than unity in 24 states. Therefore, despite the dominance in the absolute numbers by six states, the trend of higher crash fatalities on rural interstates than on other rural roads was observed in more than three of five states that increased their speed limits. The fact that rural interstate fatality counts were not higher than expected in a minority of states is consistent with the statistical uncertainty associated with analyses of data from individual states.

In summary, the results of this analysis show that the 65 mph limits increased rural interstate fatalities in 1987 by approximately 15 percent, there was no evidence of a similar trend in states that retained 55 mph limits, and the increase was not limited to only a few states.

ACKNOWLEDGMENTS

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ISHIB Calls for Abstracts on Hypertension in Blacks

The 5th International Interdisciplinary Conference on Hypertension in Blacks has issued a call for abstracts for its conference, to be held May 3–7, 1990 in Long Beach, California. The announced theme for the meeting is: "A Focus on Ethnicity: Negroids, Caucasoids, Mongoloids." The deadline for abstract submission is November 1, 1989.

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Homicide, Suicide, Motor Vehicle Crash, and Fall Mortality: United States' Experience in Comparative Perspective

IAN R.H. ROCKETT, PHD, MPH, AND GORDON S. SMITH, MB, CHB, MPH

Abstract: US mortality data on motor vehicle crashes, falls, suicide, and homicide for 1980 are compared with corresponding data for France, Japan, West Germany, and the United Kingdom. Unadjusted and age-specific death rates are presented, together with age-adjusted rates of years of life lost (YLL). A large male excess in rates is typical outside the fall category. Motor vehicle crashes are the predominant cause of YLL, and the United States manifests the highest YLL rates for each sex. US fall death rates at the older ages are exceeded by those of France and West Germany. The elderly generally manifest the greatest risk of suicide; American females exhibit a unique rate decline after ages 45–54 years, however. Beyond early adulthood, US suicide rates are lower than those of France, Japan, and West Germany. US homicide rates dwarf those of the comparison countries with 16- to 29-fold differentials separating prime-risk American males aged 25–34 years from their foreign counterparts. (*Am J Public Health* 1989; 79:1396–1400.)

Introduction

In a foreword to an influential report from the National Academy of Sciences, William Foege remarked on the persistence of injuries as a "major determinant of longevity" in the United States, and globally.[1] The 1980s have furnished much basic data on injury occurrence and witnessed renewed interest in their prevention.[2–7] Federal Government support has been strengthened by the formation of a Center for Environmental Health and Injury Control at the Centers for Disease Control, the establishment of five regional university-based injury prevention research centers, and growing sponsorship of pertinent research projects and prevention programs by the National Institutes of Health.[8,9]

Nationally, injuries rank fourth as a cause of death behind heart disease, cancer, and cerebrovascular disease,[10] but they are the premier killer of Americans between ages one and 45 years,[10] and are the leading cause of years of life lost (YLL) before age 65.[11] An international study revealed marked differences in all-cause injury death and age-adjusted YLL rates for the United States, France, Japan, the United Kingdom, and West Germany.[12] The aim of the present paper is to shed light on these findings through an examination of data on unintentional motor vehicular and fall mortality, suicide and homicide—the leading causes of injury mortality in the United States.[4] The four comparison countries are of particular interest due to similarities they share with the United States in terms of development and mode of government, and because of their close political and economic links to this country. All five countries have populations exceeding 55 million, and thus may be considered demographically large.

Methods

Data are adapted from 1980 age-, sex-, and cause-specific mortality tabulations published by the World Health Organization (WHO).[13] Total injury deaths (E800–E999) and deaths from motor vehicle traffic crashes (E810–E819), falls (E880–E888), suicide (E950–E959) and homicide (E960–E969) are classified under an abbreviated three-digit external cause code according to the basic tabulations list of the ninth revision of the *International Classification of Diseases* (ICD-9).[14]

YLL rates are adjusted for age by means of a direct standardization procedure,[15] using the 1980 US population as the reference. YLL represent the summation of the products of the number of deaths at each age, here starting at age one, and the years of life remaining up to the age ceiling, here age 65. Since the WHO age data are abridged, age at death for decedents is estimated as the mid-point of the respective age groups.

Results

Crude Rates and Causal Components

France manifests the highest crude injury death rates for either sex (116 per 100,000 males and 73 per 100,000 females) (Table 1). The United States has the second highest rate for males (105 per 100,000), West Germany the second highest rate for females. Male rates surpass female rates in all five countries, and the magnitude of the male excess ranges from 50 percent in the United Kingdom to 180 percent in the United States.

Motor vehicle crashes, falls, suicide, and homicide collectively account for between two-thirds and four-fifths of total injury deaths in each country. However, homicide is a major cause of death only in the United States. Homicide rates in the remaining four countries approach individual mortality rates for fire, poisoning, drowning, and other transportation (data not shown).

Except in Japan, motor vehicle crashes are the leading cause of injury mortality among males, with the United States having the highest sex-specific rates. Motor vehicle crashes are also the leading cause of injury mortality among females in the United States, but in the remaining countries they are surpassed by suicide, falls, or both. Male predominance universally characterizes motor vehicular mortality and suicide, but the sex differential is sharply reversed with respect to fall mortality in France, West Germany, and the United Kingdom.

Age-Specific Rates

Both sexes exhibit a bimodal age-specific pattern of motor vehicular mortality, irrespective of country (Figure 1). Peaks occur at ages 15–24 years and over 74, with male rates greatly exceeding female rates. Japan and the United King-

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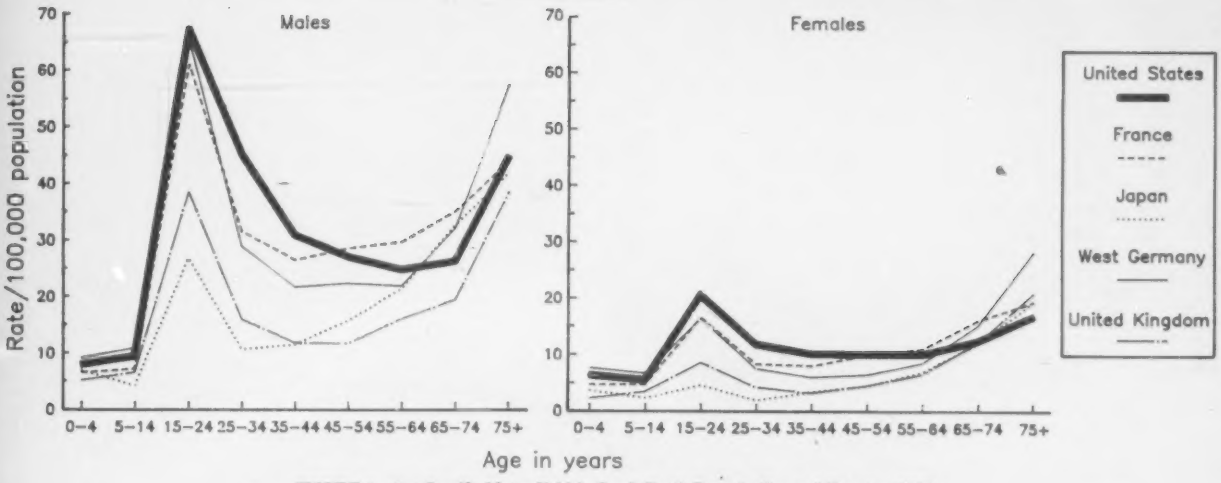

FIGURE 1—Age-Specific Motor Vehicle Crash Death Rates by Sex and Country, 1980

dom manifest the lowest motor vehicular death rates for both sexes, although there is a trend toward convergence at the upper end of the age spectrum. The United States has the highest motor vehicular death rate for females from ages 15 through 44 years, and the lowest rate in the over 74 population.

Fall mortality is profoundly selective of the elderly (Figure 2). In the over 74 age group, which reflects the greatest risk, there is considerable cross-national variation by sex, with rates peaking among French and West Germans. In France, West Germany, and the United Kingdom, female rates are approximately 50 percent higher than corresponding male rates. Japan has by far the lowest national rates, and US rates also are relatively low.

Generally, age-specific suicide rates are highest in France, Japan, and West Germany, with the elderly most at risk in all countries (Figure 3). Male rates universally exceed female ones. American females manifest a unique decline in their suicide rate after ages 45–54.

Homicide rates are usually lower than suicide rates, except in the two youngest age groups, where there is a major departure from the norm by the United States because of extremely high homicide rates among adolescents and younger adults of both sexes (Figure 4). In the prime-risk 25–34 age group, the US rate for males ranges from 16 to 29 times other national rates. The US rate for females in that age group, while less than one-fourth that for US males, is still more than triple the second highest national male homicide rate, that of West Germany.

Years of Life Lost

Motor vehicular, suicide, and fall YLL rates are invariably much greater for males than females (Table 2). This finding is repeated with US homicide YLL rates, which for each sex marginally surpass corresponding suicide rates, and dwarf other national homicide YLL rates. Suicide produces the highest sex- and cause-specific YLL rates in Japan; elsewhere it is motor vehicle crashes. Male suicide YLL rates are similar in the United States, France, Japan, and West Germany, and are at least double the British rate. Female suicide YLL rates are highest in France, Japan and West

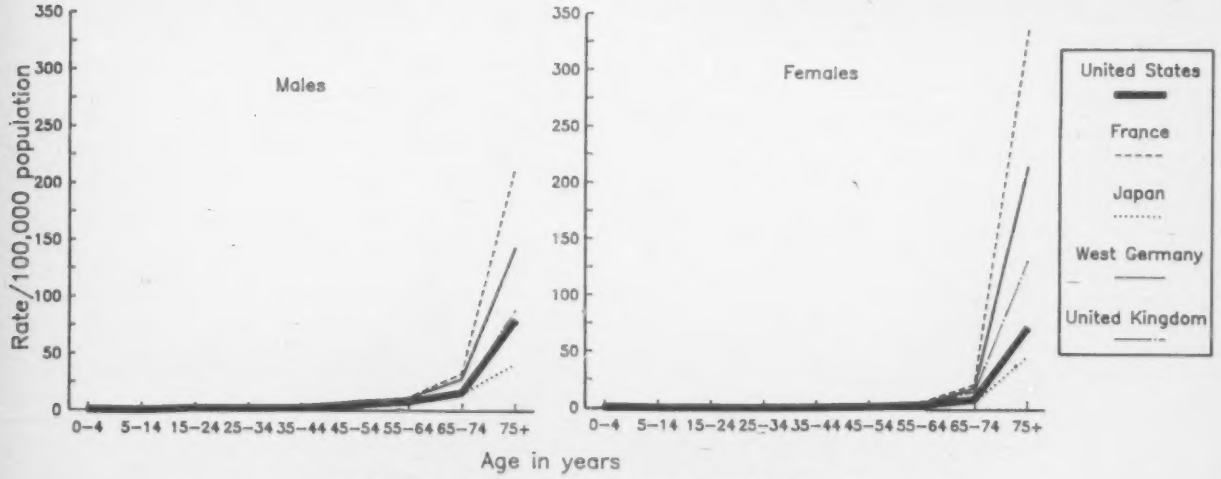

FIGURE 2—Age-Specific Fall Death Rates by Sex and Country, 1980

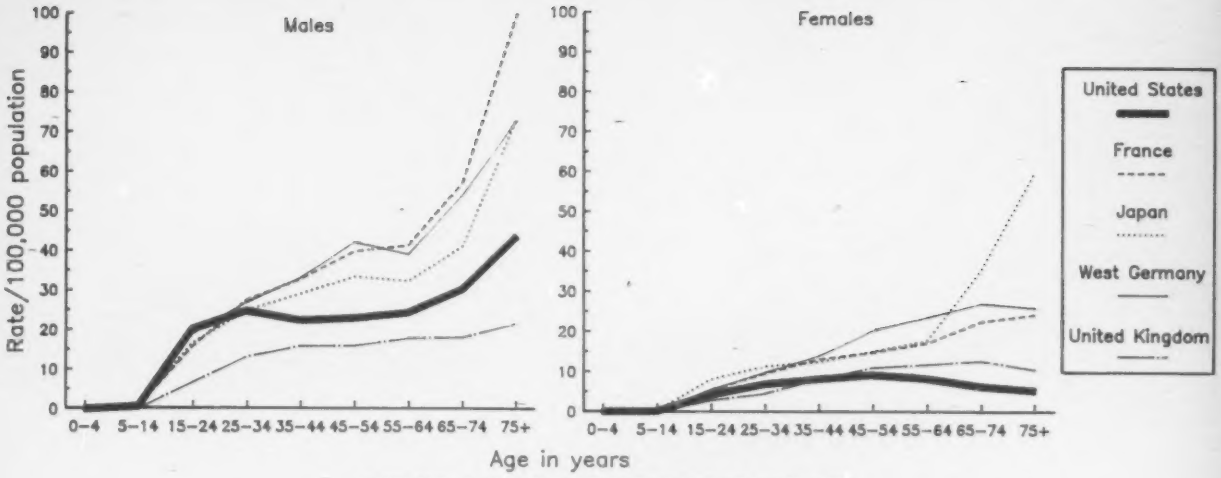

FIGURE 3—Age-Specific Suicide Rates by Sex and Country, 1980

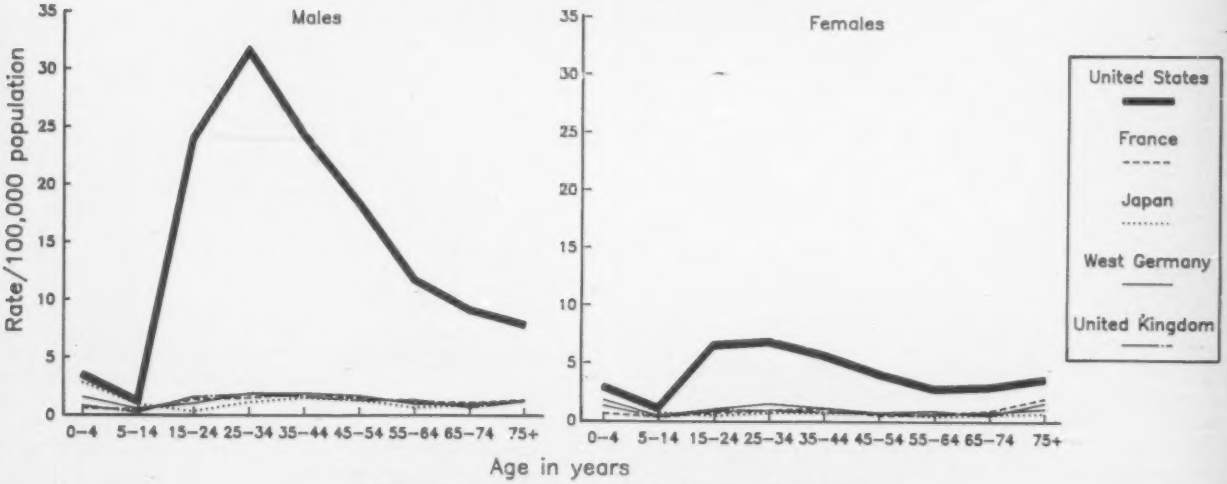

FIGURE 4—Age-Specific Homicide Rates by Sex and Country, 1980

Germany, and approach the British rate for males. Fall YLL rates are consistently small by sex and across countries.

Discussion

Focusing on 1980 motor vehicle crash, fall, suicide, and homicide data, this research has pointed to substantial differences in the injury mortality profiles of the United States and four other major Western bloc countries: France, Japan, the United Kingdom, and West Germany. Motor vehicle crashes are the predominant cause of injury mortality in the United States, and are also the predominant cause of YLL between ages one and 65 years in all countries under review, except Japan. The sharp peaks in motor vehicular death rates, which are exhibited at ages 15–24 years, have also been observed in many other countries.[16]

A better single indicator of road death risk than an overall population-based mortality rate is number of deaths per motor vehicle distance traveled. In 1980, the United States and the United Kingdom both registered 2.1 road deaths per 100 million vehicle kilometers traveled.[17] The Japanese figure was marginally higher (2.25). In contrast, the French and West German figures were 4.6 and 3.8, respectively. All countries in the study manifested appreciable declines on the exposure measure between 1970 and 1980; from 68 percent for Japan to 30 percent for the United States.[17,18] Similar declines occurred in the overall motor vehicular mortality rates.[13,19] A more protracted downward trend has also been noted for select populations.[20]

A recent US study revealed large inter-county differences in motor vehicle crash mortality rates, which varied inversely with population density and income.[21] The authors speculated that this variation reflected differences in a host of underlying variables, including road quality and design, speed, vehicle type and condition, seat belt use, and access to emergency care. These and other variables, such as alcohol use,[5] vehicle safety design features,[3] access to safer modes of transport, such as trains,[5] and seat belt legislation and related timing, compliance, and enforcement[22] should be integral to analytic epidemiologic research aimed at addressing spatial and temporal variations in the road toll.

TABLE 1—Injury Mortality by Sex, Cause and Country: Rates and Percentage Distribution, 1980*

Sex/Cause	United States		France		Japan		West Germany		United Kingdom	
Male	Rate	%	Rate	%	Rate	%	Rate	%	Rate	%
Motor Vehicle	34.4	32.6	30.5	26.3	15.1	24.2	30.4	34.7	17.6	33.7
Fall	6.4	6.1	15.0	12.9	5.1	8.2	11.6	13.2	6.9	13.2
Suicide	18.6	17.6	28.0	24.2	22.2	35.5	28.3	32.3	11.0	21.1
Homicide	17.0	16.1	1.2	1.0	1.1	1.8	1.3	1.5	1.2	2.3
Other Injury	29.1	27.6	41.2	35.6	19.0	30.3	16.0	18.3	15.5	29.7
All Injury	105.5	100.0	115.9	100.0	62.5	100.0	87.6	100.0	52.2	100.0
Female										
Motor Vehicle	12.0	31.7	10.8	14.8	4.8	16.9	11.0	21.1	6.9	19.4
Fall	5.4	14.3	30.0	41.1	2.5	8.8	19.3	37.1	12.1	34.0
Suicide	5.4	14.3	11.1	15.2	13.1	46.1	14.1	27.1	6.7	18.8
Homicide	4.4	11.6	0.8	1.1	0.8	2.8	1.0	1.9	0.8	2.2
Other Injury	10.6	28.1	20.3	27.8	7.2	25.4	6.6	12.8	9.1	25.6
All Injury	37.8	100.0	73.0	100.0	28.4	100.0	52.0	100.0	35.6	100.0

*Rates per 100,000 population.

Fall mortality primarily strikes the elderly. Yet geriatric fall deaths are probably markedly undercounted because many of the victims die of complications stemming from the initial injury, and may have a consequential condition coded as their underlying cause of death.[23–25] It is unknown how much of this contributes to the differences observed in this study. There has been a secular decline in the US fall death rate, with a reduction of 35 percent occurring between 1970 and 1980 alone.[5] This trend may be a product of improved medical care, such as hip surgery and earlier mobilization after hip fracture.[4,5] But also unknown is how much of the observed cross-national fall mortality differentials can be explained by quality of care considerations.

Both validation of the WHO cause-of-death data and estimation of the amount of variation in rates attributable to different national coding practices lie outside the scope of this study. However, an explanation was sought for the relatively high percentage of unintentional injury deaths among French males in 1980 that were classified by WHO under the rubric of "all other accidents," and were reflected in the residual injury mortality categories in Tables 1 and 2. The French age mortality distribution for "other" unintentional injuries corresponds to the distribution for motor vehicular injuries, probably due to a frequent tendency by French physicians to write "accident" (coded as unspecified unintentional injury) on the death certificate as the sole external cause descriptor in a fatal motor vehicular trauma case.*

Like motor vehicular death rates, suicide rates vary dramatically by country, age, and sex. The very high rate observed among elderly Japanese females may be a legacy of the unusual degree of sociocultural acceptance that was traditionally accorded suicide in Japan.[26,27] Only the United Kingdom registers lower rates than the United States over most of the age spectrum. American females manifest the lowest national rates among the middle aged and elderly, and the decline in their rates after ages 45–54 is unique. Since American males do not repeat the trend, the finding probably cannot be explained by differential misclassification. However, the stigmatized nature of suicide, coupled with prohibitive clauses in life insurance policies, inevitably raises questions concerning underreporting.[6] Case ascertainment is also affected by definition. While not conclusive, a review of studies on the validity and reliability of official suicide statistics underlines a claim that these data are of sufficiently high quality to justify their use in international epidemiologic comparisons.[28] Cross-national analytic studies, which incor-

*Personal communication, Rachid Salmi, MD, France, February 3, 1989.

TABLE 2—Years of Life Lost (YLL) Due to Injury by Sex, Cause and Country: Age-Adjusted Rates and Percentage Distribution, 1980*

Sex/Cause	United States		France		Japan		West Germany		United Kingdom	
Male	Rate	%	Rate	%	Rate	%	Rate	%	Rate	%
Motor Vehicle	12.5	38.4	10.6	26.3	4.8	31.9	11.0	48.9	6.4	46.0
Fall	.7	2.2	.8	3.2	.7	4.8	.8	3.7	.6	4.5
Suicide	4.9	15.1	5.4	20.3	5.0	32.9	5.8	25.6	2.4	17.3
Homicide	5.8	17.9	.4	1.5	.3	2.3	.4	1.8	.4	2.9
Other Injury	8.6	26.4	9.1	48.7	4.3	28.1	4.5	20.0	4.1	29.3
All Injury	32.5	100.0	26.3	100.0	15.1	100.0	22.5	100.0	13.9	100.0
Female										
Motor Vehicle	4.1	42.8	3.2	38.4	1.2	22.9	3.4	45.6	1.7	36.6
Fall	.2	2.1	.2	2.7	.1	1.9	.2	3.2	.1	3.1
Suicide	1.4	14.4	2.0	23.5	2.3	45.0	2.2	29.4	1.1	23.5
Homicide	1.6	16.4	.2	2.6	.3	5.0	.3	4.3	.2	4.8
Other Injury	2.3	24.3	2.8	32.8	1.3	25.2	1.3	17.5	1.7	32.0
All Injury	9.6	100.0	8.4	100.0	5.2	100.0	7.4	100.0	4.8	100.0

*YLL rates per 1,000 population.

porate determinants and correlates that have been identified in recent literature reviews,[29–31] may yield new insights into suicide etiology.

Although homicide accounts for fewer deaths in the United States than either motor vehicle crashes or suicide, US rates are grossly disparate from those in the comparison countries on all three measures utilized in this study. In 1980, handguns featured in an estimated 11,522 US homicides[32]; about half of the total. Handguns were also implicated in fourth-fifths of cases of firearm-related murder and nonnegligent manslaughter,[33] yet constituted only 30 percent of the national civilian firearm stock.[32] The foregoing statistics seem all the more alarming when one considers that in 1980 there were only eight handgun-related homicides in the United Kingdom,[32] which strictly regulates the sale and distribution of firearms, as do France, Japan, and West Germany. The US handgun-related homicide rate was 500 times the British rate, and the overall homicide rate was two-and-a-half times the rate of Northern Ireland,[13] a country in a state of civil war. Moreover, the homicide rate for both sexes rose by 30 percent between 1970 and 1980.[13,19] Results of a recent study suggest that restricting handgun access would be the most effective way to reduce the high US homicide rate.[34] The epidemic of firearm-related homicide (and other violence) warrants unprecedented ameliorative efforts by government agencies at all levels.

Detection of large international differences in disease rates has spawned hypotheses which, following testing, have advanced both understanding of disease etiology and prevention.[35] For example, the determination of such differences in cancer incidence rates[36,37] is credited with stimulating an array of analytic studies indicating that dietary factors have both causative and inhibitory roles in human carcinogenesis.[38] Similar progress could ensue from detection and analysis of international differentials in injury mortality and morbidity.

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AT

Determinants of Polychlorinated Biphenyls (PCBs), Polybrominated Biphenyls (PBBs), and Dichlorodiphenyl Trichloroethane (DDT) Levels in the Sera of Young Children

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Abstract: Serum samples from 285 4-year-old Michigan children were evaluated for levels of 11 environmental contaminants. Polychlorinated biphenyls (PCBs) were found in half the samples tested; polybrominated biphenyls (PBBs) in 13–21 percent; dicholorodiphenyl trichloroethane (DDT), in more than 70 percent. Nursing (Mothers' milk) was the principal source of these exposures. Congener-specific analysis documented the presence of at least one highly toxic PCB congener, 2,3',4,4',5-pentachlorobiphenyl. The data demonstrate the multigenerational impact of female exposure to persistent organic environmental contaminants. (*Am J Public Health* 1989; 79:1401–1404.)

Introduction

Halogenated polycyclic hydrocarbons are among the most ubiquitous and persistent contemporary environmental contaminants. Polychlorinated biphenyls (PCBs), once used in a broad range of industrial products, are found at particularly high concentrations in the fatty tissue of fish feeding in polluted waters, such as Lake Michigan, and in animal products from farms with contaminated silos.[1,2] Polybrominated biphenyls (PBBs), formerly used primarily as flame retardants, were accidentally mixed with cattle feed in Michigan in 1973–76, leading to widespread dairy and meat contamination throughout the state.[3] Dichlorodiphenyl trichloroethane (DDT), developed to control malaria-carrying mosquitoes, became one of the world's most extensively used pesticides in the 1950s and 1960s.[4] Dichlorodiphenyl dichloroethene (DDE) is its most stable and persistent metabolite.

PCBs, PBBs, and DDE are not readily excreted, except by lactation.[5] Placental passage has been documented for PCBs and PBBs,[6–8] although much larger quantities are transferred to the infant postnatally in breast milk due to its high lipid content.[9] A positive relation between duration of nursing and serum PCB level in young children has been reported in three studies, one in Germany[10] and two in Japan.[5,11]

Methods

The study included two cohorts of children evaluated at age 4 years. The "fish exposure cohort" consisted of 236 children recruited in 1980–81 when 8,482 women delivering infants in four western Michigan hospitals were surveyed regarding their consumption of Lake Michigan sport fish.[12] "Contaminated fish consumption" was defined as the sum of annual Lake Michigan fish consumption, weighted by the PCB content of the species consumed, in the present or past, whichever was greater.[13] All women consuming at least 11.8 kg over a six-year period (4.0 percent of those interviewed) were invited to participate in the study. A small proportion (4.6 percent) of those who did not consume these fish were also recruited and constituted 22.7 percent of the final sample. Of those assessed as infants, 75.4 percent were seen again in the present study. The children evaluated at age 4 did not differ from those not retained in the sample in terms of cord and maternal serum PCB levels but were somewhat higher in socioeconomic status (SES) and maternal fish consumption and milk PCB levels.[14]

The "farm exposure" cohort consisted of 87 children who had not previously been assessed as infants. Their families were enrolled in long-term studies of exposure to PBB or PCB contaminated farm products conducted by the Michigan Department of Public Health. The PBB families had lived on or received food from quarantined farms;[15] the PCB families had eaten milk and meat from farms whose animals consumed silage contaminated with PCBs.[16]

Information regarding family SES and residence, maternal age, weeks of breast feeding, maternal contaminated fish consumption during and before pregnancy, and child's contaminated fish consumption during the previous year was obtained by maternal interview at the four-year assessment. When compared with similar information obtained from the fish exposure cohort during infancy, test-retest reliabilities (Pearson r) ranged from 0.68 for maternal fish consumption to 0.99 for maternal age (median = 0.81).[14] The four-year maternal fish consumption report was also validated in relation to maternal serum PCB level obtained at delivery ($r = 0.37$, $P = 0.000$). Venous blood was obtained from 285 of the 323 children. Specimens were obtained from 250 children at age 4, 35 children at age 5. Levels of PCBs, PBBs, DDT, and lead did not differ significantly at the two ages.

Total serum PCB level was evaluated by the Michigan Department of Public Health by packed column electron capture gas chromatography, adapting the Webb-McCall method[17] to a computer data system, in which Aroclors 1016 and 1260 were used as reference standards.[18] Quantitation was also provided for PBBs, Total DDT (p,p'-DDT + p,p'-DDE), hexachlorobenzene, beta HCB, oxychlordane, heptachlor epoxide, trans-nonachlor, and mirex. The same laboratory had previously provided data on PCB and PBB levels in umbilical cord serum, maternal serum collected two to three days after delivery, and maternal milk for the fish

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TABLE 1—Environmental Contaminants Found in the Sera of Young Children

	Total N	Children with Trace Levels (1 ng/mL) or Greater			
		%	Mean	s.d.	Range
PCBs					
Fish exposure	205	52.2	4.18	3.29	1.00–19.40
Farm exposure	80	48.8	4.82	4.81	1.00–23.30
PBBs					
Fish exposure	205	12.7	2.44	1.50	1.00–6.40
Farm exposure	80	21.3	2.95	2.66	1.00–9.50
Total DDT					
Fish exposure	202	69.8	4.36	3.87	1.00–21.40
Farm exposure	79	77.2	4.24	4.47	1.00–143.20*
Lead†					
Fish exposure	194	—	0.30	0.16	0–1.01
Farm exposure	77	—	0.32	0.17	0.10–1.21

Means are ng/mL, except for lead μmol/L. The laboratory detection limit for PBBs and DDT is 1 ng/mL; for PCBs, 3 ng/mL. 26.3% of the fish exposure children and 23.8% of the farm exposure children had PCB levels of 3 ng/mL or greater.

*Two outliers (143.2 and 73.4) are recoded to 15 for mean and s.d. calculation, as recommended by Winer.

†Mean, s.d., and range are based on all cases tested.

exposure cohort using the same analytic method.[13] Quality control has been maintained consistently since 1979 in interlaboratory comparisons following protocols established by the Centers for Disease Control and the Environmental Protection Agency. The four-year blood samples were also analyzed for lead by atomic absorption spectrophotometry using the Delves cup method.

With the exception of the breast milk PCB and four-year blood lead levels, all of the exposure measures were positively skewed (skew > 2.5) and normalized by means of log X + 1 transformation. Means and standard deviations are reported in terms of the original distributions; the log transforms were used in the correlation and regression analyses. Child's contaminated fish consumption was measured in terms of current annual kg consumed (log X + 1) with species weighted to reflect degree of PCB contamination.[13] Weeks of nursing was transformed by recoding seven values greater than 3 standard deviations above the mean to 1 point greater than the highest observed value, as recommended by Winer.[19]

Results

PCBs were detected in about half of the 4-year-olds tested; PBBs, in relatively few samples; and DDT, though banned for many years, in a large majority of the children (Table 1). Although the mothers in the fish exposure cohort had eaten more Lake Michigan fish before and during pregnancy than their farm exposure counterparts, 7.6 kg/yr versus 1.2 kg/yr, PCB and DDT levels were similar in the two groups of children. PBBs, which had been a specific contamination problem on many of the farms, were somewhat more prevalent in the farm exposure cohort.

DDE averaged 89.0 percent of the total DDT in the children's sera, which is similar to the 86.7 percent reported for a more highly exposed, predominantly Black, rural sample in the early 1980s.[20] Except for hexachlorobenzene, which was present at low levels (< 2.0 ng/mL) in three cases, none of the other organochlorine pesticides tested were detected in the four-year samples. Lead levels were generally low, although 11 children had levels between 0.72 and 1.21 μmol/L.

The relation of several hypothesized determinants to four-year body burden is shown in Table 2. All predictors with zero-order correlations significant at $P < 0.10$ were also included in multiple regression analyses, which adjusted statistically for confounding among the predictors. While cord serum level was unrelated to PCB body burden at age 4 ($P = 0.615$), maternal milk PCB level and weeks of breastfeeding jointly explained 59.7 percent of the variance in four-year PCB level ($P = 0.000$). In the absence of nursing, the children's body burdens diminished with time as growth reduced the proportion of the original *in utero* dose (Table 3). Prolonged breastfeeding, by contrast, was associated with substantial increases in PCB burden. Where maternal milk PCB level exceeded 1000 ng/mL, each additional week of nursing was associated with a 0.19 ± 0.03* ng/mL increase in the child's four-year serum PCB level. Where maternal milk level was less than 600 ng/mL, the increase associated with each week of nursing was 0.09 ± 0.02 ng/mL. Socioeconomic status (SES) and maternal age were not significant predictors of four-year PCB serum level after adjustment for nursing exposure in the regression analysis. The SES effect was attributable to the fact that higher SES mothers nursed for longer periods; the maternal age correlation, to higher PCB levels in the milk of older mothers.

Maternal milk PBB level and weeks of nursing jointly

*Unstandardized regression coefficient ± s.e. from a univariate regression of four-year serum PCB level (prior to log transformation) on weeks of nursing

TABLE 2—Effects of Pre- and Postnatal Predictors on Four-year Serum Contaminant Levels

	N	PCBs		PBBs		DDT		Lead
		Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted
Cord serum level	126	0.05	—	0.29	0.34	—	—	—
Maternal milk level	107	0.29	0.33	0.39	0.31	—	—	—
Weeks of breastfeeding	285	0.66	0.70	0.42	0.42	0.61	0.56	−0.11
Socioeconomic status	285	0.26	0.07	0.23	0.09	0.29	0.08	−0.21
Maternal age	285	0.15	0.01	0.09	—	0.26	0.11	−0.15
Rural residence*	285	0.01	—	0.02	—	0.12	0.13	0.03
Child's fish consumption	285	0.08	—	0.02	—	0.03	—	0.01
Multiple R		—	0.78	—	0.65	—	0.64	—

Unadjusted values are zero-order Pearson correlation coefficients. Adjusted values are standardized regression coefficients from analyses in which each contaminant was examined in relation to the set of predictors for which values are shown. All PCB, PBB, and DDT values below the laboratory's detection limits were recoded to 0 before being subjected to correlational and regression analysis.

*Coded 0 for urban or suburban, 1 for rural.

TABLE 3—Comparisons of Cord and Four-year Serum PCB Levels in Relation to Duration of Breastfeeding

Months of Breastfeeding	N	Cord Serum PCB level	Four-year Serum PCB level
0.0–2.9	66	2.8	0.6
3.0–5.9	17	2.2	2.3
6.0–11.9	30	2.6	4.4
12.0+	13	2.1	5.5

Means are ng/mL.

explained 31.5 percent of the variance in four-year serum PBB level (P = 0.000). Although four-year PBB body burden was correlated with cord serum level as well, inspection of a scatterplot relating the breast milk and four-year PBB levels suggested that the relation between these variables was curvilinear. When a quadratic term for milk level was added to the regression, the multiple R increased significantly to 0.69, and the standardized regression coefficient for cord serum PBB level was no longer significant, $\beta = 0.10$, P = 0.384. Moreover, a case by case examination of the data on which the PBB regression was based indicated that all the children with detectable four-year serum PBB levels had been nursed for at least four months.

Although no data were available on breast milk DDT levels, duration of nursing explained 36.8 percent of the variance in four-year serum DDT level (P = 0.000). DDT levels were also higher in children in rural areas and in the children of older mothers, even after adjustment for weeks of nursing (Ps = 0.007 and 0.032, respectively). Because the total DDT in these children consisted primarily of DDE, the correlation and regression coefficients for these predictors in relation to DDE were virtually identical to those shown for total DDT. No regression was computed for lead because the magnitude of its relations with these predictors was small.

The Webb-McCall isomer identification method records chromatographic elution peaks, each of which is comprised of one or more specific PCB congeners (Figure 1). Peaks 203, 232/244, 332, and 372 were statistically more prevalent in the farm exposure cohort, suggesting that the farm children may

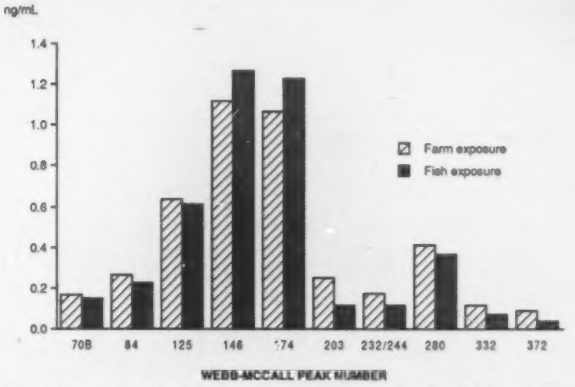

FIGURE 1—Webb-McCall chromatographic elution peak values by exposure cohort.

have been exposed to sources of PCBs to which the fish cohort children were not exposed. Despite these differences, the overall pattern of elution peaks was highly similar in the two cohorts and closely matched the pattern found by Humphrey[21] in adults and regular fish eaters. Although many different PCB mixtures have been deposited in the environment,[22] these data confirm that certain congeners are preferentially accumulated in both adults and children.

Serum extracts from 20 children with PCB serum levels in excess of 3.0 ng/mL were pooled into two composite samples—one for the boys (N = 9); the other for the girls (N = 11)—and analyzed at the US Environmental Protection Agency Large Lakes Research Station at Grosse Ile, Michigan, by means of capillary column gas chromatography.[23] The type and concentration of PCB congeners found in the two pooled samples were highly consistent (Table 4), making it unlikely that the pattern observed was determined by one or two aberrant children. The most prevalent congeners, Nos. 153, 138, 180, and 118, were also among the most prevalent in human milk and serum samples evaluated in prior studies.[25–29] No. 118, a constituent of Webb-McCall Peak 125 (Figure 1), is a mono-*ortho* analog of the highly toxic

TABLE 4—Most Prevalent Congeners in Pooled Serum Samples at Age 4 Years

		Sample 1 (11 females)		Sample 2 (9 males)	
Congener No.[24]	Structure	Mean	%	Mean	%
153	2,2′,4,4′,5,5′	1.50	19.6	1.30	15.8
138	2,2′,3,4,4′,5′ *	1.30	17.0	1.15	14.0
163	2,3,3′,4′,5,6 *				
180	2,2′,3,4,4′,5,5′	0.62	8.1	0.56	6.8
118	2,3′,4,4′,5	0.35	4.6	0.53	6.5
170	2,2′,3,3′,4,4′,5	0.35	4.6	0.32	3.9
005	2,3 *	0.27	3.5	0.04	0.5
008	2,4′ *				
099	2,2′,4,4′,5	0.22	2.9	0.22	2.7
187	2,2′,3,4′,5,5′,6 †	0.21	2.7	0.16	2.0
182	2,2′,3,4,4′,5,6′ †				
031	2,4′,5 *	0.21	2.7	0.22	2.7
028	2,4,4′ *				
018	2,2′,5	0.14	1.8	0.15	1.8
Total		7.65‡	67.5§	8.20‡	56.7§

From capillary column gas chromatographic analyses. Means are ng/mL.
*Sum.
†Average.
‡Total PCBs from the pooled sample.
§Percentage of the sample comprised of the congeners listed on this table.

coplanar PCB congeners, which elicit effects resembling those caused by 2,3,7,8-TCDD. This congener, which has also been detected at relatively high concentrations in human milk in the US,[25,26] Germany,[27] and Japan,[28] is likely to provide a major contribution to the overall toxicity of these children's PCB exposure.[26,30]

Discussion

The present data implicate nursing as the principal source of PCB and PBB exposure in these children and a significant source of DDT exposure as well. Although prenatal PCB exposure has been shown to affect physical and behavioral development,[12,31,32] the quantity transferred *in utero* is apparently too small to detect in serum by age 4 years. In contrast to transplacental exposure, developmental deficits have not been linked to postnatal nursing exposure in North American children,[33,34] although dermatological effects from nursing have been documented in the infants of occupationally exposed women in Japan.[35]

Postnatal exposure to the halogenated hydrocarbon contaminants was greater for higher SES children, because their mothers tended to breastfeed for longer periods. Postnatal fish consumption by the child had little effect on PCB body burden, because most children ate less than one fish meal per year. While virtually all of the children with PCB levels of 3.0 ng/mL or greater had breastfed for at least six weeks, one child who had nursed for only two weeks but had eaten 84 contaminated fish meals in the previous year also had an elevated PCB body burden at age 4.

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Commentary

Fluoridation Referendum in La Crosse, Wisconsin: Contributing Factors to Success

RHYS B. JONES, DDS, MS, DOUGLAS N. MORMANN, MS, AND TIMOTHY B. DURTSCHE, DDS

Abstract: Residents of La Crosse, Wisconsin approved a public referendum in favor of water fluoridation on April 5, 1988. The vote, 57 percent supportive, culminated a two-year community effort. Three public referenda had been defeated in the past. Contributing to the success of this recent campaign were: broad-based community support led by a 34-member Citizens for Better Dental Health in La Crosse Committee; American Dental Association/Wisconsin Division of Health/US Public Health Service consultation and support; knowledgeable and supportive press coverage; the timing of the ballot to coincide with the Wisconsin Presidential Primary; and local chiropractic support to offset chiropractic anti-fluoridation leadership. La Crosse, population 50,000, was the largest fluoride-deficient community in a nine-state upper Midwest area. (*Am J Public Health* 1989; 79:1405–1408.)

Introduction

Water fluoridation, the purposeful supplementation of naturally occurring fluoride to optimal levels for the prevention of dental caries, is one of the most successful public health measures in history.[1–3] Nevertheless, despite over 40 years documentation of safety, efficacy, cost-effectiveness, and practicality, and the unqualified endorsements of every major health organization, water fluoridation is far from universal in the United States. At the present time, approximately 63 percent of the US citizens have access to community drinking water containing dentally significant levels of fluoride (0.7 ppm).*

From a public health perspective, centralized decision-making by local health administrations affords the best opportunity for fluoridation implementation in the 42 states which have no legislatively mandated fluoridation.[4,5] Unfortunately, decisions on fluoridation in many communities are made in a highly charged political arena.[6,7] A number of fluoride-deficient communities in the US have failed to fluoridate because of initiative defeat in public referenda.[8,9] Fluoridation remains one of the few, if not the only, public health measures to be put to the vote of the public. Of the 150 referenda recorded by the Dental Disease Prevention Activity, Centers for Disease Control from 1980 through 1988, 64 percent (96) ended in failure for fluoridation proponents. Oftentimes proponents are ill-prepared for the political nature of a fluoridation campaign. By contrast, over the same time period, in 258 communities where the decision was left to administration (city councils or health boards), 77 percent (199) implemented fluoridation* (Table 1).

The purpose of this discussion is to outline key contributing factors in a recently successful fluoridation initiative in La Crosse, Wisconsin. A 20-month community effort resulted in a 57 percent positive vote for fluoridation. These contributing factors to success may be applicable to fluoridation strategies in other fluoride-deficient communities where the issue is decided by public referendum.

La Crosse Historical Perspective

La Crosse, 50,000 population, was the largest fluoride-deficient community (0.1 ppm) in a nine-state upper Midwest region. Although 93 percent of Wisconsin residents on public water supplies have optimally fluoridated water, La Crosse has a long history of antifluoridation obstructionism. On three occasions, voters rejected fluoridation in public referenda despite widespread support from health professional organizations. In 1953, by council action, the city fluoridated for three months until antifluoridation forces led by area chiropractors pressured officials to reconsider. Soon after, a first referendum failed with only 18 percent positive vote. The fluoridation equipment was disconnected and sold.

A second referendum failed in 1967 with 43 percent support; a third in 1969 gained only 29 percent support. Antifluoridation influence continued in the 1970s and 1980s driven by a prominent local chiropractor whose family had led the fight against fluoridation for over 30 years. Tactics used by La Crosse anti-fluoridationists through the years copied national anti-strategies capitalizing on the concerns of

TABLE 1—Fluoridation Implementation in US Communities, by Decision-making Process, 1980–88

Year	By Referendum		By Governing Body	
	Yes	No	Yes	No
Year				
1980	7	33	19	14
1981	4	10	26	8
1982	6	13	28	6
1983	6	13	20	14
1984	6	5	37	4
1985	5	4	15	4
1986	6	9	21	4
1987	4	3	18	5
1988	10	6	15	0
Total	54 (36%)	96 (64%)	199 (77%)	59 (23%)

SOURCE: Dental Disease Prevention Activity, Centers for Disease Control.

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*Dental Disease Prevention Activity, Centers for Disease Control, Atlanta, GA.

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society at the time. Fluoridation was alleged to be a Communist plot in the 1950s, a cancer causing "toxic waste" and "poison" in the 1960s and 1970s, and by the 1980s linked to aging, Alzheimer's disease, AIDS and "freedom of choice."

Renewed Fluoridation Initiative

La Crosse community leaders renewed fluoridation efforts in 1986 in response to a perception that a majority of a new city council would be supportive. A local committee was formed led by a representative of the La Crosse District Dental Society and the county public health department director. Efforts were directed toward convincing an 18-member city council to implement fluoridation without public referendum by convincing councillors of the health benefits of fluoridation as well as demonstrating public support for the initiative. Community support was strong through four public hearings and led to a June 1987 city council 10 to 8 vote for fluoridation, the first positive vote in 34 years. Despite the positive vote, the council delayed funding for 60 days thus allowing opponents the opportunity to petition for public referendum. This allowed fluoridation opponents to obtain the 3,000 signatures necessary to force a referendum. The pro-fluoridation majority of the council set the date for the referendum at the next major election, April 5, 1988, the Wisconsin Presidential Primary, rather than at an earlier special election date for a school bond. The wording of the ballot, an important component of successful fluoridation referenda, read in a straightforward and simple manner as follows: "Should the City of La Crosse fluoridate its water supply?"

Factors Contributing to Success

When compared to the previous defeats, the following five factors could be identified:

Broad-Based Community Support

A broad support base is necessary to assure success once a fluoridation issue is politicized.[4,8] Soon after the establishment of the referendum date, the original pro-fluoridation committee formed a campaign organization, "Citizens for Better Dental Health in La Crosse." The organization included representatives from health professional associations, the public health sector, hospitals, business and industry, labor, university, and civic and community organizations. The Citizens for Better Dental Health met twice a month for five months preceding the April referendum, planning and implementing an educational and political campaign for fluoridation. A subcommittee structure centered on a number of campaign tasks. The agenda reflected a strong local commitment with knowledgeable and credible spokespersons. Strategy for political success included: no debates or public forums that give antifluoridationists credibility; no distractions from a central theme of better health; less time on technical merits and more time on targeting groups of voters, identifying supporters and encouraging their turnout (see Appendix for further details).

Scientific Press Coverage

Scientific evaluation of the fluoridation issues by an influential press was an asset to the fluoridation initiative. Print media coverage in La Crosse was extensive and well-researched. The *La Crosse Tribune*, with a circulation of 40,000, is the largest newspaper in southwest Wisconsin. The fluoridation issue provoked much public debate as evidenced by 142 letters-to-the-editor (pro and anti) published by the newspaper during the course of the initiative.

It was of great benefit that the health reporter for the *Tribune* (a college chemistry major) was knowledgeable in the history, science, and efficacy of water fluoridation. This resulted in positive and informative health reporting on the issue. Headlines reflected positive leads such as "Fluoride—History Shows No Reason for Controversy—What's the Fuss?" Supportive editorials as one entitled "Let's Get Beyond the Pseudo-Science" appeared throughout the campaign. When spurious newspaper advertisements were run by opponents, follow-up stories refuted erroneous claims. For example, an antifluoridation paid advertisement was entitled "Fluoridation Causes Corrosion of Pipes. Vote No to Fluoridation." The following day an article appeared discrediting the corrosion charge through interviews with the La Crosse Water Utility manager and Centers for Disease Control officials in Atlanta, GA and citing research and fluoridation endorsements from the National Institutes of Health, Bell Laboratories, the American Water Works Association, and the National Association of Corrosion Engineers. The decision of the local press to forego sensationalizing the issue in deference to accurately educating the public was a critical factor toward success.

ADA and Governmental Agency Support

The La Crosse committee utilized a number of sources for consultative support. The American Dental Association (ADA) provided technical and financial support. National experts gave council testimony and committee consultation through ADA sponsorship. The ADA's Water Fluoridation Campaign Package and other written and audio/visual materials were provided. ADA letters of endorsement were sent to the city and the *La Crosse Tribune*.

The Wisconsin Division of Health through the State Dental Director's office provided technical assistance throughout the course of the initiative. Literature reviews, strategy development, and analyses of other fluoridation referenda were utilized by the committee. A state cost-sharing grant for fluoridation equipment was available to the community.

The community also used the services of the US Public Health Service. Consultation and letters of support were utilized through the Dental Disease Prevention Activity of Centers for Disease Control in Atlanta and the US Surgeon General's Office, Washington, DC. The most notable letter of support was from US Surgeon General C. Everett Koop. Dr. Koop's letter to the *La Crosse Tribune*, published just prior to the referendum date, applauded the fluoridation initiative. It was accompanied by a front page article entitled "Surgeon General to Voters: Fluoridate."

Wisconsin Presidential Primary

If fluoridation approval is subject to public referendum, the timing of the ballot is an important factor for success. It is generally perceived that fluoridation referenda should coincide with primary or general elections that promise the largest voter turnout ensuring a broader community participation rather than in smaller special issues elections.[8] The La Crosse referendum was set to coincide with the 1988 Wisconsin Presidential Primary. Fortunately, the Wisconsin Primary in the spring of 1988 was of national significance. Presidential candidates George Bush, Michael Dukakis, Jesse Jackson, and Albert Gore all campaigned in the La Crosse area. Voter interest and turnout was high. A pre-election *La Crosse Tribune*-sponsored poll was conducted by the political science department of the University of Wisconsin-La Crosse found 58 percent of city voters in favor of fluoridation,

27 percent opposed, and 15 percent undecided (Table 2). Support for fluoridation was highest among the better educated and younger voters. Among college graduates, 71 percent favored fluoridation while 62 percent of those with some college and 37 percent of those who never attended college supported the issue. Only 41 percent of citizens over age 65 supported fluoridation. Post-election analysis of voter turnout identified high participation of younger voters who had been targeted by the profluoridation organization.

Chiropractic Support

Chiropractors were among early opponents to fluoridation in the United States. Some continue to oppose fluoridation in many areas as being an infringement of personal freedom, a dictate of organized medicine, and incompatible with chiropractic nutritional philosophy.[10,11] More recently, some chiropractors are active as proponents to fluoridation and several chiropractic organizations are reaching out to the scientific community by endorsing the scientific principles of public health.[12]

For the first time in La Crosse, chiropractors were on both sides of the fluoridation issue. A few chiropractors presented supportive public testimony and actively campaigned for fluoridation. The controversy in the local chiropractic community gained statewide attention, often overshadowing other issues, and may have contributed to a loss of credibility for the primary chiropractic opponents to fluoridation. Sentiments of a proponent chiropractor are reflected in this statement which appeared in a Madison, Wisconsin newspaper: "Chiropractors are interested in public health. Fluoridation is an extremely important issue. Chiropractors have a drugless healing profession, but the feeling that fluoridation is medication is absurd. It is an essential nutrient that naturally occurs."[13]

Summary

The majority of public referenda on water fluoridation in US communities are defeated at the polls. A "confusion hypothesis" has been formulated to explain such a continuing negative voter response to fluoridation initiatives.[7,10,14] Voters are often confused by apparent contradictory information from proponents and opponents of fluoridation. The general public, unable to evaluate highly technical information, credits antifluoridationists as scientific spokespersons. The scientific community has not been all that effective in helping the public understand who is an expert and who is not.[7]

In La Crosse, the Citizens for Better Dental Health were able to overcome a long history of antifluoridation influences through educational and political strategies. Broad-based community support, consultative assistance, scientific press coverage, the timing of the ballot, and chiropractic support all contributed to the success of the initiative.

TABLE 2—Fluoridation Support by Age Group: Pre-Referendum Survey

Age	% Favor	% Oppose	% Undecided
18–25	61	12	27
26–34	66	24	10
35–54	62	28	10
55–64	58	25	17
Over 65	41	48	11
Overall	58	27	15

SOURCE: La Crosse Tribune, March 27, 1988

APPENDIX A
La Crosse Fluoridation Campaign Activities

Citizens for Better Dental Health in La Crosse

Committee with broad-based community coalition.
Endorsement by physicians and dentists.
Endorsement by civic and health organizations (American Cancer Society, local hospitals, Committee on Aging, Chamber of Commerce, AFL/CIO).

Campaign Headquarters

Placed in a highly visible downtown center location.
Opened headquarters with a press conference.
Coordinated campaign activities.
Created headquarters' theme: "Come in and take the taste test" (to counter an anti misinformation campaign regarding the taste of fluoridated water).

Speakers' Bureau

Provided knowledgeable public speakers in support of fluoridation.
Assigned speakers to civic organizations, labor groups, parent associations, and others.

Media Strategies

Identified media contact expert interviewees.
Organized positive and timely letters-to-the-editor.
Developed newspaper and radio advertisements.

Campaign Literature

Designed informational brochures for direct mail distribution, door-to-door literature drop, speaking engagements, and community locations (malls, businesses, professional offices).
Printed and distributed yard and window signs for homes, schools, and businesses.

Fund Raising

Solicited $10,000 in donations from civic/professional organizations, foundations, business, and individuals.
Print design and media production donated by a major local industry.

Getting Out the Vote

Identified and registered fluoridation supporters.
Organized special initiatives i.e., schools (parent-to-parent contact) and university (student voter registration drive).
Arranged car-pooling assistance to the polls.
Assisted with absentee ballots.
Utilized 750 volunteers in the campaign.

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Alfred Yankauer, MD, MPH, to Retire as Journal Editor; Search Committee Actively Seeking New Editor

Alfred Yankauer, MD, MPH, has indicated he plans to retire as editor of the *American Journal of Public Health* next year, effective July 1 1990. The announcement of his retirement appeared in the September issue of *The Nation's Health*, along with information about the newly appointed search committee—which is soliciting nominations for his replacement—and a list of the qualifications for the position of editor.

Dr. Yankauer will complete his 15th year as editor before stepping down next summer; his service spans the period July 1975–July 1990. Only two editors have served longer: George Rosen, MD, who preceded Dr. Yankauer, served for 16 1/2 years (August 1957–December 1973), and Mazÿck P. Ravenel, MD, served 16 years as editor (January 1925–December 1940) and was then named emeritus editor in 1941. All three editors were members of the editorial board or the editorial committee before being named editor. Fourth in line for length of service was C.-E. A. Winslow, DrPH, who held the position for 10 1/2 years (April 1944–November 1954) before retiring for health reasons.

Since the *American Journal of Public Health* was established in 1911, only 11 persons have occupied the position of editor. The four editors named above filled the role collectively for 58 years, while seven others shared the honor a total of 22 years.

In announcing his desire to retire, Dr. Yankauer stated that, "It was a decision not lightly made," because he said he has both enjoyed and learned from the experience. "However, at this point in my life, there are other things that I want and need to do," he added.

Each of the 11 editors of the Journal served that post with distinction and made a lasting impact on the Journal, its readers, the public health profession, and the health of the public. Under their respective leadership over eight decades, the Journal has grown in stature and scientific credibility. Its reputation for accuracy, relevance, and timeliness in the field of peer-reviewed professional journals has been maintained and enhanced by their devotion to its content and to its objectives.

Over the years, the Journal's table of contents has reflected current thinking about the health status of mankind and what needs to be done to improve it. The Journal has been a repository throughout the 20th century for articles, studies, and evaluations authored by hundreds of dedicated health professionals.

As the Editor Search Committee carries out its difficult task of identifying potential candidates for the position of Journal editor, they will be seeking an individual with broad public health interests, a strong historical perspective, an abiding concern for the health and well-being of others, and exceptional vision.

Nominations for Journal editor should be submitted as soon as possible, so that the search committee can complete its deliberations by the end of this year. Names of potential candidates, along with support materials, should be sent to the Editor Search Committee, c/o Executive Director's Office, American Public Health Association, 1015 15th Street, NW, Washington, DC 20005. A list of the criteria for selection of an editor appeared in the September issue of *The Nation's Health*, or may be obtained on request to APHA headquarters. Tel: (202)789-5600.

Public Health Briefs

Cocaine and Heroin Dependence Compared: Evidence from an Epidemiologic Field Survey

JAMES C. ANTHONY, PHD, AND KENNETH R. PETRONIS, MS

Abstract: Analyzing self-report interview data from a multi-site collaborative study of mental disorders in the community, we examined suspected differences between 611 cocaine users and 126 heroin users in relation to indicators of drug dependence. Cocaine users were less likely to report dependence-related problems as compared to heroin users. These epidemiologic sample data underscore concerns about the generalizability of published clinical sample data on self-reported cocaine dependence. (*Am J Public Health* 1989; 79:1409–1410.)

Introduction

Recently Hasin, *et al*, described 75 cocaine users and 67 heroin users, drawn from 308 patients in an alcohol treatment unit. Answering standardized drug questions in the Diagnostic Interview Schedule (DIS), cocaine users reported having felt dependent on cocaine just as frequently as heroin users reported having felt dependent on heroin. Further, cocaine users and heroin users differed little on other reported dependence indicators (withdrawal, loss of control, tolerance). While citing this as evidence that cocaine has dependence-generating potential comparable to that of heroin, the authors cautioned that drug users in treatment might have had different experiences than users in the general population.[1]

Previously, our research group reported on cocaine use and cocaine problems in the community, based on DIS data from the National Institute of Mental Health (NIMH) Epidemiologic Catchment Area (ECA) Program.[2] For this paper we re-analyzed the data for direct comparison to the data reported by Hasin.[1]

Methods

Collaborators at four ECA sites obtained interviews with 13,538 subjects between 1981 and 1984 after drawing probability samples of adult household residents in Baltimore, Maryland, St. Louis, Missouri, Durham, North Carolina, and Los Angeles, California. To assess psychiatric conditions, including drug-related problems, staff administered the DIS soon after sampling. The mean participation rate was 76 percent (range 68–79 percent). (New Haven, Connecticut ECA data sets do not include drug variables needed for this analysis.)

Hasin, *et al*, describe psychometric characteristics of the DIS.[1] Elsewhere, we have published data from a DIS validity study,[3] descriptions of DIS drug data,[2,4,5] and reports on sampling and other methods used in the ECA program.[6,7]

Analyses for this paper follow specifications of the clinical sample paper. The results shown are unweighted characteristics of the sample; use of sampling weights increases differences between the two studies.

Results

Among ECA subjects, 57 reported having used heroin every day for at least two weeks ("daily heroin use"); 69 reported using heroin at least six times, but never daily for two weeks. An additional 611 subjects reported having used cocaine (but not heroin) at least six times; 69 of these users reported having used cocaine every day for at least two weeks ("daily cocaine use"). These users were sociodemographically similar to those in the clinical sample (data available upon request to authors). Whereas alcohol disorders affected virtually all of the clinical sample, in the ECA sample they affected only 35 percent of the non-daily cocaine users, 52 percent of the daily cocaine users, and about 60 percent of the heroin users.

Table 1 shows for daily and non-daily cocaine and heroin users in the ECA sample the proportion who reported feeling dependent on cocaine, unsuccessfully cutting down on cocaine use, cocaine tolerance, or cocaine withdrawal.

ECA cocaine users were less likely to report cocaine-related problems than users in Hasin's study: 2.6 percent of ECA non-daily users and 22.1 percent of ECA daily users reported feeling dependent on cocaine compared to 14 percent and 79 percent, respectively, in Hasin's study; for unsuccessfully attempting to cut down on cocaine use, corresponding figures were 2.4 percent and 11.6 percent vs 14 percent and 64 percent; for tolerance, 7.7 percent and 42.0 percent vs 26 percent and 64 percent. Finally, 1.0 percent of ECA non-daily users and 13.0 percent of daily users reported feeling withdrawal sickness upon stopping or cutting down on cocaine use, as compared to 19 percent and 76 percent in Hasin's sample.

Our sample and Hasin's sample also differed in relation to heroin experience, although not as markedly as with cocaine. Among ECA non-daily and daily heroin users, respectively, 7.2 percent and 78.6 percent reported having felt dependent on heroin, as compared to 11 percent and 85 percent in Hasin's study. For loss of control, comparable figures were 1.5 percent and 40 percent vs 16 percent and 63 percent; for heroin tolerance, 12 percent and 68.4 percent vs

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TABLE 1—Dependence Indicators for Cocaine and Heroin, by Patterns of Use, based on Data from the Epidemiologic Catchment Area Surveys in Baltimore, St. Louis, Durham, and Los Angeles, 1980–84

	Cocaine				Heroin			
	Group 1 (N = 542 6+ times, Never daily for 2 weeks)		Group 2 (N = 69 Daily 2 weeks or longer)		Group 3 (N = 69 6+ times, Never daily for 2 weeks)		Group 4 (N = 57 Daily 2 weeks or longer)	
Dependence Indicators	%	n	%	n	%	n	%	n
Ever felt dependent on drug	2.6 (4 missing)	14	22.1 (1 missing)	15	7.2	5	78.6 (1 missing)	44
Ever unable to cut down on amount used	2.4	13	11.6	8	1.5	1	40.4	23
Ever felt tolerance to drug's effects	7.7 (7 missing)	41	42.0	29	11.8 (1 missing)	8	68.4	39
Ever experienced withdrawal	0.9 (3 missing)	5	13.0	9	10.1	7	75.4	43

Differences in proportions between groups 1 vs 3 and groups 2 vs 4 were as follows (95% confidence limits):

	Group 1 vs. Group 3	Group 2 vs. Group 4
Felt dependent	−4.6 (−10.9, 1.6)	−56.5 (−71.1, −41.9)
Unable to cut down	0.9 (−2.2, 4.1)	−28.8 (−43.6, −14.0)
Tolerance	−4.1 (−12.1, 3.9)	−26.4 (−43.2, −9.6)
Withdrawal	−9.2 (−16.4, −2.1)	−62.4 (−76.1, −48.7)

26 percent and 85 percent; for heroin withdrawal experience, 10 percent and 75 percent vs 21 percent and 83 percent.

Unlike the cases reported by Hasin, ECA daily cocaine users were much less likely than daily heroin users to report problems, as shown by negative signs on the upper confidence limits for each estimated difference (Table 1).

Discussion

This epidemiologic study's evidence calls into question the generalizability of results from the prior study of patients in an alcohol treatment unit. Responding to identical self-report interview questions, smaller proportions of cocaine users in the community sample reported dependence-related problems. Moreover, daily cocaine users in this sample were substantially less likely to report dependence-related problems, as compared to daily heroin users.

One hypothesis about these differences in study results is that drug users in alcohol treatment are more willing to give a complete report about drug-related problems. Alternatively, alcoholism or admission to alcoholism treatment may index especially intensive patterns of drug use or increased risk for problems with other drugs, especially cocaine. If so, clinical samples are unreliable sources of data on the frequency of drug hazards in the community, as discussed elsewhere.[1,2]

Putting these results in perspective, there is strong laboratory evidence on reinforcing properties of cocaine. Across several species, cocaine is self-administered repetitively in patterns that fulfill broadly accepted case definitions for drug dependence.[8,9] These interview data do not contradict that evidence.

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Improving Patient Comprehension of Literature on Smoking

CATHY D. MEADE, RN, PHD, JAMES C. BYRD, MD, MPH, AND MARTHA LEE, PHD

Abstract: This study was designed to determine if simplification of smoking literature improved patient comprehension. Subjects (N = 129) were randomized in three groups: to receive no smoking booklet, receive one written at the 5th grade, or the 10th grade reading level. Subjects receiving the 5th grade booklet showed 13 percent better comprehension than those receiving the 10th grade booklet, and 18 percent better than the controls. Comprehension of written smoking materials can be improved by adjustment of the reading grade level. (*Am J Public Health* 1989; 79:1411–1412.)

Introduction

Printed materials on smoking are commonly distributed to patients in hospitals, clinics, or smoking cessation programs.[1–5] Such materials communicate, reinforce, and extend the educational message presented verbally by practitioners.[6,7]

Studies indicate that the median level of most patient educational materials is grade 10, a reading level often greater than the majority of its intended target population.[8–13] In a review of 49 educational brochures specific to smoking, the median reading level was 10.5 while the median reading level of patients (N = 258) was grade 6.[14]

Comprehension and acceptability of written materials should improve if materials are made easier to read; however, few data are available to either support or refute this idea. Reported studies have lacked control groups, pretest measures,[15–17] randomization, or description of reliability of the instruments.[15–16] No study reported has yet evaluated the effect of simplification of smoking literature on patient comprehension. We report here such a study.

Methods

Two booklets on smoking, each entitled "Quit Smoking and Feel Good," were developed based on a review of the smoking literature and a panel of experts. The booklets mirrored each other in color, design, writing style, print type, length, and organization. The Minnesota Interactive Readability Approximation Microcomputer Program was used to adjust the readability level of one booklet to grade 5 and the other to grade 10.[18]

To evaluate smoking knowledge and recall, 24 questions were developed by the investigators with a panel of experts based upon the content of the booklets. The reliability of the test was found to be at an acceptable level (r = 0.73 Cronbachs alpha).[19] Questions were systematically divided into two 12-question tests (pretest and posttest) based on results of the reliability measures, item difficulty, logical pairing of questions, and question content. The pretest and posttest were each comprised of six multiple choice and six true/false questions.

Smoking subjects (N = 129) were selected from three Primary Care clinics based in a public hospital, a veterans hospital, and a community health center. Selection criteria included being 18 years or older, English speakers, English readers, visually able to read 12 point typeset, and able to give free consent.

Subjects were randomly placed into one of three groups: control, experimental group 5 (E5), or experimental group 10 (E10). All subjects were first administered the pretest. Control group subjects (N = 44) received no smoking booklet. Subjects in E5 (N = 44) received a smoking booklet written at grade 5 reading level, those in E10 (N = 41) one written at a grade 10 reading level.

All subjects were administered the posttest, the Wide-Range Achievement Test (WRAT) Level II (a word recognition test which estimates reading grade level)[20] and were then asked questions about their educational background and other demographic information.

Results

The sample was comprised of 74 males (57 percent) and 55 females (43 percent); ages ranged from 18 to 88 with a mean of 43 years. There were 85 Blacks (66 percent) and 44 Whites (34 percent). Chi-square analyses revealed that there were no significant differences in sex, ethnic background, income, education, or WRAT between control, E5, or E10. Age was significantly different between the groups, with a mean of 49 years for control, 41 years for E5, and 40 years for E10. The mean grade level of reported education among subjects was grade 11 and the mean grade level according to the WRAT was grade 7, a difference of 4 grades (paired t = 12.62, 95% CI: 3.31, 4.52).

Prescores, postscores, and score differences were calculated for the three groups (Table 1). The score improvement (Postscore − Prescore) ÷ Prescore, showed a 4 percent increase in scores for the control group, a 22 percent increase for E5, and a 9 percent increase for E10. Using one-way analysis of variance, no significant differences were found in the prescores between control, E5, or E10. Significant

TABLE 1—Summary of Scores by Booklet Group

Range	Booklet Groups		
	Control (N = 44)	E5 (N = 44)	E10 (N = 41)
Prescore (3,12)			
Mean	6.68	7.25	7.17
SD	2.42	2.16	2.24
Postscore (1,12)			
Mean	6.93	8.82	7.80
SD	2.50	2.33	2.84
Score Difference (−6,6)			
Mean	0.25	1.57	0.63
SD	2.28	2.54	2.38
95% CI	(−0.44,0.94)	(0.80,2.34)	(−0.12,1.39)
Score Improvement (Postscore − Prescore) ÷ Prescore	3.7%	21.6%	8.7%

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TABLE 2—Comparison of Scores by Booklet and WRAT

Groups	Booklet*/ WRAT	N	Prescore (Mean ± SD)	Postscore (Mean ± SD)	Score Difference	95% CI
I	Low/low	23	6.43 ± 1.75	7.87 ± 2.69	1.43	(0.21,2.66)
II	Low/high	21	8.14 ± 2.20	9.86 ± 1.24	1.71	(0.70,2.73)
III	High/low	20	5.90 ± 2.00	6.05 ± 2.86	0.15	(−1.11,1.41)
IV	High/high	21	8.38 ± 1.77	9.48 ± 1.54	1.10	(0.19,2.00)

*Low booklet = 5th grade reading level booklet
High booklet = 10th grade reading level booklet
High WRAT = WRAT score > grade 7
Low WRAT = ≤ grade 7

differences were found between the groups in their postscores ($p = 0.0033$), and in their raw score differences ($p = 0.0335$).

Subjects in groups E5 and E10 ($N = 85$) were subdivided into four groups according to their WRAT score and grade level of booklet received (Table 2). Definitions for high and low booklet and high and low reader were based on expected reading proficiency of subjects within a two year range of the readability of the available study booklets. A comparison of scores was then conducted to investigate differences between the groups.

Results indicated that subjects with high WRAT scores had higher prescores and postscores than subjects with low WRAT scores. The largest score differences (1.43 and 1.71) were noted in the groups (I and II) who received the fifth grade booklet regardless of their WRAT scores. The group which exhibited the smallest increase in score difference (0.15) was the low reader who received the tenth grade booklet (Group III).

Using the prescore and WRAT as covariates, results indicated that the booklet had significant main effects on score differences ($p = 0.0002$). Adjusted score difference means for control was 0.08 (SEM = 0.3028), for E5 was 1.73 (SEM = 0.3005), and for E10 was 0.81 (SEM 0.3115).

Discussion

Since education has been noted as a factor in smoking cessation programs, success may be related to the participants' acceptability and understandability of the information.[1] Our results support the need for continued development of educational materials at low reading levels. Producers of literature on smoking need to apply reading analyses to their texts. Readability formulas can assist in estimating the reading level of materials by offering an objective measure of text difficulty. Factors such as format, visual appeal, patient interest, and motivation also need to be considered when writing health messages.

We suggest that user tests (pretests and posttests) be incorporated into smoking cessation programs to help determine if a message is being comprehended by the participant. Such tests can offer information on clients' smoking knowledge over time when planned interventions are administered.[21–23]

Findings also indicated that educational level is a poor predictor of reading ability. Years of schooling do not identify reading ability, a finding supported by other investigators.[9,11,14,24] We suggest that an instrument such as the WRAT be used as a more accurate predictor of reading skills. The WRAT can be administered and scored in a manner of minutes.

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Cigarette and Smokeless Tobacco Use among Connecticut Adolescents

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Abstract: Despite data that the rate of boys' cigarette use has declined nationwide, boys' total tobacco use may actually have remained high because of their substantial use of smokeless tobacco. We surveyed anonymously a random sample of entire classes of grade 7–12 boys and girls from 59 Connecticut towns concerning tobacco use. Despite girls' higher daily cigarette use, boys' total daily tobacco use was higher than that of girls in grades 7–9, and only slightly lower in grades 10–12. (*Am J Public Health* 1989; 79:1413–1414.)

Introduction

Boys' cigarette use has generally been perceived as having peaked in the late 1960s (14.7 percent regular use in 1968 across grades 7–12), declined by 1979, and stabilized[1] with girls smoking more than boys. In a 1979 survey of grades 7–12, girls reported regular smoking at a rate of 12.7 percent and boys 10.7 percent.[1] The annual National Institute on Drug Abuse (NIDA) survey of high school seniors indicates a daily smoking rate of 18.7 percent in both 1986 and 1987 and a monthly rate of 29.4 percent in 1987, consistent since 1979.[2]

The mid-1970s might have marked the beginning of a large increase in adolescent smokeless tobacco use, first recognized in print a few years later.[3,4] By the time of the Surgeon General's 1986 report on smokeless tobacco,[5] it was apparent that many boys were using smokeless tobacco in grade school and continuing its use throughout high school. Minimal smokeless tobacco use by females has been reported except in particular regional pockets.[6–11] Since many effects of the two products are identical, it seems appropriate to document total tobacco use for both girls and boys.

As part of our research on smokeless tobacco use, we conducted a 1987 statewide survey in Connecticut assessing total tobacco use in grades 7–12. Based on surveys,[5,12] and on earlier data from Massachusetts,* we expected to find relatively low smokeless tobacco use.

Methods

Subjects and Design

All 169 Connecticut towns were blocked by population density and socioeconomic status (SES). Within the resultant matrix, proportional numbers of towns were chosen randomly, and middle and high schools were selected randomly from these towns. Requests were made sequentially to public school superintendents (75 percent agreed) and then to principals (89 percent agreed) for permission to collect anonymous, self-administered student questionnaires. Schools whose superintendents or principals refused permission were replaced with schools of similar population density and SES. Schools were asked to choose entire classrooms of students such that either regular or a combination of high and low academic track students would be represented. Further blocking by sex and grade within school was carried out in order to collect approximately 3 percent of all Connecticut students in grades 7–12 representing public, town-based schools.

Questionnaires, which were sent to and returned from schools by mail, took approximately 35 minutes to complete. Teachers were instructed to administer the questionnaires in a standardized fashion to students present at day of assessment.

Smokeless tobacco and cigarette daily use rates were determined separately by respondents' selection of one of 10 use-rate categories. Separate questions assessed "chewing leaf tobacco" and "dipping oral snuff." Previous focus group discussions had indicated that few adolescents used leaf tobacco, but nevertheless called both forms "chew." Although data were collected concerning brand names purchased and used, the general terms "smokeless tobacco" or "chew" best capture student terminology in this sample.

Due to school-required anonymity and the extensive sample size desired, no biochemical verification procedures were employed and no attempt was made to track absentees or dropouts, making these data comparable to earlier surveys. Accordingly, younger students may have underreported tobacco use more than older students and smokeless tobacco may have been underreported more than cigarettes. We received 7,457 usable questionnaires from 106 schools from 59 towns.

Results

Cigarette Use

As expected, there was a positive linear grade trend for smoking peaking at grade 11. Averaged across all grades, boys (11.2 percent) and girls (13.8 percent) reported smoking cigarettes daily at statistically different rates (see Table 1). The reversal from the boys' higher daily use rates in grades 7–9 (9.1 percent versus girls' 7.6 percent) to grades 10–12 (boys, 13.2 percent; girls, 20.7 percent) reflects a significant sex by grade interaction.

Smokeless Tobacco Use

Only 5.7 percent of girls had ever tried smokeless tobacco compared to 36.7 percent of the boys, a reliably large difference. Daily use by girls across the entire sample was only 0.3 percent, while daily use for boys across all grades averaged 5.5 percent, reaching 8.0 percent by grade 12. Weekly rates, although higher, paralleled daily rates. For boys, the higher the grade, the higher the daily smokeless tobacco use.

Total Tobacco Use

Total daily tobacco use, defined as daily use of either cigarettes or smokeless tobacco, increased significantly by grade, peaking at grade 11. Averaged across all grades, total tobacco use was comparable for girls (14.1 percent) and boys (14.8 percent). Like cigarette use, total tobacco use was

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TABLE 1—Percent Daily Substance Use by Adolescents as a Function of Substance, Sex, and School Grade

Sex	Grades						
	7	8	9	10	11	12	Total
Male							
Cigarettes Only	6.2	6.4	11.0	10.8	13.6	11.1	9.4
Smokeless Only	1.3	3.6	4.4	3.0	4.3	6.8	3.6
Cigarette and Smokeless	.7	1.7	2.7	2.5	2.8	1.2	1.8
Total Tobacco	8.2	11.7	18.1	16.3	20.7	19.1	14.8
Total (*n*)	(839)	(698)	(528)	(591)	(506)	(503)	(3,667)
Female							
Cigarettes Only	4.1	8.1	10.8	18.0	23.0	21.1	13.8
Smokeless Only	.4	.3	.5	.0	.3	.2	.3
Cigarette and Smokeless	.0	.1	.0	.0	.0	.0	.0
Total Tobacco	4.5	8.5	11.3	18.0	23.3	21.3	14.1
Total (*n*)	(702)	(670)	(627)	(606)	(640)	(545)	(3,790)
Total							
Cigarettes Only	5.3	7.2	10.9	14.5	18.8	16.3	11.6
Smokeless Only	.9	2.0	2.3	1.5	2.1	3.3	1.9
Cigarette and Smokeless	.4	1.0	1.2	1.3	1.2	.6	.9
Total Tobacco	6.6	10.2	14.4	17.3	22.1	20.2	14.4
Total (*n*)	(1,541)	(1,368)	(1,155)	(1,197)	(1,148)	(1,048)	(7,457)

NOTE: Use of a cigarette is derived in the text by combining the rate of cigarette use with cigarette plus smokeless tobacco use. Smokeless tobacco use is calculated in analogous fashion.

higher for boys (12.0 percent) than girls (8.0 percent) for grades 7–9, while girls' use (20.9 percent) was higher than boys (18.5 percent) for grades 10–12.

Discussion

Boys' cigarette use seemed to fluctuate less by grade than did girls' use. However, between one-fourth and one-half of total daily tobacco use by boys is from smokeless tobacco. As noted in previous research,[14] the lower overall use of tobacco in grade 12 might be accounted for by a higher school dropout rate for tobacco users. Data from Connecticut, a relatively low-use state for smokeless tobacco, suggest previous underestimation of the extent of smokeless tobacco use among adolescent boys in the Northeast. This implies that the national reduction and comparatively low rate of boys' cigarette smoking over the past decade may have been compensated for by an increase in smokeless tobacco use. In fact, boys' total tobacco use may have been comparable to girls' use over these years.

Accordingly, school-based educational efforts need to focus on use of both substances. Questionnaire responses indicated that smokeless tobacco is popular because of the belief that "it is less harmful than cigarettes," "does not hurt others," and "can be done anywhere." While health education may have had an impact on knowledge and attitudes concerning cigarette smoking, smokeless tobacco use is thus not defined by male adolescents as equally dangerous. In Connecticut, legislation to restrict school tobacco use has rarely included smokeless tobacco.

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Mentholated Cigarette Use among Multiphasic Examinees, 1979–86

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Abstract: Mentholated cigarette use was studied in relation to age and race in 29,037 current smokers who were Kaiser Permanente Medical Care Program members. The percentages of mentholated cigarette users were much higher in Blacks and Asians than in Whites, especially in the younger age groups. A marked inverse relationship between mentholated cigarette use and age was present in Blacks and Asians; mentholated cigarette use showed little difference with age in Whites. (*Am J Public Health* 1989; 79:1415–1416.)

Introduction

For a prospective cohort study to determine the health effects of smoking low-yield cigarettes, information was collected from 1979 to 1986 about the smoking habits of members of Kaiser Permanente Medical Care Program who participated in multiphasic health check-ups. Data from this study regarding mentholated cigarette use were analyzed for this report to determine whether previous reports of greater mentholated cigarette use in Blacks would be confirmed in our study population and to study age trends.[1,2] Data regarding Asians are included in this report because of the substantial number of Asian smokers in the study population.

Methods

An automated multiphasic health checkup has been offered since 1964 to about 25,000 persons per year at the Kaiser Permanente Medical Care Program medical centers in Oakland and San Francisco.[3] Starting in July 1979, examinees were asked to complete a supplementary questionnaire that explored their smoking habits in detail.[4] In mid-1980 the check-up was discontinued at the San Francisco facility. By the end of 1986, the questionnaire had been completed by 114,934 examinees (approximately 86 percent of the examinees), of whom 31,428 (27.3 percent) were current smokers. Information on whether or not the cigarettes were mentholated was available for 92 percent of the current smokers. The tar yield was obtained from Federal Trade Commission estimates for each brand and type of cigarette.

If subjects had more than one check-up, only the first was included in this analysis. Age-adjusted proportions of mentholated cigarette users were obtained by the direct method, with the age distribution of the entire study population used as the standard. Mentholated cigarette use habits were examined in the 29,037 current smokers ages 15–79 years of Black, White, or Asian race.

Results

The sex, race, and age distribution of the current smokers is shown in Table 1. The percentage of mentholated cigarette use was higher in women than in men in all age groups for Blacks and Whites, and in most age groups for Asians (Figure 1). There was a marked inverse relationship between age and mentholated cigarette use in Blacks and in Asians, while there was relatively little difference in mentholated cigarette use with age in Whites. Blacks had the highest age-adjusted proportion of mentholated cigarette users (55.5 percent for females, 49.9 percent for males), Asians the second highest (47.9 percent, females, 35.7 percent, males), and Whites the lowest (27.1 percent, females, 18.1 percent, males).

The mean number of cigarettes smoked per day (adjusted for age, race, and sex) was slightly lower in mentholated than in nonmentholated users: 16.2 versus 17.1; 95% confidence interval of the difference = −1.2, −0.7. Mentholated cigarette users were similar to nonmentholated users in their reports of how often they inhaled, depth of inhalation, and how much of the cigarette they smoked. Alhough the mean tar yield of mentholated cigarettes was lower than that of nonmentholated cigarettes for both Blacks (15.5 mg vs 16.0 mg) and Whites (13.8 mg vs 14.0 mg), the mean tar yield of cigarettes smoked by Blacks (adjusted for age and sex) was greater than that of Whites (15.9 mg vs 14.1 mg, 95% CI of the difference 1.7, 2.0).

The switching patterns between mentholated and nonmentholated brands were examined in the 1,688 Black smokers under the age of 40 years for whom follow-up information regarding smoking habits was obtained by a mailed questionnaire (mean interval between baseline questionnaire and follow-up was 4.5 years). Follow-up information was obtained on a slightly greater proportion of nonmentholated than mentholated cigarette users (32 vs 28 percent). The percentage of quitters was slightly higher in nonmentholated users than in mentholated users (17.5 vs 15.4 percent), while the percentage of switchers (from nonmentholated to mentholated or vice-versa) was much higher in the nonmentholated users (14.6 vs 3.6 percent). Using a proportional hazards model to adjust for age and sex and to account for varying length of follow-up, nonmentholated users were 4.2 times more likely to switch than mentholated users (95% CI 2.8, 6.4), while quitting was equally likely in both groups.

Discussion

As others have reported, we found a much higher proportion of mentholated cigarette use in Black smokers than in White smokers. There is a very strong inverse relationship between age and mentholated cigarette use in Blacks. The data suggest that

TABLE 1—Number of Current Cigarette Smokers by Age, Sex, and Race

	Age (years)							
	15–19	20–29	30–39	40–49	50–59	60–69	70–79	Total
Male								
Black	86	777	1323	1031	678	342	57	4294
White	108	1275	2346	1442	1219	769	193	7352
Asian	23	272	454	286	187	74	14	1310
Female								
Black	249	1816	2046	1238	776	302	32	6459
White	238	1914	2441	1523	1452	1023	252	8843
Asian	35	242	216	140	103	42	1	779

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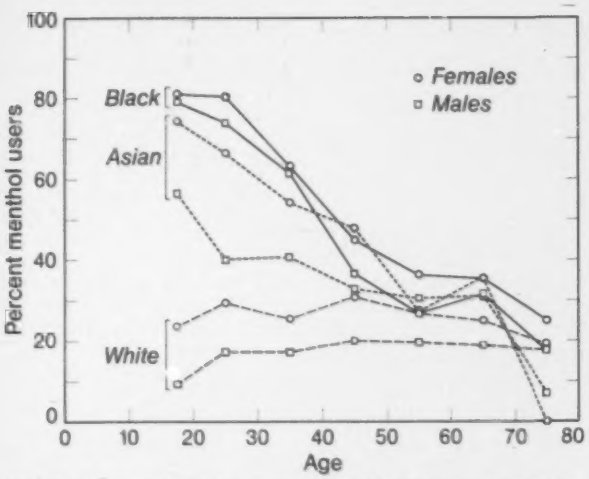

FIGURE 1—Percent Menthol Users among Current Cigarette Smokers by Race, Sex, and Age

the steep decline in the proportion of mentholated cigarette users with age in Blacks can be explained predominantly as a cohort effect, since the findings regarding switching from cigarettes of one mentholation status to another in this study suggest that the prevalence of mentholated cigarette use increases over time in younger Black smokers.

There are few data on the health effects of mentholated cigarettes. A recent case-control of esophageal cancer showed no significant change in risk due to mentholated cigarette use.[5] Our findings have potentially important implications for research into the health effects of mentholated cigarettes. The rates of some smoking-related cancers (e.g., esophagus, lung) are more common in Blacks, at least in part because Blacks are more likely to be smokers.[4] A theoretical basis for a causal relationship between mentholation and cancer exists in that menthol combustion produces carcinogenic compounds such as benzo[a]pyrenes.[5] It has also been hypothesized that the cooling sensation due to menthol may allow deeper, prolonged inhalation of cigarette smoke, thereby increasing the effective yield of tar and other toxic chemicals from the cigarette.[5] This study did not show self-reported differences in inhalation habits between Blacks and Whites, but that may well be due to the anesthetizing effects of menthol.

If mentholation does cause cancer, it may contribute substantially to the excess incidence of certain smoking-related cancers in Blacks relative to Whites. A potential consequence of the very large differences in the prevalence of mentholated cigarette use in younger Blacks relative to Whites would be a widening of the difference between Blacks and Whites of these incidence rates in the future, when younger Blacks reach the higher risk age range for cancer.

ACKNOWLEDGMENTS

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State-Specific Progress Toward the 1990 Objective for the Nation for Cigarette Smoking Prevalence

PATRICK L. REMINGTON, MD, MPH, THOMAS E. NOVOTNY, MD, DAVID F. WILLIAMSON, MS, PHD, AND ROBERT F. ANDA, MD, MS

Abstract: We predicted the smoking prevalences for 1990 for each state in the US, assuming that the decline in each state from 1985–1990 would be the same as the decline in the US from 1965–1985. In 1985, only three states had smoking prevalences less than 25 percent. Based upon the observed decline in smoking in the US from 1965–1985 of 0.5 percent per year, we predict that only seven states will have smoking prevalences less than 25 percent by 1990. States need to consider current smoking prevalence and achievable rates of decline when setting objectives for 1990 and beyond. (*Am J Public Health* 1989; 79:1416–1419.)

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Introduction

As part of its 1990 Health Objectives for the Nation, the US Public Health Service has established a goal that by 1990 the proportion of adults who smoke should be reduced to less than 25 percent.[1] A recently published midcourse review indicated that this objective likely will be met.[2] Despite the apparent success at the national level, however, the variation in the prevalence of smoking from state to state[3] suggests that a prevalence of smoking of less than 25 percent may not be an achievable objective for all states. To address this issue, we predicted 1990 smoking prevalences for all 50 states and the District of Columbia by applying a range of estimates of decline in smoking prevalence to the observed state-specific smoking prevalences for 1985.

Methods

The state-specific smoking prevalence estimates were based on data obtained in September 1985 from the Current

Population Survey conducted by the Bureau of the Census.[4] In the Current Population Survey, 60,000 households were surveyed in 1,973 counties, cities, and minor civil divisions in the United States. The data were weighted by age, sex, and race to represent the total civilian, non-institutionalized population of each state. To have estimates comparable with previously published national data, we included only persons 20 years of age or older. Current smokers were defined as persons who had smoked 100 or more cigarettes in their lifetimes and who currently smoked. When respondents were not available at the time of the household interview (59 percent of men, 27 percent of women, 42 percent overall), their smoking status was provided by proxy.

We used data from the National Health Interview Surveys from 1965, 1976, 1980, and 1985 to estimate various rates of decline in smoking prevalence among all adults 20 years of age and older. These estimates were age-adjusted to the 1985 US adult population.[5] A current smoker was defined as a person who had smoked at least 100 cigarettes and who smoked at the time of the survey. Proxy responses were not used in the National Health Interview Surveys.

The annual rate of decline in smoking prevalence from 1965–1985 was applied to each state's smoking prevalence in 1985, to project a smoking prevalence for 1990. To account for possible variability in the rate of decline from state to state, we performed a sensitivity analysis. We similarly projected state-specific smoking prevalences for 1990 using 2 times and ½ times the observed rate of decline from 1965–1985. This range in the rate of decline was subsequently compared to the rates of decline in each inter-survey period (1965–76, 1965–80, 1976–80, 1976–85, and 1980–85) in the US. We used the actual state prevalence estimates in all calculations, rounded to the nearest percent for presentation.

Results

In the United States between 1965 and 1985, the age-adjusted prevalence of smoking among adults 20 years of age or older decreased from 40.4 to 30.4 percent, a rate of decline of 0.50 percent per year. In 1985, state-specific smoking prevalences ranged from 15 percent in Utah to 37 percent in Nevada; only three states had smoking prevalences less than 25 percent (Figure 1). If the observed annual rate of decline in the United States from 1965–1985 occurs in each state from 1985–1990, only four additional states will have smoking prevalences less than 25 percent by 1990.

The results of the sensitivity analysis are presented in Table 1. If the decline from 1985–1990 is one-half the observed decline from 1965–1985 (0.25 percent per year), then only three states will have prevalences less than than 25 percent by 1990. Conversely, if the annual decline from 1985–1990 is two times the observed national decline from 1965–1985 (1.0 percent per year), then 25 states will have prevalences less than 25 percent.

The lower bound of the estimate for decline in smoking used in the sensitivity analysis (0.25 percent per year) is less than any decline observed between 1965–1985 (0.39 percent per year between 1965–1976). Similarly, the upper bound of the estimate (1 percent per year) is greater than any decline observed during that period (0.7 percent per year from 1976–1980). When these are applied to the 1985 US smoking prevalence, we predict a smoking rate for the US in 1990 of 27.9 percent (range 25.4 percent to 29.1 percent) (Figure 2).

Discussion

Using national data on the decline in smoking prevalence, we predicted the prevalence of smoking for each state for 1990. We have shown that few states will have smoking prevalences less than 25 percent by 1990, assuming that the decline observed in the United States from 1965–1985 occurs

TABLE 1—State-Specific Smoking Prevalences in 1985 and Predicted for 1990

States	Smoking prevalence in 1985** (%)	Annual Decline in Smoking from 1985–1990* .25%/year Projected Smoking Prevalence in 1990 (%)	.5%/year (%)	1%/year (%)
Alabama	30	29	28	25
Alaska	36	35	34	31
Arizona	30	28	27	25
Arkansas	31	29	28	26
California	26	25	23†	21†
Colorado	29	28	26	24†
Connecticut	29	28	27	24†
Delaware	32	31	29	27
District of Columbia	31	30	29	26
Florida	31	29	28	26
Georgia	32	31	30	27
Hawaii	27	26	25	22†
Idaho	24†	23†	21†	19†
Illinois	29	28	26	24†
Indiana	33	32	31	28
Iowa	28	26	25	23†
Kansas	30	29	27	25
Kentucky	36	35	33	31
Louisiana	29	28	27	24†
Maine	29	28	27	24†
Maryland	30	29	27	25
Massachusetts	28	27	26	23†
Michigan	34	33	32	29
Minnesota	29	28	26	24†
Mississippi	31	30	28	26
Missouri	28	26	25	23†
Montana	26	25	23†	21†
Nebraska	24†	23†	22†	19†
Nevada	37	36	34	32
New Hampshire	31	29	28	26
New Jersey	28	26	25	23†
New Mexico	28	27	26	23†
New York	29	27	26	24†
North Carolina	32	30	29	27
North Dakota	27	25	24†	22†
Ohio	32	31	30	27
Oklahoma	33	32	30	28
Oregon	27	26	25	22†
Pennsylvania	29	27	26	24†
Rhode Island	34	33	32	29
South Carolina	27	26	25	22†
South Dakota	28	27	26	23†
Tennessee	31	29	28	26
Texas	31	30	28	26
Utah	16†	14†	12†	10†
Vermont	31	29	28	26
Virginia	33	32	31	28
Washington	28	27	26	23†
West Virginia	33	32	31	28
Wisconsin	26	25	24†	21†
Wyoming	32	31	30	27
States below 25% (number)	(3)	(3)	(7)	(25)

*Trend data from the National Health Interview Surveys were used to estimate the range in rates of decline in smoking for each state from 1985 to 1990. The 1990 projected prevalences were made using the actual 1985 state-specific prevalence but are rounded to the nearest percent for presentation.

**The prevalences for 1985 are from the 1985 Current Population Surveys, for adults 20 years of age or older.

†Smoking prevalence less than 25 percent.

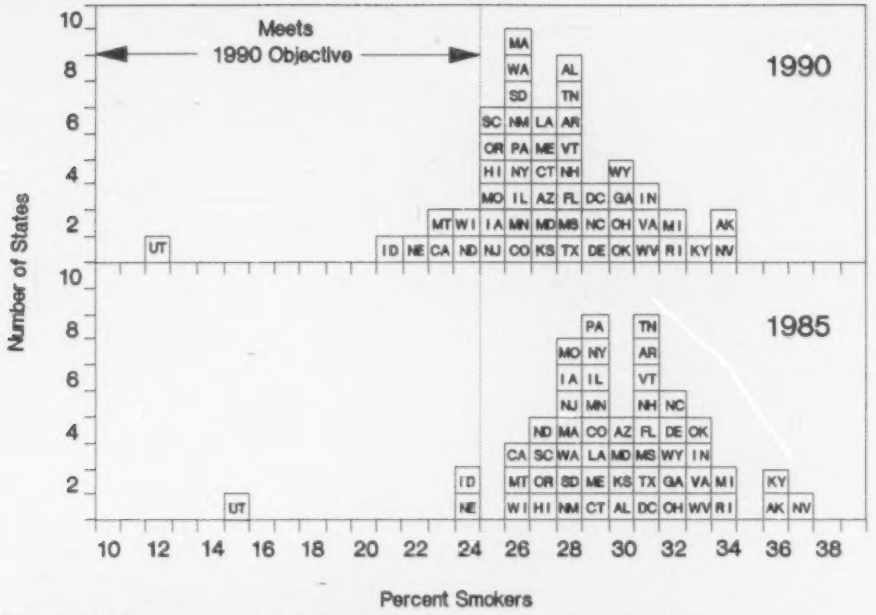

FIGURE 1—Distribution of State-Specific Smoking Prevalences in 1985 and Projected for 1990
Data for 1985 are from the Current Population Surveys for adults 20 years of age or greater. Rates for 1990 are projected assuming a 0.5% annual decline in smoking in each state from 1985–1990. Each square is identified with its 2-digit state abbreviation.

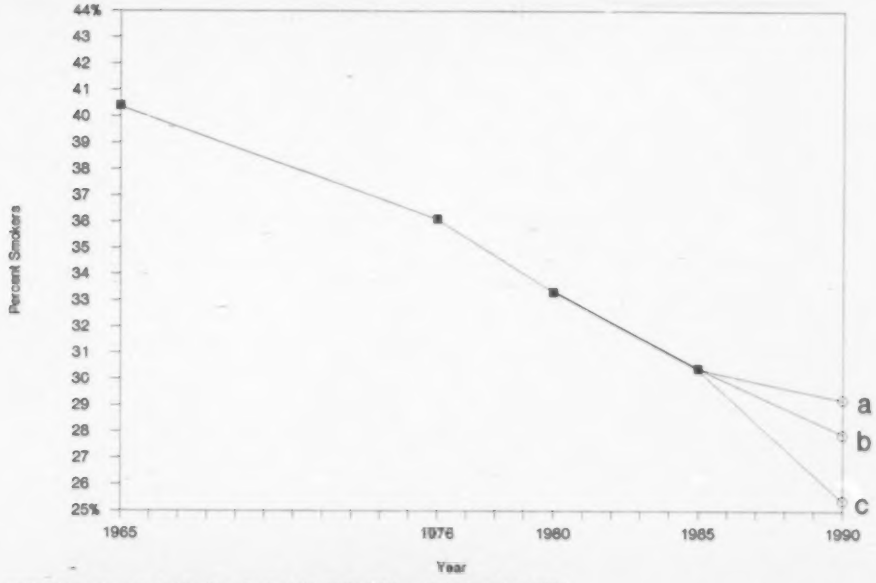

FIGURE 2—Trends in Smoking in the US, 1965–1985 and Projected to 1990
Data for 1965, 1976, 1980, and 1985 are from the National Health Interview Surveys for adults 20 years of age or greater, and are age-adjusted to the 1985 U.S. population. Rates for 1990 are projected assuming an annual rate of decline of 0.25%/year (a), 0.5%/year (b), and 1.0%/year (c), respectively.

in each state from 1985–1990. In fact, in contrast to the conclusion of the Midcourse Review, our analysis suggests that it is unlikely the prevalence of smoking will be less than 25 percent in the United States overall by 1990.

When setting objectives for cigarette smoking prevalence for 1990 and beyond, it is important to consider the methods that will be used to monitor progress toward achieving that objective. For example, although the surveys used in this report were both in-person surveys, slightly different methods were used to assess the prevalence of smoking. In the

TABLE 2—Trends in the Prevalence of Smoking for Adults 20 years of age or greater, United States, 1965–1985

Year	Percent Smokers	Percent Decline/Year from 1965	from 1976	from 1980
1965	40.4	—	—	—
1976	36.1	.39	—	—
1980	33.3	.47	.70	—
1985	30.4	.50	.63	.58

Data are from the National Health Interview Surveys for adults 20 years of age or greater, and are age-adjusted to the 1985 US Census[5]

National Health Interview Survey, respondents were directly questioned about their smoking habits, whereas in the Current Population Surveys, proxy responses were commonly used to determine their smoking status (59 percent of the responses among men were provided by proxy).

When possible, it is important to use state-specific information on trends in smoking when setting objectives. Many states now use data from the Behavioral Risk Factor Surveillance System to estimate the prevalence of smoking.[6] However, telephone surveys generally provide smoking estimates lower than those obtained from in-person surveys. The prevalence estimates from the 22 states that conducted random digit-dialed (self-respondent only) telephone surveys as part of the Behavioral Risk Factor Surveillance System in 1985 were, on the average, 2 percent to 3 percent less than the in-person estimates provided in this report.[3] These lower estimates likely result from telephone noncoverage and lower response rates among smokers in telephone surveys.[7] Nevertheless, this surveillance system has proven useful to states and should be used in the future to set objectives and monitor trends in smoking if these biases are accounted for.

Although predicting smoking prevalences in any state five years hence is difficult, this paper provides a range of estimates that states may consider when setting their own objectives. Because many programs to reduce the prevalence of cigarette smoking are implemented at the state level, it is important for each state to set reasonable objectives for the future.[8] One of the purposes in setting forth an objective is to promote new initiatives. Therefore, selecting a target that will be reached without intervention is inappropriate. Similarly, setting objectives that are clearly not achievable is equally inappropriate. To this end, we hope that this report encourages setting objectives based on a careful review of available data and an assessment of the likely impact of future intervention efforts.

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The News on Smoking: Newspaper Coverage of Smoking and Health in Australia, 1987–88

SIMON CHAPMAN, PHD

Abstract: All newspaper articles (n = 1601) mentioning tobacco or smoking drawn from a 12-month census of 30 Australian capital city newspapers in 1987–88 were coded for content, "slant," and apparent origin. Overall, 62 percent of articles were positive in their orientation toward smoking control objectives, 17 percent were negative, and 21 percent were neutral. Newspapers owned by Rupert Murdoch published 55.7 percent positive and 23.7 percent negative articles compared to 68.0 percent positive and 13.7 percent negative articles in the other large chain of Australian newspapers. (*Am J Public Health* 1989; 79:1419–1421.)

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Introduction

Male smoking prevalence has declined in Australia from an estimated 72 percent in 1945[1] to a 1986 level of 31.9 percent.[2] Female smoking prevalence has risen slightly in the same period from 26 percent[1] to 28.8 percent.[2] Almost all of the commentary on this decline in male smoking rates has been preoccupied with the impact of government-sponsored and health agency-sponsored mass health information and persuasion campaigns,[3] and the introduction of smoking control legislation and regulation.[4–7]

Since 1983, most States in Australia have conducted mass-media-based public awareness campaigns although the largest of these efforts has had a budget considerably less

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than the typical cigarette advertising campaign.

Preoccupation with the impact of defined public sector interventions—such as purchased media time and space, school programs, and smoking cessation efforts—has tended to obscure the impact of a principal means by which the public is exposed to messages about smoking: the news media. This study reports a content analysis of a census of all coverage of smoking issues in capital city newspapers in Australia in a sample period in 1987–88.

Methods

All articles dealing exclusively with or mentioning tobacco or smoking published in all 30 Australian capital city newspapers were obtained from a commercial press clipping service for the 12 months May 1987–April 1988. The newspapers included "quality" and popularist newspapers and Sunday editions. A total of 1,601 clippings were thus obtained.

Coding

Each clipping was coded for the publication in which it appeared; date of publication; type of item (news item, feature article, news brief, letter, editorial); apparent origin; principal content; and its "slant" or orientation. These classifications are described in the Appendix. To assess whether the coder's ratings for slant were idiosyncratic, a randomly selected subsample of 1 percent of the clippings (16 articles), was coded by nine peope working in four different smoking control agencies around Australia; a Cohen's Kappa of 0.84 was obtained, indicating high inter-coder reliability (external validity).[8]

Table 1 shows the principal content of all articles, cross-tabulated by slant. News and commentary on the December 1987 ban on smoking on all domestic airlines (187 items), and on the introduction of the Victorian Tobacco Control Act (106 items) which banned all outdoor and cinema tobacco advertising in that State and raised State tax on tobacco, were widely covered. Articles on passive smoking appeared at 125 percent greater frequency than the next most common principal category (advertising and promotion).

When articles were classified by type of article and slant (Table 2), there was a similar distribution for all main categories with the predictable exception of the fewer neutral letters.

Newspaper ownership in Australia is dominated by two main groups, News Corporation owned by Mr Rupert Murdoch, and John Fairfax and Sons. Positive articles in Murdoch-owned papers (55.7 percent) were less common, compared to Fairfax-owned (68 percent) and other-owned (62.2 percent); negative articles were more common in Murdoch papers (23.7 percent), compared to 13.7 percent in Fairfax-owned and 17.2 percent in other-owned.*

When articles were classified by apparent origin and slant (Table 3), the expected polarizations toward positive articles generated or written by health workers, agencies, and physicians, and negative articles inspired or written by the tobacco and advertising industries were found.

Discussion

Because this study examined only one year of publications in one country, no longitudinal or international comparisons can be made. Comparisons of prevalence with articles on drugs or alcohol would require explicit but debatable hypotheses about some ideal level of equivalence or difference and tone of reporting about various topics. In the absence of any reasonable or accepted standards of what ought to be the frequency of tobacco reporting, little can be concluded, other than to suggest that any notion that Australian newspapers are loathe to print items that may offend their tobacco advertisers carries little support.

Evidence from the USA,[9–12] and Australia[13] has demonstrated that there is an inverse relation between the acceptance of tobacco advertising by magazines and the appearance of articles on tobacco. Because all of the newspapers in this study accepted tobacco advertisements, this is not the case with this sample of newspapers in Australia. While Australian newspapers in the past have leaned more toward a negative coverage of proposed bans on tobacco advertising,[14] the evidence here shows no support for the "gag of the tobacco dollar" view. Despite the Murdoch-owned newspapers printing more negative articles than those

TABLE 1—Principal Content of Tobacco-Related Articles by Slant

Slant of Article	Total	Positive (%)	Negative (%)	Neutral (%)
Passive smoking	534	319 (59.4)	117 (21.4)	98 (18.2)
Advertising/promotion	237	136 (53.3)	46 (18.0)	55 (21.6)
Cessation	114	97 (85.1)	5 (4.4)	12 (10.5)
Health issues	215	175 (81.4)	19 (8.8)	19 (8.8)
Taxation	212	88 (41.5)	54 (25.5)	71 (33.5)
Miscellaneous	289	182 (63.4)	34 (11.2)	73 (25.4)

Highest ranking issues: Smoking on aircraft (187), Victorian legislation commentary (106), workplace smoking bans (78), sporting sponsorship by tobacco (75), overviews on smoking and health (66), smoking on public transport (63), calls to ban tobacco advertising (58), how to stop smoking (54). Full breakdown available from author.

TABLE 2—Slant of Tobacco Articles by Type of Publication

Type	Positive (%)	Negative (%)	Neutral or N/A (%)
Article	280 (62.9)	51 (11.5)	114 (25.6)
Feature	70 (61.4)	12 (10.5)	32 (28.1)
News brief	252 (59.9)	56 (13.3)	114 (26.8)
Letter	302 (64.4)	137 (29.2)	30 (6.4)
Editorial	6 (50.0)	4 (33.3)	2 (16.7)
Contributor Column	1 (20.0)	2 (40.0)	2 (40.0)
Cartoon	13 (38.2)	9 (26.4)	12 (35.3)
Advice column	8 (100)	—	—
Part of wider article	65 (69.9)	5 (5.4)	22 (24.7)
Totals	997 (62.3)	276 (17.2)	328 (20.5)

$\chi^2 = 119.91$ ($p < 0.001$ 2df)

*A full breakdown for all 30 newspapers is available from the author.

TABLE 3—Origin of Article by Slant of Article

Origin	Number	Positive (%)	Negative (%)	Neutral or N/A (%)
No obvious source	553	267 (48.3)	70 (12.7)	196 (35.4)
Doctor/health worker/agency	291	258 (88.7)	13 (4.5)	20 (6.9)
Tobacco or advertising industry	65	—	52 (80.0)	13 (20.0)
Citizen	404	255 (63.1)	119 (29.5)	30 (7.4)
Politician	187	131 (70.1)	8 (4.3)	48 (25.7)
Wire service	121	86 (71.1)	14 (11.6)	21 (17.4)
Total	1601	997 (62.3)	276 (17.2)	328 (20.5)

owned by other publishers, Murdoch newspapers still published 2.4 times as many positive articles as negative.

Explanations of the difference between the apparent editorial or owners' policies with US magazines and Australian newspapers require further study, but may lie in two directions: First, articles in magazines are often planned well in advance and therefore may be open to considerably more explicit or latent censorship than those submitted by journalists in the more immediate world of newspaper writing. However, this view would appear to hold little credence in this study: 114 feature-length articles on tobacco were coded, of which 61 percent were positive and only 11 percent negative, the lowest negative proportion for any major category of article. Like magazine journalism, feature article writing in newspapers is often directed from editorial level down to journalists, so the evidence suggests a newspaper editorial climate conducive to the production of "good news" on smoking in Australia.

The second explanation is that tobacco advertising is more economically important to magazines than to newspapers and that magazines would be thus less inclined to "bite the hand that feeds them." *The Age* newspaper, which ran many more articles on tobacco than any other in the sample, receives about $1.00 from cigarette advertising for every $200 from all advertising,[15] compared to about $1 in $60 for newspapers generally.[16] Australian magazines, by contrast, receive about twice the total proportion of advertising dollars from tobacco as do newspapers.[16] This explanation is more consistent with the greater and more positive coverage of smoking issues in newspapers compared to that in magazines.

APPENDIX A

Coding Classification

Principal content was first coded liberally, and then subsumed into six principal classifications (passive smoking, advertising and promotion, smoking cessation, health issues, taxation, and miscellaneous). "Origin" refers to the apparent origin of the item. News items appear after a process of selection by journalists and editors about what is newsworthy. This selection occurs from sources which frequently include groups with explicit interests in having certain views of issues appear in print. Both the promotion and control of smoking are issues which have vested interest groups keen on having the media publicize news and commentary favorable to their respective interests.[17] A 1973 study of nearly 3,000 items published in the *New York Times* and the *Washington Post* found that 58.2 percent of stories came from "routine" channels (press releases, semi-official documents etc).[18] The concept of "pronunciamento"[17]—or the direct transfer of information, ideas or accusations from interest groups to readers via virtually unaltered transcripts of their press releases—appeared to be applicable to many items in the sample.

"Slant," coded either positive, negative, or neutral/not applicable, refers to the extent to which any article could reasonably be said to be supportive of a social climate antithetical to smoking. Such judgment begs the question of who is doing the judging.[19] I approached this by considering how a person concerned to advance smoking control objectives would judge any given article. Would a smoking control advocate be pleased or displeased to see a particular article appearing in print? Articles judged as being likely to be read by a person favorably disposed toward smoking control, as enhancing the general view that smoking and those institutions, interests, laws and policies that support it are undesirable were coded "positive." Articles that would be understood by such a person as conveying a view of smoking as in some way positive, relatively unimportant compared to other issues, or which were critical of laws, policies and individuals advocating restricting or opposing smoking were coded "negative". Articles which were judged as being neither clearly positive or negative, which evidenced a clear attempt at balancing health and tobacco industry views, or which were simple announcements about (for example) a forthcoming rise in tobacco excise, were coded "neutral/not applicable."

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Influenza Immunization in the Elderly: Knowledge and Attitudes Do Not Explain Physician Behavior

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Abstract: The records of 847 elderly clinic patients were reviewed and the 95 primary care physicians who managed their care were surveyed to determine factors related to their ordering of influenza immunization. Overall, 41 percent of the eligible patients were offered vaccine (range 0–90 percent). Physician offering of vaccine was unrelated to patient age, sex, or race but was higher in patients seen on multiple visits (47 vs 39 percent) and in those vaccinated during the prior year (61 vs 35 percent). Physician offering of vaccine was not associated with perceived incidence of severe side effects or estimates of vaccine efficacy. (*Am J Public Health* 1989; 79:1422–1424.)

Introduction

The elderly have been shown to be at high risk of death and serious illness following influenza infection.[1,2] The Centers for Disease Control[3] and the American College of Physicians[4] have recommended that routine annual influenza immunization be offered to all patients age 65 and older. Such immunization has been shown to be a cost-effective preventive health practice.[5–7] However, less than 20 percent of those over age 65 or otherwise considered to be at high risk receive influenza vaccination each year.[8]

Previous studies have evaluated factors influencing patients' decisions to receive or refuse influenza vaccine,[9–12] but there is little information about what affects physicians decisions to offer vaccination. Physicians appear to know the indications for use of the vaccination, but fail to translate this knowledge into clinical practice.[13]

We studied the frequency and determinants of influenza immunization to see whether differences in physician attitudes, knowledge, or perceived side effects of the vaccine could account for the variation in immunization practices.

Methods

All patients age 65 and older seen in the Primary Care Clinic at the Milwaukee County Medical Complex from October through December of 1984 were identified and their medical records reviewed to determine if they were offered influenza vaccination. Patients were considered eligible if they had not already been vaccinated that season, had no history of egg anaphylaxis or severe reaction to prior vaccination, and did not have an acute febrile illness. Immunization was considered to have been offered by the physician if an order was written or if the patient's refusal of immunization was noted. Demographic data, history of influenza vaccination the prior year, and the type of patient visit (continuing care or visit to a different physician) were also recorded.

Following the study period, a telephone survey was conducted by a non-medical interviewer of all 95 physicians (90 residents and five faculty) who provided direct patient care. Rates of offering influenza vaccine were calculated for the 82 physicians (77 residents and five faculty) who saw at least five eligible patients during the study period. Categorical data were analyzed using the chi square statistic and correlation coefficients (Pearson) were calculated for continuous variables.

Results

The charts of 847 (99.2 percent) of the 854 patients age 65 years and older seen during the study period were reviewed; 812 were eligible for influenza immunization. Overall, 332 (41 percent) of those eligible were offered vaccine; however, individual physicians varied widely in their rates of offering vaccine to their patients, with a range of 0–90 percent. Vaccination was refused by 9 percent of those to whom it was offered. Patient demographics, clinic visit characteristics, and their relation to being offered vaccine are shown in Table 1. Vaccination during the prior calendar year was the strongest determinant of being offered vaccine.

The telephone-administered questionnaire was completed by 88/90 residents and four of five faculty (97 percent overall). Over 80 percent knew the recommended time of year to vaccinate, that influenza vaccine can be given simultaneously with pneumococcal vaccine, and that the objective of influenza immunization is to reduce morbidity and mortality. More than 75 percent correctly responded that influenza immunization is contraindicated in the presence of an acute febrile illness or history of egg anaphylaxis. However, 40 percent incorrectly thought that use of systemic steroids contraindicated vaccination.

Many physicians overestimated the occurrence of rare or unrecognized side effects. Anaphylaxis was estimated to occur >1 percent of the time by 25 percent of physicians, and Guillain-Barré syndrome was believed to occur in >1 percent of recipients by 15 percent of physicians. No association was found between vaccine offering rates and knowledge of incidence rates for anaphylaxis or Guillain-Barré syndrome.* Most physicians (69 percent) considered influenza vaccine to be 70–90 percent effective in preventing infection, but estimates of high vaccine efficacy were not correlated with increased offering rates.

Table 2 shows physician attitudes towards immunizing individuals with various high-risk conditions. The category "age 65 or over" received fewer highest strength recommendations than categories of chronic obstructive pulmonary disease, asthma, residency in a nursing home, renal insufficiency and diabetes. The strength of conviction to immunize

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*Data available on request to author.

TABLE 1—Patient and Clinic Visit Characteristics: Relation to Vaccine Use

Category	N	N (%) Offered Vacine	Odds Ratio (95% CI)	Adjusted Odds Ratio* (95% CI)
Sex				
Female	569	242 (43)	1.26	1.14
Male	243	90 (37)	(.91, 1.73)	(.97, 1.34)
Race				
Black	416	181 (44)	1.25†	1.09
White	376	145 (39)	(.93, 1.67)	(.94, 1.33)
Other	20	6 (30)		
Age (years)				
65–74	465	196 (42)	1.13	1.06
≥75	347	136 (39)	(.84, 1.52)	(.93, 1.22)
Number of visits				
1	601	233 (39)	.72	.85
2 or more	211	99 (47)	(.52, .995)	(.72, 1.01)
Provider seen				
Usual	639	272 (43)	1.4	1.12
New	173	60 (35)	(.97, 2.0)	(.93, 1.35)
Vaccinated in prior year				
Yes	191	117 (61)	2.99	1.73
No/unknown	621	215 (35)	(2.1, 4.2)	(1.49, 2.04)

*Adjusted by multivariate logistic regression
†Black vs all others

TABLE 2—Percentage Distribution of Strength of Physicians' Recommendations to Vaccinate by Patient Risk Factors

Risk Factor	Percent of Physicians Indicating Category of Recommendation*				
	5	4	3	2	1
Chronic Obstructive Pulmonary Disease	84	10	5	1	1
Asthma	77	11	9	2	1
Nursing Home Resident	73	15	8	1	3
Renal Insufficiency	59	16	17	7	1
Diabetes	55	24	16	1	3
Age >65 years	36	26	34	3	1
Heart Failure	29	15	35	10	11
Anemia	8	9	54	12	17

*5 = very strongly, 1 = not recommended

elderly patients was only weakly correlated with offering rates ($r = .297$, $p = .004$).

Discussion

Physicians may not use influenza vaccine because of uncertainty about its indications, doubts about its efficacy, or failure to consider vaccination during patient encounters.[14] The recent report by Setia, *et al*,[13] based on 14 physicians, found that only 33 percent of 540 nursing home patients were immunized despite their physicians' strong beliefs in the efficacy and low risk of vaccination. Our study confirms that physicians' perceptions of vaccine efficacy and their strength of conviction to offer vaccination were high but still were not strongly associated with higher rates of vaccination.

While not correlated with the use of vaccine, the perceived incidence of severe vaccine side effects was markedly higher than the actual incidence reported in the literature. Anaphylaxis is an extremely rare occurrence after influenza vaccination,[15] and Guillain-Barré syndrome has not been associated with vaccination since the swine flu vaccine of 1976.[16–18] Educational efforts should emphasize the low risk of major vaccine side effects.

The overwhelming majority of physicians understood key recommendations concerning vaccine use and accurately assessed its efficacy. Other investigators[19,20] have noted that knowledge of the efficacy and recommended use of influenza vaccine does not guarantee its appropriate administration. Moreover, Cohen[20] found that educational seminars for physicians were less important than patient chart-based checklist reminders in changing vaccination behavior.

We recognize several limitations of our findings. This study was conducted in an academic hospital-based outpatient clinic and the results may not be generalizable to the community setting. Certain patients could have declined receipt of the vaccine following discussions with their physicians or have informed the physician that they received the vaccine elsewhere without documentation in the chart. However, given the relatively high offering rate and the fact that such failures would underrepresent the intent to immunize, it is unlikely that this occurred frequently. As this clinic is the sole source of care for most of this indigent population, it is unlikely that many received the vaccine from alternate sites in the community.

Attempts to increase vaccine use should focus on measures other than changing physician attitudes or increasing their knowledge about the vaccine. Strategies which do not rely on individual physician decision-making should be developed to increase appropriate vaccination of the elderly.

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RWJ Foundation Grants Target Children with Mental Illness

Eleven states and the District of Columbia were selected this summer to launch a national initiative to improve services for US children with serious mental illness. The grants were awarded by the Robert Wood Johnson Foundation under its $20.4 million Mental Health Services Program for Youth.

Selected to receive one-year planning grants of $100,000 under the initial phase of the program are: California, the District of Columbia, Kentucky, Michigan, North Carolina, North Dakota, Ohio, Oregon, Pennsylvania, Vermont, Washington, and Wisconsin. Based on the strength of their plans to better coordinate and finance home- and community-based services for mentally ill children, up to eight of the projects will be awarded four-year grants of up to $2.4 million to carry out their plans. The initiative is intended to improve care for the estimated three million US children whose mental disorders—ranging from autism to depression—typically result in long-term disability or institutionalization, foundation officials said.

Directed for the foundation by Mary Jane England, MD, vice president of medical services at the Prudential Insurance Company in Roseland, NJ, the five-year program will allow states to focus their efforts on communities or geographic areas ranging in population from 300,000 to 600,000. The grants require collaboration among the many state and local agencies responsible for planning, providing and financing mental health care for children.

Since the Foundation was established as a national philanthropy in 1972, it has awarded more than $996 million in grants to improve the health of adolescents, children, the elderly, the homeless, the mentally ill, people with AIDS and others. For further information, contact the: Robert Wood Johnson Foundation, College Road, PO Box 2316, Princeton, NJ 08543-2316. Tel: (609) 452-8701.

Occupational Exposure to Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) among Embalmers: A Pilot Seroprevalence Study

SALLY BUNCE TURNER, MS, LAUREEN M. KUNCHES, MPH, KATHLEEN F. GORDON, MS, PATRICIA HYLAND TRAVERS, MS, AND NANCY E. MUELLER, ScD

Abstract: We performed a serosurvey of 133 embalmers in an urban area where human immunodeficiency virus (HIV) infection is prevalent. Although we found histories of needlesticks to be common, and the seropositivity rate of hepatitis B virus (HBV) (13%) was approximately twice that of a blood donor comparison group, HIV antibody was uniformly absent in 129 embalmers who denied HIV risk factors, and present in one of four with self-described risk behaviors. The risk of HBV infection was higher among embalmers who have worked more than 10 years, relative risk (RR) 16.2 (95% confidence interval 2.1, 126.5), did not routinely wear gloves, RR 9.8 (CI 3.4, 28.5), or are employed in the city of Boston, RR 4.7 (CI 1.8, 12.0). (*Am J Public Health* 1989; 79:1425–1426.)

Introduction

There is widespread concern in the health care community about occupational exposure to human immunodeficiency virus (HIV). The occupational groups at risk of exposure are those who are also at risk of hepatitis B virus (HBV) infection. Although a substantial number of health care workers have been examined for the risk of both infections, limited information is available on the risk of HBV infection[1,2] and none is available on the risk of HIV infection to embalmers.

Previous studies have shown that HBV has greater potential for nosocomial spread than HIV.[3–7] The purpose of this pilot study is to evaluate the risk of transmission of both infections in relation to work practices in a group of embalmers working in eastern Massachusetts.

Methods

The target population was composed of embalmers from the greater Boston and Worcester, Massachusetts, metropolitan areas. Embalmers were eligible for enrollment if, in their career, they had embalmed 20 or more bodies. The data were collected over a seven-month period, from August 1986 to February 1987.

An oral questionnaire elicited demographic information regarding age, sex, ethnicity, marital status, past medical history, occupational history, and the risk factors for HBV and HIV infection: history of renal dialysis, hepatitis, cancer, bleeding disorder, blood transfusion, receipt of globulin preparation or hepatitis B vaccine, tattoos, and intravenous drug use.

All serum samples were tested for hepatitis B surface antigen (HBsAg), hepatitis B surface antibody (HBsAb), and hepatitis B core antibody (HBcAb) by enzyme-linked immunosorbent assay (ELISA) (Abbott Laboratories, North Chicago, Illinois). Antibody to HIV was measured with the Abbott HTLV-III/LAV ELISA (Abbott Laboratories, North Chicago, Illinois). A repeatedly reactive sample was confirmed by the Western blot technique. Sera that tested positive for HBcAb were screened for antibody to the delta antigen (Abbott Anti-Delta).

Comparison of the seroprevalence among subgroups of the population are expressed as relative risks (RR), estimated by the prevalence odds ratios, with their 95 percent confidence intervals.[8] Variables shown to be risk factors for HBV were included in a logistic regression procedure (Statistical Analysis System Inc., Cary, NC) to evaluate potential confounding.

Results

Of 108 study subjects who had no history of hepatitis B vaccination, 14 (13 percent) had serologic evidence of HBV infection. Of these, HBsAg (with HBcAb) was detected in one, HBcAb alone in one, and HBsAb in 12. Of the 12 with HBsAb in serum, 11 had HBcAb as well. Therefore, the frequency of HBcAb in all participants, including those with any marker, was 9.7 percent (13 of 133). None of the study subjects with serologic markers for HBV infection had evidence of coinfection with the delta virus.

Twenty-five embalmers reported being vaccinated with hepatitis B vaccine. None of the vaccinees had evidence of infection with HBV (HBsAg or HBcAb) and will therefore be considered "seronegative" for purposes of the following analysis.

Years of employment, age, and number of bodies embalmed were significantly associated with HBV infection (Table 1). When these interrelated variables were entered into a logistic regression procedure, employment for more than 10 years correlated most strongly with prior infection (adjusted odds ratio = 85.9); no additional predictive value was added to the regression model by age or number of bodies embalmed.

Embalmers who do not routinely wear gloves were almost 10 times more likely to have serologic markers of HBV infection than those who do. Controlling for duration of employment strengthened this association (adjusted odds ratio = 16.7). No correlation was found between HBV markers and reported use of gowns or aprons, shoe coverings, goggles, glasses, or face masks.

Subjects employed in Boston were more likely to have evidence of HBV infection than those employed outside of Boston. After adjusting for years of employment and use of gloves, Boston's embalmers were 12 times more likely to have HBV markers than others. All subjects with evidence of previous infection with HBV reported sustaining one or more accidental needlesticks during their career. However, the

From the Division of Community Health Services and the Department of Nursing, Boston Department of Health and Hospitals (Turner); the Department of Epidemiology (Mueller) and the Occupational Health Program (Travers) at Harvard School of Public Health; the Department of Public Health, Massachusetts Center for Disease Control (Kunches); Boston University School of Nursing (Travers); and the Department of Nursing, New England Deaconess Hospital (Gordon). This paper, submitted to the Journal April 4, 1988, was revised and accepted for publication April 7, 1989.

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TABLE 1—Risk Factors for HBV Infection

Variables	Total	Serologic Evidence of HBV Infection	Relative Risk (95% CI)
Employed > 10 years as embalmer	66	13 (20)	16.2 (2.1, 126.5)
Embalmed ≥ 1000 bodies	67	12 (18)	6.9 (1.5, 32.8)
Do not always wear gloves	18	7 (39)	9.8 (3.4, 28.5)
Employment in Boston	37	9 (24)	4.7 (1.8, 12.0)
Greater than 35 years of age	67	11 (16)	3.6 (1.2, 11.2)
Eat or drink while embalming	48	7 (15)	1.8 (0.66, 4.7)
Smoke while embalming	43	6 (14)	1.6 (0.50, 4.3)

*Figures in parentheses denote percentages.

number of known HBV cases embalmed or recognized HBV needlesticks was not related to HBV infection. Three of the four subjects who disclosed non-occupational risk factors had evidence of previous HBV infection.

When information on history of previous employment in other occupations involving blood exposure was analyzed, there was no observed association with prevalence of HBV markers. Twenty-one percent of embalmers with HBV markers reported having clinically overt hepatitis in the past.

All 133 subjects were tested for antibodies to HIV. One of four self-described members of recognized high-risk groups tested positive for HIVAb; all other subjects were seronegative.

Discussion

During the embalming procedure, embalmers often come into contact with large amounts of blood as the vascular system is drained. Depending on the cause of death and whether an autopsy has been performed, they may be required to handle various body parts and tissues, to make numerous incisions and subsequently suture the incised tissue. However, compared to other health care professions, embalmers have been largely ignored by researchers evaluating the risk of occupationally transmitted infections. The results of this study indicate that embalmers are at increased risk of infection with HBV when compared to the general population,[9] but have not shown increased risk of HIV infection. Overall, the seroprevalence of hepatitis B among health care workers ranges from 5 to 32 percent.[9–15] The 13 percent prevalence of hepatitis B markers among the non-vaccinated subjects indicates that embalmers are a moderately high-risk group of health care professionals. The finding that 79 percent of these individuals had simultaneous HBcAb and HBsAb is consistent with the results of Dienstag and Ryan,[9] who reported that frequent blood exposure, and in general more intense exposure, was correlated with the prevalence of both antibodies.

Recently concern over exposure to asymptomatic HIV carriers has motivated improved infection control practices among health care workers. Embalmers currently maintain these standards for known AIDS (acquired immunodeficiency syndrome) cases, but need additional education to emphasize adherence to universal precautions. The Centers for Disease Control recommends the routine use of gloves, masks, protective eyewear, gowns, and waterproof aprons for all persons performing or assisting in postmortem procedures.[16]

The study participants recalled having embalmed a total of 298 AIDS cases, compared to 285 confirmed AIDS deaths in Massachusetts when the study was initiated. This comparison suggests that the estimates of occupational exposure to AIDS cases are accurate.

Little emphasis has been placed by this occupational group on the risk of exposure to HBV in the workplace. Because this study demonstrates that the risk of occupationally acquired HIV infection is extremely low, and the risk of occupationally acquired HBV infection is substantial, emphasis should be placed on prevention of transmission of HBV during embalming by the use of universal blood and body fluid precautions,[16] and routine immunization with hepatitis B vaccine.

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Health Care Cost Containment Measures and Mortality in Hennepin County's Medicaid Elderly and All Elderly

GREGORY L. LINDBERG, MA, NICOLE LURIE, MD, MSPH, SUSAN BANNICK-MOHRLAND, MD, ROBERT E. SHERMAN, PHD, AND PAUL A. FARSETH, BA

Abstract: In Minnesota, several health care cost containment measures occurred about the time Medicare's Prospective Payment System (PPS) was implemented. These included a moratorium on additional nursing home beds, preadmission screening of nursing home applicants, and rapid growth in HMO (health maintenance organization) enrollment by Medicare recipients. Hospital days per elderly Medicaid recipient decreased by 38 percent for those in nursing homes and by 35 percent for those not in nursing homes from 1982 to 1984. By 1986, hospital days per recipient had decreased 53 and 55 percent, respectively, from the 1982 level.

Age-adjusted mortality rates for elderly Medicaid nursing home residents for the period 1977 through 1986 showed an increasing trend after 1982. Estimated age-adjusted mortality rates for the entire County population, which had decreased steadily from 1970 to 1982, rose significantly above the projected rate in 1984, 1985, 1986, and 1987.

We conclude that, coincident with the institution of the PPS and other health care cost containment measures, use of hospital care has fallen for all elderly Medicaid recipients, age-adjusted mortality rates among those in nursing homes have increased, and the mortality rate trend for the total Hennepin County elderly population has stopped declining. (*Am J Public Health* 1989; 79:1481–1485.)

Introduction

Medicare's transition to a system of "fixed price" hospital reimbursement based on diagnosis-related groups, the prospective payment system (PPS) has triggered concerns about the consequences, particularly the chance that the poor and the chronically ill may be at increased risk of underservice.[1] At some point there is trade-off between cost and quality of care. Two manifestations of this trade-off might be an increase in age-adjusted mortality rates for Medicare patients and signs of premature hospital discharge to nursing homes.

In a cross-sectional area study of Medicare spending levels, Hadley, found that greater Medicare spending per beneficiary is associated with significantly lower mortality rates.[2] He also reported that national mortality rates for those age 65 and older have either changed little or actually increased since 1982, after declining throughout the 1970s. In Wisconsin, Sager, *et al*, reported a shift in the site of death from the hospital to the nursing home and an increase in the age-specific death rate among elderly nursing home residents from 1982 to 1985.[3] Comparing 12-month periods immediately before and after the institution of the PPS, they found a 72 percent increase in the rate of hospitalization and a 26 percent decline in hospital length of stay among elderly Medicaid nursing home patients. In a later study, Sager, *et al*, found that shifts in the location of death from hospitals to nursing homes were most pronounced in states with large declines in hospital length of stay and high population enrollments in health maintenance organizations (HMOs).[4] In a survey of nursing homes in Portland, Oregon, Lyles[5] found a 26 percent increase in the age-adjusted mortality rate among nursing home residents from 1983 to 1984. Lewis, *et al*,[6] reported that Medicare patients discharged for the first time to skilled nursing facilities in 1984 were more likely to be bed-confined, comatose, or have indwelling catheters than those discharged in 1980; however, these trends began before the institution of the PPS.

Meyer-Oakes, *et al*,[7] found a significant reduction in length of hospital stay but no change in hospital mortality post-PPS. However, they did not examine the possibility of increased post-discharge mortality. Shortell and Hughes, using 1983–84 data from 981 hospitals in 45 states found that the ratio of actual to predicted death rates for Medicare recipients with one of 16 selected diagnoses was 6 to 10 percent higher in those states with the most stringent rate reviews and 5 to 6 percent higher in those with the strictest procedures for reviewing applications for certificate of need.[8]

Fitzgerald, *et al*,[9] reported an increase in short- and long-term admissions to nursing homes after PPS for hip fracture patients in Indianapolis. Linn, *et al*,[10] have reported an increase in complication rates in patients undergoing head and neck cancer surgery after implementation of PPS.

The PPS was implemented in Hennepin County, Minnesota (the greater Minneapolis area) over a period of several months beginning in October 1983. Roughly coinciding with the implementation was the imposition of a statewide moratorium on new nursing home beds. State and county efforts to divert nursing home applicants through preadmission screening and alternative care grants were phased in beginning in 1982 and were fully implemented by 1986. The number of nursing home beds in Hennepin County has remained relatively stable over the past 10 years at around 12,000 and there has been a major increase in recent years in the provision of home health care services. For example, expenditures in Hennepin County under the state and county funded Alternative Care Grant Program increased from $200,000 in 1983 to $5 million in 1988. This represents only a small portion of the total public and private expenditures for these services. In addition, since the end of 1984, Medicare enrollees have been shifting from fee-for-service to prepaid, capitated care. As of December 31, 1986, 47 percent of all Twin Cities Metropolitan Area residents age 65 or older were enrolled in HMOs (Minnesota Department of Health, personal communication).

The present study describes changes in hospital use and

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age-adjusted mortality rates that have occurred in Hennepin County following the implementation of these policy initiatives. Examined are: hospital utilization by elderly (65 and older) Medicaid recipients; the age-adjusted mortality rate for the elderly Medicaid caseload; and the age-adjusted mortality rate trend for the total elderly population in Hennepin County. The Medicaid caseload was selected for analysis because it represents a vulnerable population that is of special concern to state and local government and because health care utilization data were readily available through the Minnesota Department of Human Services. Members of this caseload differ from the general elderly population in that they are more likely to be from low income groups, may be sicker, and are more likely to have used extensive medical care or nursing home services.

Methods

Hospital Utilization Data

Data on hospital utilization by institutionalized elderly Hennepin County Medicaid recipients were obtained from the Minnesota Medicaid Information System. The Minnesota Department of Human Services provided research files containing annual client eligibility summaries and hospital payment records for the years 1980 through 1986. These included all Medicare claims in which Medicaid was a financial participant. The years, 1980 through 1982, represent a period unaffected by the PPS or local cost containment policies.

Recipients were categorized as institutionalized or non-institutionalized based on their living arrangement codes. The living arrangement code indicated the recipient's status at the last eligibility review or living arrangement change before the end of each year. Those in nursing homes or state hospitals were considered institutionalized while others were considered noninstitutionalized. During the years examined, the number of elderly institutionalized Medicaid recipients ranged from 6,800 to 7,348, and the number of elderly noninstitutionalized recipients ranged from 3,630 to 3,907. Hospital utilization and mortality rates were calculated for the full population of elderly recipients each year. There have been no major changes in Medicaid eligibility criteria or procedures from year to year that would have significantly altered the character of the caseload.

An additional analysis examined changes in hospital utilization during the last six months of life among elderly Medicaid recipients who died. Recipients selected for this analysis were those whose cases closed because of death during the last half of the year, and who had at least six continuous months of eligibility preceding their death.

The tabled hospital utilization data are presented with estimated 95 percent confidence intervals. The percentage differences in utilization between years are also presented in the text with 95 percent confidence intervals.

Mortality Data

Mortality among the Medicaid caseload was determined from annual Medicaid client eligibility files based on case closings in which death was coded as the reason for closing. To identify mortality trends over the longer term, these files were examined for 1977, the earliest year available, through 1986. Age-specific mortality rates for institutionalized and noninstitutionalized elderly Medicaid recipients were computed by five-year age intervals for each year. Age-adjusted mortality rates were then computed for these groups using, as a standard population, the mean of the age distributions for the first and last years of the time series (1977 and 1986).[11]

Age-adjusted mortality rates for the entire elderly population of Hennepin County (both Medicaid and non-Medicaid) for the period 1970–87 were derived using the death certificate data maintained by the Minnesota Department of Health and age-specific population projections developed for Hennepin County.[12] Death certificate data in Hennepin County are considered accurate and complete, and the population projections for the elderly population utilized a cohort-component procedure which is thought to result in estimation accurate to one percent or so over the study time frame. (Population estimates for the 1970–80 period were developed through linear interpolation of census data.)

Linear regression on logarithmicly transformed data was used to estimate the age-adjusted mortality rate trend during the period preceding the implementation of cost containment measures (i.e., prior to 1983). Subsequent age-adjusted mortality observations were examined to see if they fell within the 95 percent confidence interval (for observations) of the pre-cost containment trend.

Results

Changes in Hospital Utilization

There was a marked reduction in hospital days per Medicaid recipient beginning in 1983. This is due not only to a substantial reduction in average hospital length of stay, but also a large decrease in hospitalization rates (Table 1). From 1982 to 1984 (the years before and after implementation of the PPS) hospital days per Medicaid recipient per year fell 38 percent (95% CI = 32.5, 43.2) for institutionalized and 35 percent (95% CI = 26.8, 43.5) for noninstitutionalized recipients.

Hospitalization rates continued to decrease from 1984 to 1986 for both institutionalized and noninstitutionalized recipients. Length of stay decreased further as well, but at a lesser rate. The total reductions in hospital days per person for the four-year period from 1982 to 1986 were 53 percent (95% CI = 48.9, 57.6) and 55 percent (95% CI = 48.2, 62.6) for institutionalized and non-institutionalized recipients, respectively.

We also examined the change in hospital use among those recipients who died. We hypothesized that those who died would, because of the severity of their illness, experience a smaller proportionate reduction in hospital care than the general caseload. The results shown in Table 2 indicate that, from 1982 to 1984, decedents experienced percentage reductions in hospitalization that were similar to those for the general elderly Medicaid population. Total average hospital days during the last six months of life decreased by 41 percent (95% CI = 28.4, 54.6) for institutionalized decedents and by 39 percent (95% CI = 16.3, 61.7) for noninstitutionalized decedents. From 1984 to 1986, there was no significant change in this measure.

Changes in Mortality Rates

Figure 1 presents the age-adjusted mortality rate for institutionalized elderly Medicaid recipients in Hennepin County for 1977–86. The mortality rate shows an increasing trend, although the number of pre-1983 observations is small and large fluctuations that occurred in 1978 and 1979 appear out of character with other year-to-year changes. An examination of the age-adjusted mortality rates for the remainder of the state's institutionalized Medicaid caseload showed that identical fluctuations occurred in all areas of the state during

TABLE 1—Hospital Utilization among Medicaid Recipients Age 65 and Older

	Year						
	1980	1981	1982	1983	1984	1985	1986
Institutionalized							
Number of Recipients	6800	7070	6996	7122	7296	7348	7239
Admissions per year per 1000 recipients	361 (343.2, 378.8)	379 (397.2, 360.8)	387 (369, 405)	356 (338.6, 373.4)	322 (304.9, 339.1)	297 (281.1, 312.9)	258 (244.5, 271.5)
Mean LOS per admission	12.2 (11.75, 12.65)	11.5 (11.09, 11.91)	11.3 (10.87, 11.73)	9.7 (9.33, 10.07)	8.4 (8.09, 8.71)	8.3 (7.93, 8.67)	7.9 (7.57, 8.23)
Hospital days per recipient per year	4.4 (4.13, 4.67)	4.4 (4.14, 4.66)	4.4 (4.14, 4.66)	3.5 (3.72, 3.28)	2.7 (2.52, 2.88)	2.5 (2.32, 2.68)	2.0 (1.84, 2.16)
Non-institutionalized							
Number of Recipients	3817	3907	3632	3630	3776	3846	3630
Admissions per year per 1000 patients	496 (467, 525)	475 (446.8, 503.2)	538 (506.8, 569.2)	468 (438.8, 497.2)	415 (388.5, 441.5)	340 (316.5, 363.5)	293 (271, 315)
Mean length of stay per admission	11.2 (10.6, 11.8)	10.5 (9.9, 11.1)	10.1 (9.4, 10.8)	8.8 (8.3, 9.3)	8.5 (7.8, 9.2)	7.8 (7.4, 8.2)	8.3 (7.3, 9.3)
Hospital days per Recipient per year	5.6 (2.07, 2.73)	5.0 (4.59, 5.41)	5.4 (4.97, 5.83)	4.1 (3.75, 4.45)	3.5 (3.15, 3.85)	2.6 (2.34, 2.86)	2.4 (2.07, 2.73)

95% Confidence intervals appear in parentheses.

TABLE 2—Hospitalization Utilization among Medicaid Decedents Age 65 and Older during the Last Six Months of Life

	Year						
	1980	1981	1982	1983	1984	1985	1986
Institutionalized							
Number of Decedents	474	512	550	546	615	569	596
Approximate number of Admissions per 6 months/1000 decedents*	572 (560, 635)	607 (545, 669)	573 (514, 632)	476 (422, 530)	452 (402, 502)	415 (365, 465)	443 (393, 493)
Approximate mean length of stay	13.1 (11.5, 14.7)	12.1 (10.8, 13.4)	11.3 (N/A)**	9.9 (N/A)**	8.4 (7.5, 9.3)	9.0 (7.7, 10.3)	8.7 (7.7, 9.7)
Hospital Days per Recipient in final 6 months	7.5 (6.3, 8.7)	7.3 (6.2, 8.4)	6.5 (5.5, 7.5)	4.7 (3.8, 5.6)	3.8 (3.2, 4.4)	3.7 (2.9, 4.5)	3.8 (3.1, 4.5)
Non-institutionalized							
Number of Decedents	48	65	75	58	78	49	51
Approximate number of Admissions per 6 Months/1000 decedents*	1354 (1079, 1629)	1015 (805, 1225)	1240 (1028, 1452)	983 (760, 1206)	1115 (922, 1308)	878 (639, 1117)	1196 (948, 1444)
Approximate mean length of stay	14.8 (10.6, 19)	13.4 (10.1, 16.7)	15.7 N/A**	13.8 N/A**	10.7 (7.2, 14.2)	13.9 (8.9, 18.9)	15.4 (4.1, 26.7)
Average # of Hospital Days per Recipient in final 6 months	20.0 (14.6, 25.4)	13.6 (9.4, 17.8)	19.5 (14.8, 24.2)	13.6 (8.9, 18.3)	11.9 (8.5, 15.3)	12.2 (5.6, 18.8)	18.4 (5.0, 31.8)

95% confidence intervals appear in parentheses.

*In this tabulation "admissions" were counted as the number of different months during which a decedent was admitted. Since a decedent might have more than one admission in a single month, these counts slightly underestimate the true admission rate, and result reciprocally in slightly overestimating the mean length of stay.

**Data needed for the calculation of this confidence interval are not available for these years.

those years, suggesting the possibility of an infectious disease epidemic. There was no consistent trend in age-adjusted mortality rates for the much smaller noninstitutionalized caseload.

To determine whether these changes were specific to the institutionalized Medicaid elderly, we also examined age-adjusted mortality rate trends in the general population of approximately 110,000 persons age 65 and over (Medicaid and non-Medicaid) in Hennepin County. Figure 2 shows that the age-adjusted mortality rates for Hennepin County elderly

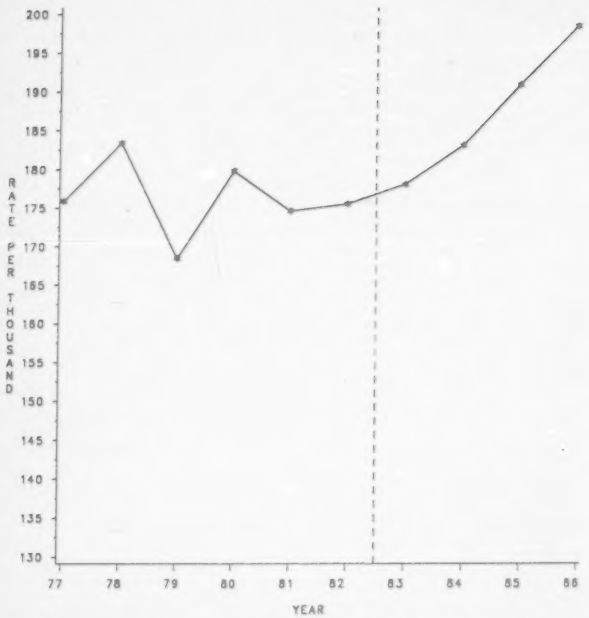

FIGURE 1—Age-Adjusted Mortality Rate, Institutionalized Medicaid Recipients, Age 65+

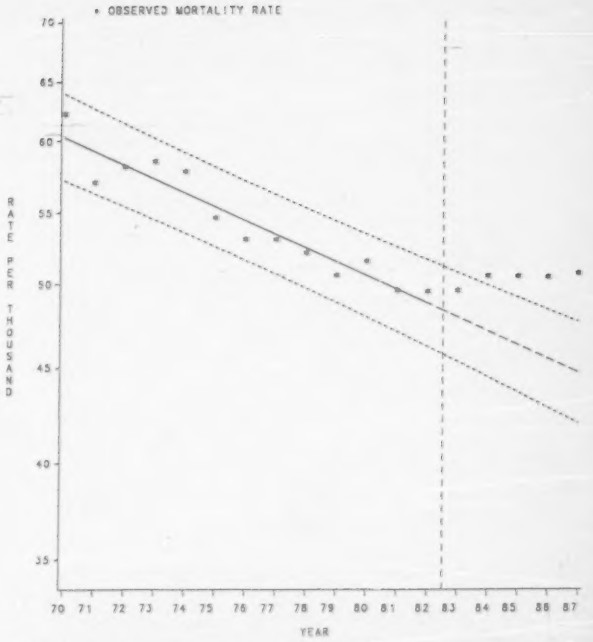

FIGURE 2—Age-Adjusted Mortality Rate for Estimated and Projected Hennepin County Population, Age 65+; Linear Regression and 95% Confidence Intervals for Years 1970–82 Projected for 1983–87

followed a roughly linear pattern of decline during the pre-cost containment period, falling from 62 per 1,000 in 1970 to 48/1,000 in 1982, a 22.5 percent reduction. Nationally, during a comparable 13-year period, 1968–80, there was a 24.2 percent reduction in age-adjusted mortality for persons age 65 and above.[13]

A linear regression line was fitted to the log-transformed 1970–82 observations to estimate the trend during the pre-cost containment period. Both the regression estimate and the 95 percent confidence interval for the pre-cost containment observations were projected through the year 1987 for comparison with observations in the post-cost containment period (1984–87). Figure 2 shows that, during 1984–87, the years following the institution of the PPS and other cost containment measures, the estimated age-adjusted mortality rates have ceased to fall and, in fact, are above the 95 percent confidence interval associated with the trend of the previous 13 years. While this does not represent an increase in the mortality rate from pre-cost containment years, it is a noteworthy departure from the mortality rate trend. It should be noted that the random variation in the mortality rate does not permit the identification of the specific year in which the mortality rate stops declining. We have selected 1983 as a break point because of the major policy changes instituted that year. Even if the break in the trend had actually begun in an earlier year, our findings of significant deviations in 1984–87 would still hold.

Discussion

These data include a large reduction in hospital admission rates and average length of stay by Hennepin County Medicaid recipients following the implementation of the PPS and other cost containment measures. The reduction in average hospital length of stay comes as no surprise. Data published in 1986 by the Health Care Financing Administration indicate that the average number of hospital days per Medicare admission to Minnesota decreased from 9.4 in 1982 to 7.3 in 1985—a 22.3 percent reduction.[14] The National Hospital Discharge Survey showed a 13.9 percent decline nationally in the average length of stay among patients age 65 and above from 1982 to 1985.[15] To a lesser extent, length of stay also declined for younger patients (11.4 percent for 45–64 year olds and 5.9 percent for 15–44 year olds). The large reduction in hospital admission rates among Medicaid recipients in our study differs from the experience in Wisconsin as reported by Sager, but is consistent with the National Hospital Discharge Survey data* and with impressions from the field that physicians have reduced their use of hospital care in response to pressure from third party payers. The reduction in days of hospital care per Hennepin County Medicaid recipient from 1982 to 1986 (53 percent for institutionalized and 55 percent for non-institutionalized recipients) far exceeds the amount one might estimate by looking only at changes in average length of stay.

The decrease in hospital care was not limited to the "healthier" members of the caseload. Recipients who died had comparable reductions in the first year following implementation of the PPS, although not in subsequent years.

Of more concern is the trend toward increasing age-adjusted mortality for the institutionalized elderly Medicaid recipients and the cessation of the downward trend in age-adjusted mortality for the general elderly Hennepin County population as a whole.

There are several possible explanations for these findings and we do not know which of them contributes most heavily. Reductions in hospital use—either decreased admis-

*The National Discharge Survey showed a decrease of 7.6 percent from 1982 to 1985 in discharges per 1,000 age 65 and older, from 428.1 to 393.2.[15]

sion rates or shorter length of stay—may have contributed to halting the downward trend in mortality rates. Changes in quality of care, including premature or inappropriate hospital discharge to nursing homes of severely ill patients or reduced intensity of appropriate in-hospital services, may also explain these findings. Both of these explanations are consistent with some of the hypothesized but unproven effects of PPS.

An alternative or contributory explanation may be the changing nursing home case mix resulting not only from early hospital discharge under PPS but from the moratorium on new nursing home beds and the introduction of preadmission screening and expanded alternative care services. By keeping less severely ill patients out of nursing homes, these programs may have the effect of increasing the severity of illness, and thus the mortality rate, in the nursing home population. While this is a plausible explanation for the increasing mortality rate in the Medicaid nursing home caseload, it does not explain the leveling of the mortality rate trend in the general elderly Hennepin County population. In addition, increased mortality rates became apparent before nursing home utilization controls were fully implemented and before the Hennepin County Medicaid nursing home caseload stopped growing (it peaked in mid-1985). The moratorium on nursing home bed construction followed the largest expansion of nursing home beds in 10 years. Full implementation of preadmission screening was not reached until mid-1986.

Finally, there have been secular changes in attitudes about appropriate levels of aggressiveness in the care of the very old and of the terminally ill. Many physicians, patients and family members now favor less intensive or heroic treatment of such patients, which may result in lowering the mean age at death and in increasing death rates in nursing homes.

Several potential limitations must be considered in interpreting these data. First, the data are from one county (albeit a very large one) and may not be consistent with trends in other areas. A preliminary examination of statewide Medicaid data has, however, revealed similar trends in nursing home mortality rates throughout Minnesota. Second, the use of case closing codes to calculate mortality rates for Medicaid recipients may introduce some error, although Hennepin County Medicaid staff were confident that the reporting of deaths is complete and accurate for those in nursing homes and there was no indication that coding practices changed over time. Third, the finding of a change in the mortality trend for the general Hennepin County elderly population is dependent upon the accuracy of the Hennepin County population projections used in the analysis. If the population projections erred on the low side, they would yield an inflated age-adjusted mortality rate. While the accuracy of the projections cannot be verified until after the 1990 census, they are thought to be reliable for the relatively stable elderly population. The projections agreed closely with other Hennepin County population projections made independently by the State Demography Unit and by the State Health Department (personal communication).[16] The substitution of those projections in our analysis would not have altered our findings.

We cannot conclude that the change in the Hennepin County mortality rate trend is caused by the policy changes we have noted, but the data are troubling and underscore the need for analyses at multiple levels. Needed first is an examination of age-adjusted mortality trends at multiple sites, controlling for differences in population characteristics, Medicaid eligibility, utilization control policies, health care resources, and the prevalence of HMO enrollment. In addition, there is an urgent need to examine the access to care and the quality and appropriateness of care received by elderly Medicare and Medicaid patients, particularly those with life-threatening or terminal illnesses, and those in nursing homes.

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Prevalence of Depressive Symptoms in a Japanese Occupational Setting: A Preliminary Study

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Abstract: We measured the prevalence of depressive symptoms in 2,190 Japanese tax office workers using the Japanese version of the Center for Epidemiologic Studies Depression Scale (CES-D). Score distribution by sex was more symmetrical and the mean score of each sex was higher than in the United States population. A high level of depressive symptoms was found in 15.2 percent of males and 10.6 percent of females by controlling for age and marital status. Males aged 50 years and over had more depressive symptoms than other male age groups. Perceived stress, related both to family life and the workplace, was associated with a high level of depressive symptoms. "Long-distance marriage" ("business bachelorhood"), peculiar to Japanese occupations, had little influence on depressive symptomatology. (*Am J Public Health* 1989; 79:1486–1489.)

Introduction

In industrial countries, social and workplace environments have become more complicated and stressful. Recent epidemiologic studies in the United States and some European countries have found depressive disorders to be the most common among mental disorders in the community.[1] Advances in the psychiatric epidemiology of depression include the introduction of operational classificatory schemes[2] and the application of conventional self-rating measurements to population prevalence studies.[3–6] In Japan, there are few epidemiologic studies in these areas.

Self-rating depression scale, the Center for Epidemiologic Studies Depression Scale (CES-D),[7] has been widely applied to population surveys in the US and satisfactory levels of validity and reliability have been reported.[3–14] The CES-D was translated into Japanese by Shima, *et al*,[15] in 1985. In a comparison between depressed patients and normal controls, they found that the CES-D in Japanese translation had clinical validity and that its standard cut-off point of 15/16 was appropriate for Japanese use.[15] We also found the CES-D had a good level of concurrent validity with other indices of depression and psychological distress and an acceptable level of internal reliability in a sample of Japanese workers.[16,17]

The CES-D has also been applied in several surveys of other ethnic groups,[18–23] but it has been rarely used in an occupational setting, although some studies reported the values of depressive symptomatology based on the CES-D for a subsample of the employed in the surveyed community populations.[6,8,12,14,19,24,25] In this study we present basic data on the prevalence of depressive symptomatology as measured by the CES-D in a Japanese occupational setting.

Methods

Subjects

All 2,190 workers ages 19–63 years of 29 tax offices in Hokkaido, the northern part of Japan, were asked to complete the CES-D, other psychiatric scales, and several additional questions about sociodemographic status and psychosocial factors. The survey was carried out in September 1986. Complete CES-D data were obtained from 2,016 subjects (92.1 percent; 1,870 males, 142 females, and four with sex not specified).

The subjects were approximately the same educational level (years of education = 13), stable income-earners; all were full-time employees. Few of the subjects were separated or divorced. Generally they seemed to be representative of the workers of the Japanese government offices such as the tax office, even with respect to the low proportion of female workers.

Measurements

The Japanese translated version of the CES-D[15] has 20 items rated on a four-point Likert scale ("rarely or none of the time" = 0 to "most or all of the time" = 3). A score of 16 or higher indicates a high level of depressive symptoms as used both in the United States[7–10] and Japan.[15] Each psychosocial factor was measured by one item on a five-point rating scale with response categories (and scores) from "not at all (1)" to "a large amount (5)".

Analyses

The mean CES-D score and the proportion of subjects with high level of depressive symptoms value (CES-D score

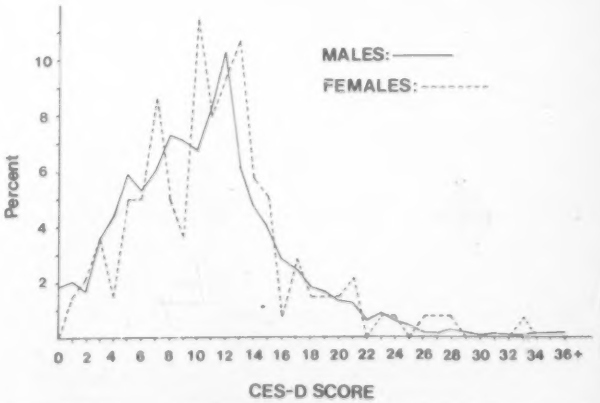

FIGURE 1—Percentage distribution of the Japanese version CES-D score by sex in a sample of Japanese workers.

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of 16 or higher) were calculated by sociodemographic status and psychosocial factors. The adjusted values, controlling for the effects of age (generation) and/or marital status, were also estimated. Because there were few separated or divorced subjects, this category was combined with the "never married". Analyses were performed separately by sex.

TABLE 1—Mean CES-D Score and Percentage of the Subjects with High Level of Depressive Symptoms (CES-D score of 16 or more) among Japanese Workers Ages 19–63 Years by Sociodemographic Status and Psychosocial Factors

	Males					Females				
		Mean CES-D score		% of High Scorers			Mean CES-D score		% of High Scores	
Factors	N	Crude	Adjusted	Crude	Adjusted	N	Crude	Adjusted	Crude	Adjusted
Total sample	1,793 (77)[a]	10.5	10.5	15.0	15.2	132 (10)	11.1	10.5	14.1	10.6
Sociodemographic Status										
Age (years)[b]										
19–29	414	11.2	10.2	18.8	12.2	56	11.6	11.1	21.4	18.3
30–39	633	9.9	10.1	11.9	13.3	20	12.1	12.5	15.0	17.2
40–49	267	10.3	10.7	12.7	15.4	25	9.8	10.1	0.0	2.1
50–63	479 (77)	10.7	11.1*	16.9	19.3*	21 (10)	10.5	10.9	12.5	15.5
Marital status[c]										
Married	1,423	10.1	10.0	12.7	12.1	64	10.0	10.0	6.3	7.6
Never married or separated/divorced	370 (77)	11.9	12.2*	23.8	26.0***	68 (10)	12.1	12.1*	22.1	20.8*
Number of children[d]										
0	163	10.1	10.3	14.7	16.4	14	10.1	9.9	7.1	3.3
1	315	10.5	10.6	14.9	15.6	14	8.6	8.6	7.1	5.7
2	804	9.9	9.8	11.6	11.1	31	10.6	10.7	6.5	8.3
3+	141	10.3	10.2	11.4	10.8	5	9.6	9.7	0.0	3.2
Residential status[c]										
Living with family	1,373	10.2	10.2	13.2	13.4	102	10.8	10.9	13.7	14.1
Long-distance marriage	154	10.8	10.6	16.2	14.4	3	5.7	4.5	0.0	0.0
Living alone	254 (89)[a]	11.7	11.6**	23.2	23.3***	24 (13)	12.4	12.3	20.8	19.8
Psychosocial factors										
Conjugal Relationship[d]										
Very good (1)	305	8.0	8.0	4.6	4.8	14	8.5	8.3	0.0	0.0
2	505	10.0	10.0	12.5	12.6	15	9.0	8.8	0.0	0.0
3	594	11.0	11.0	15.8	15.6	28	11.2	11.4	14.3	15.0
Bad/very bad (4/5)	10 (9)	23.1	23.3***	70.0	70.9***	— (7)	—	—	—	—
Parental relationship[d]										
Very good (1)	309	8.2	8.2	5.5	5.7	13	8.5	8.1	0.0	0.0
2	491	9.9	10.0	12.0	12.2	20	9.7	9.9	0.0	1.7
3	517	11.2	11.2	16.3	16.0	22	11.6	11.7	18.2	18.1*
Bad/very bad (4/5)	10 (96)	15.1	15.0***	20.0	19.6***	— (9)	—	—	—	—
Perceived stress related to family life										
Not at all (1)	675	8.6	8.6	6.4	6.0	35	10.3	9.5	8.6	1.9
2	860	10.8	10.8	15.7	15.9	70	11.0	11.3	14.3	17.2
3	185	13.7	13.8	32.4	32.9	21	10.6	11.0	14.3	16.4
A large amount (4/5)	49 (101)	18.1	17.9***	55.1	53.8***	5 (11)	16.4	16.0	40.0	37.4
Perceived stress related to the workplace										
Not at all (1)	286	7.6	7.5	2.8	2.2	18	7.0	6.6	0.0	0.0
2	867	9.4	9.5	9.1	9.2	62	10.4	10.6	6.4	6.1
3	494	12.4	12.5	24.3	24.6	35	11.0	10.9	14.3	14.0
A large amount (4/5)	121 (102)	16.6	16.5***	47.9	47.8***	16 (11)	17.7	17.3***	56.3	54.5***
Seeking medical counseling										
Yes (wanted)	216	12.0	12.1	26.4	26.3	6	18.7	18.6	50.0	49.6
No (not wanted)	1,306 (348)	10.1	10.2***	13.6	13.7***	99 (37)	10.0	10.0***	11.1	11.1***

Note: "adjusted" values are estimated by controlling for age (generation) and marital status.
N: Number of the subjects in the subcategory.
a) Number of missing subjects is shown in brackets.
b) The values adjusted with controlling the effect of marital status are presented.
c) The age-adjusted values are presented.
d) Only married subjects were analyzed, so that the age-adjusted values are presented.
*, **, and ***: significance level of $p < 0.05$, $p < 0.005$, and $p < 0.001$, respectively.

TABLE 2—Selected Results of Reported Mean CES-D Score and Percentage of High Level of Depressive Symptoms (CES-D score of 16 or more) for US and Mexican Workers Based on Community Surveys[a]

Study	Research Site Sample of Workers	Value of Depressive Symptomatology (adjusted value) Mean	Rate
Comstock & Helsing[8] (1976)	Washington County		%
	Whites	—	14.4 (15.1)
	Blacks	—	22.4 (12.3)
	Kansas City Whites	—	17.1 (17.8)
Eaton & Kessler[12] (1981)	HANES sample	—	13.1 (15.7)[b]
Frerichs, *et al.*[25] (1981)	Los Angeles county	8.0	14.0 (13.7)
Aneshensel, *et al.*[24] (1981)	Los Angeles county		
	Males	4.32 (4.35)	—
	Married/Unmarried	3.76/5.52 (4.14/5.26)	
	Females	5.57 (5.15)	—
	Married/Unmarried	5.17/6.23 (5.57/5.62)	
Gary[14] (1985)	A northeastern city		
	Selected Black males	10.78	—
Vega, *et al.*[19] (1987)	Tjuana, Mexico Mexican	8.07	—
Mendes de Leon & Markides[6] (1988)	San Antonio, Texas Mexican-Americans		
	Middle-aged	7.4 (7.5)	11.0 (12.0)
	Younger-aged	8.1 (8.6)	12.0 (15.0)
Present study	Hokkaido, Japan Tax workers		
	Males	10.5 (10.5)	15.0 (15.2)
	Females	11.1 (10.5)	14.1 (10.6)

Adjusted values are generally estimated by controlling for sex, age, marital status, education, and income.

a) Only the results of full-time employees are presented.

b) The effect of race was also taken into account.

Results

Figure 1 displays the percentage distribution of the CES-D scores by sex. Although the distribution of females had dichotomous peaks (scores at 10 and 13) and was not artificially smoothed, there was a much smaller number of female subjects, so that the distribution of each sex was relatively similar and not heavily skewed (males: mode = 12, skewness = 1.04; females: mode = 10, skewness = 0.94) therefore standard parametric tests on these data were deemed to be appropriate.

Table 1 shows the mean CES-D score and the proportion of subjects with high level of depressive symptoms by sociodemographic status and psychosocial factors. There were no sex differences in either indicator of depressive symptomatology. When adjusted for the effect of marital status, a weak linear relationship with age was found in males. Males ages 50 years or more had more depressive symptoms than other age groups. For other sociodemographic status, both male and female subjects who were unmarried (never married or separated/divorced) and living alone had a higher level of depressive symptoms.

Among psychosocial factors, the subjects who perceived poor relationships with spouse and with parents were more depressed as were subjects who reported more stress in their family life and in the workplace. Subjects who desired medical counseling were also more depressed.

Discussion

United States studies have reported values of depressive symptomatology based on the CES-D for the employed in community populations as displayed in Table 2. Since the present results were derived from an occupational survey, they may not be comparable to the values in the table. Mean CES-D scores for both sexes were higher in our study than in the US employed, except for the Black men surveyed by Gary[14]; nevertheless the percentages of high level depressive symptoms were similar to the US employed.

One reason for the disparity of mean score between Japan and the United States may be the difference in response frequencies of the negative and positive items. The Japanese tend to choose an intermediate response to the positive items when compared to the US population so that the score distributions of the scale of Japanese subjects show generally lower frequencies than the US counterparts at the low symptom portion. Such a tendency may reflect the sociocutural and/or personality characteristics of Japanese who tend to avoid extreme answers to anything.

In Japan, many male employees have long-distance marriages (business bachelorhood, living alone separated from family members as a result of being promoted to a new job in a distant place). This situation may last for several years. Males in the subclass of the long-distance marriages were somewhat more likely to have depressive symptoms than those who were living with family members, but far less than others who were living alone.

Kessler and Essex[26] and Gove, *et al*,[27] reported that marriage had positive effects on depression and psychological well-being. The present study supports this observation; it also demonstrates that perceived stress in family life and in the workplace is related to depressive symptomatology. Worker depression has been related to such factors as work overload and job pressures in other studies.[28,29]

The limitations of the present results include the nature of the sample and the fact that each psychosocial factor is measured by a single question; a direct question about self-perceived job stress has been reported as a good predictor of subsequent ill health.[29] This should be considered a preliminary study of the mental health of Japanese workers. Further research is needed based on several samples of Japanese workers and a more thorough exploration of stress to examine the factors affecting depressive symptomatology of Japanese workers.

ACKNOWLEDGMENTS

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15th National Primary Care Nurse Practitioner Symposium: Call for Abstracts

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Send three typed copies (300 words or less), format includes title, brief statement of purpose, methods, conclusions, and clinical application(s) or brief summary of project/paper if not research. Include author vita and indicate interest in presentation or poster session.

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Person-Years of Life Lost Due to Cancer in the United States, 1970 and 1984

JOHN W. HORM, MSc, AND EDWARD J. SONDIK, PhD

Abstract: The number of deaths due to cancer in the United States reached an all-time high of 453,450 deaths in 1984 and, due to the dynamics of population growth, will continue to increase if the risk of dying from cancer does not change.

Between 1970 and 1984, the total Person-Years of Life Lost (PYLL), the sum of the difference between the actual age at death and the expected remaining lifetime for each person who died of cancer, increased for most cancer sites as well as for all sites combined. In 1984, 6,881,281 person-years of life were lost due to cancer deaths, up from 5,303,668 in 1970. The exceptions are those cancers for which there has been major progress in either prevention or treatment; e.g., stomach and cervix uteri (prevention) and testicular, Hodgkin's disease, leukemia, and childhood cancers (treatment).

The Average Years of Life Lost (AYLL) per person dying from cancer in 1984 was generally less than in 1970. Overall, each person who died from cancer in 1984 died 15.2 years earlier than his/her life expectancy. The greatest loss was for those who died of childhood cancers (66.9 years earlier), followed by testicular cancer (35.8 years earlier). The least loss relative to the expectation of life was for those who died of prostate cancer. The 25,400 men who died from prostate cancer in 1984 died an average of nine years earlier than otherwise expected. (*Am J Public Health* 1989; 79:1490–1493.)

Introduction

Deaths due to cancer accounted for 453,450 deaths in 1984[1] in the United States, second only to heart disease. Many of these deaths may be postponed or avoided entirely through the use of appropriate interventions such as primary prevention, screening/early detection, and increased application of state-of-the-art treatment.[2] A death which occurs before the end of average life expectancy is, by definition, a premature death. The age-adjusted and age-specific cancer mortality rates do not fully address the issue of premature mortality and therefore other measures are needed.[3]

Person-Years of Life Lost (PYLL) considers a cancer death to be premature in that the death occurred earlier than it would have occurred in the absence of that cancer.

Average Years of Life Lost (AYLL) is the average of the differences between the actual ages at death and the expected remaining years of life for each person who died of cancer.

In our computation of PYLL and AYLL we have used age-based and sex-specific estimates of the length of life remaining rather than fix an upper bound on length of life at age 65 such as has been used by others.

Taken together, the PYLL and the AYLL reflect the cost of dying from cancer in terms of lost years of life expectancy. They may also be used to assist in the evaluation of cancer control progress and to provide guidance for resource allocation in cancer prevention and control. We examined PYLL and AYLL for cancer in 1970 and 1984.

Methods

The numbers of cancer deaths for both 1970 and 1984 and information on the underlying cause of death as well as demographic information such as age, sex, race, and other items for each death occurring annually in the United States were obtained from the National Center for Health Statistics (NCHS). The deaths have been categorized by anatomic site of the cancer, sex, and age at death for each calendar year.

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For each cancer death which occurred in either 1970 or 1984, the PYLL was estimated by taking the difference between the actual age at which the person died and the expectation of remaining life for an individual matched by age and sex for each of 1970 and 1984 as obtained from life tables published yearly by the NCHS.[5,6] These differences were then summed over all persons dying of cancer for 1970 and for 1984.

The AYLL is a statistical average arrived at by simply dividing the total PYLL by the number of people affected. From Table 1, 4,564 women died prematurely from cancer of the cervix uteri in 1984 for a total of 105,899 person-years of life lost or an average of 23.2 years of life lost for each of the 4,564 women.

Results

Tables 1, 2, and 3 have for both 1970 and 1984 the numbers of cancer deaths in the United States, the total number of person-years of life lost due to cancer, and the average years of life lost for those who died of cancer. The cost in average years of life lost was greater for women (17.1) than for men (13.5) in 1984, due in part to the greater life expectancy for women compared to men, 78.2 years and 71.2 years, respectively. Even though there were fewer women than men dying from cancer in 1984 (210,687 versus 242,763), the women lost more potential years of life, 3.6 million compared to 3.3 million for men. For both sexes combined, lung cancer caused the loss of the most total person-years of life in 1984 followed by breast and colorectal cancer (Figure 1). Deaths due to testicular cancer, because of the small numbers of deaths, cost the fewest total person-years of life among those cancer sites investigated.

In contrast, the greatest average years of life lost per person dying of cancer was for childhood cancers (Figure 2), followed by deaths due to testicular cancer, Hodgkin's disease, cancer of the cervix uteri, brain cancer and finally malignant melanoma, each of which caused death more than 20 years before the life expectancy.

In 1984 the most costly cancer for women was cancer of the breast which caused a loss of over 760,000 person-years of life among 39,470 women, up 28 percent from 1970. These women died an average of 19.3 years earlier than expected. Mass screening programs have suggested that the mortality

TABLE 1—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, both Sexes, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	329,375	453,450	5,303,668	6,981,281	15.1	15.2
Oral cavity	7,610	8,461	117,854	134,095	15.5	15.8
Esophagus	5,969	8,657	87,905	126,758	14.7	14.6
Stomach	16,020	14,246	209,706	191,045	13.1	13.4
Colon & rectum	45,924	57,510	615,264	744,941	13.4	13.0
Pancreas	17,885	23,154	246,952	315,020	13.8	13.6
Lung	65,003	118,565	989,335	1,757,001	15.2	14.8
Melanoma (skin)	3,146	5,377	66,900	110,765	21.3	20.6
Breast	29,912	39,720	597,050	763,511	20.0	19.2
Cervix uteri	6,547	4,564	146,261	105,899	22.3	23.2
Corpus uteri	5,468	5,965	88,100	89,563	16.1	15.0
Ovary	9,831	11,208	193,462	200,279	19.7	17.9
Prostate gland	17,249	25,400	156,281	228,597	9.1	9.0
Testis	760	401	26,693	14,343	35.1	35.8
Urinary bladder	9,011	9,714	101,023	103,176	11.2	10.6
Kidney	6,103	8,411	100,310	127,937	16.4	15.2
Brain & CNS	6,027	9,866	153,882	215,805	25.5	21.9
Hodgkin's disease	3,410	1,960	89,249	53,022	26.2	27.1
Non-Hodgkin's lymphoma	9,592	14,486	176,232	232,071	18.4	16.0
Multiple myeloma	4,417	7,470	63,042	100,736	14.3	13.5
Leukemia	14,492	17,212	333,439	320,480	23.0	18.6
Childhood	3,611	1,884	233,435	125,951	64.6	66.9

TABLE 2—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, Males, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	179,352	242,763	2,558,996	3,284,558	14.0	13.5
Oral cavity	5,495	5,764	79,810	87,527	14.5	15.2
Esophagus	4,442	6,312	62,308	90,091	14.0	14.3
Stomach	9,802	8,469	121,490	107,681	12.4	12.7
Colon & rectum	22,117	27,989	269,512	338,649	12.2	12.1
Pancreas	10,061	11,516	130,937	149,202	13.0	13.0
Lung	52,680	82,385	751,081	1,113,112	14.3	13.5
Melanoma (skin)	1,735	3,207	34,394	62,209	19.8	19.4
Breast	265	250	3,552	3,409	13.4	13.6
Prostate gland	17,249	25,400	156,281	228,597	9.1	9.0
Testis	760	401	26,693	14,343	35.1	35.8
Urinary bladder	6,257	6,600	66,853	66,762	15.3	14.4
Kidney	3,858	5,201	59,091	74,664	15.3	14.4
Brain & CNS	3,434	5,395	81,686	111,329	23.8	20.6
Hodgkin's disease	2,016	1,204	50,566	31,461	25.1	26.1
Non-Hodgkin's lymphoma	5,233	7,312	94,256	115,937	18.0	15.9
Multiple myeloma	2,340	3,737	30,269	46,449	12.9	12.4
Leukemia	8,128	9,378	174,139	166,464	21.4	17.8
Childhood	2,039	1,073	125,194	68,257	61.4	63.6

from breast cancer can be reduced by 30 percent through screening.[7,8] If so, a screening program could prevent about 11,800 breast cancer deaths per year. If these 11,800 women were to live to their full life expectancy, about 227,000 person-years of life could be saved yearly.

The 36,180 women who died from lung cancer in 1984 lost a total of 643,889 person-years of life, almost triple the 1970 figure of 238,254 person-years. In 1984, these women died an average of 17.8 years before expected, an improvement over 1970 when female lung cancer victims died 19.3 years before their life expectancy. It is believed that about 75 percent of lung cancer deaths among women are caused by cigarette smoking.[9] Preventing 75 percent of these lung cancer deaths could result in the postponement of death by some 19 years for over 27,000 women annually. This would result in the saving of over 500,000 person-years of life every year.

Additionally, 75 percent of the deaths due to cancer of the cervix uteri are thought to be preventable through screening with the Pap smear.[10] Pap smear screening could potentially extend the lives of 3,400 women by 23 years each for a total savings of 78,200 person-years of life.

Dramatic improvements have been noted in the person-years of life lost among women between 1970 and 1984 for Hodgkin's disease with a 44 percent decrease and, most dramatically, childhood cancers with less than half as many person-years of life lost in 1984 than in 1970. Smaller decreases are seen for cancers of the stomach and for

TABLE 3—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, Females, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	150,023	210,687	2,744,672	3,596,723	18.3	17.1
Oral cavity	2,115	2,697	38,043	46,568	18.0	17.3
Esophagus	1,527	2,345	25,597	36,667	16.8	15.6
Stomach	6,218	5,777	88,216	83,364	14.2	14.4
Colon & rectum	23,807	29,521	345,751	406,293	14.5	13.8
Pancreas	7,824	11,638	116,015	165,817	14.8	14.2
Lung	12,323	36,180	238,254	643,889	19.3	17.8
Melanoma (skin)	1,411	2,170	32,507	48,556	23.0	22.4
Breast	29,647	39,470	593,498	760,102	20.0	19.3
Cervix uteri	6,547	4,564	146,261	105,899	22.3	23.2
Corpus uteri	5,468	5,965	88,100	89,563	16.1	15.0
Ovary	9,831	11,208	193,462	200,279	19.7	17.9
Urinary bladder	2,754	3,114	34,171	36,414	12.4	11.7
Kidney	2,245	3,210	41,219	53,273	18.4	16.6
Brain & CNS	2,593	4,471	72,197	104,476	27.8	23.4
Hodgkin's disease	1,394	756	38,683	21,561	27.7	28.5
Non-Hodgkin's lymphoma	4,359	7,174	81,976	116,135	18.8	16.2
Multiple myeloma	2,077	3,733	32,773	54,287	15.8	14.5
Leukemia	6,364	7,837	159,300	154,015	25.0	19.7
Childhood	1,572	811	108,241	57,695	68.9	71.1

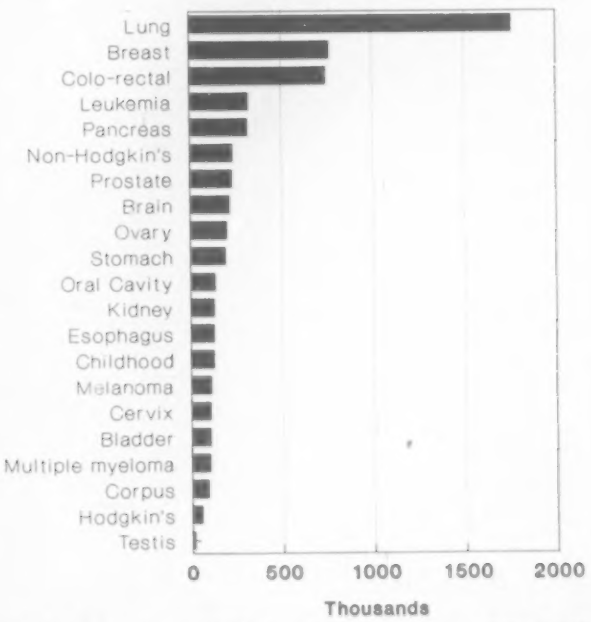

FIGURE 1—Person-Years of Life Lost (PYLL) due to Cancer, all Races, both Sexes, 1984

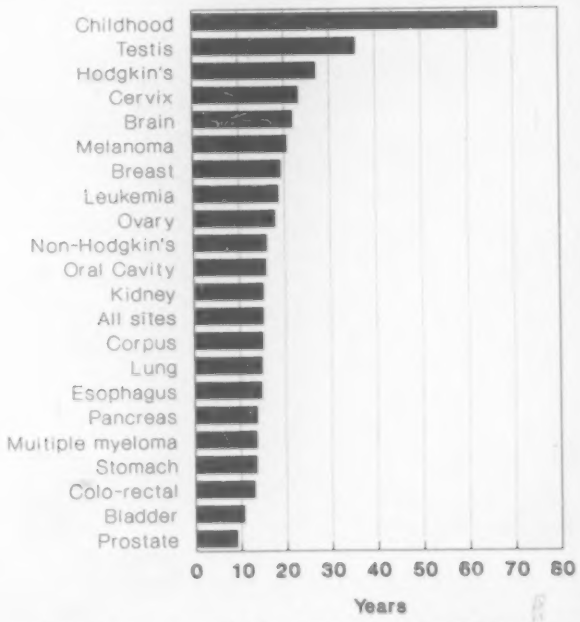

FIGURE 2—Average Years of Life Lost (AYLL) due to Cancer Deaths, all Races, both Sexes, 1984

leukemias.

Among men, lung cancer was the most deadly cancer causing the loss of the most person-years of life in 1984. Over 1.1 million person-years of life were lost among 82,385 men who died of lung cancer an average of 13.5 years earlier than otherwise expected. If 80 percent of these deaths could be avoided through prevention, almost 900,000 person-years of life could be gained annually.

Colorectal cancer deaths accounted for 338,649 lost person-years of life in 1984, up by 26 percent since 1970. Each man who died from colorectal cancer in 1984 died, on the average, 12.1 years before he would have otherwise expected to die.

Deaths from prostatic cancer among 25,400 men cost almost 229,000 person-years of lost life in 1984 as these men died nine years before their average life expectancy would predict.

Five cancer sites among those examined actually demonstrated decreases in the number of person-years of life lost due to cancer deaths between 1970 and 1984. Both testicular and childhood cancer had decreases in the person-years of life lost of about 45 percent from 1970 to 1984 amounting to

a savings of 69 thousand person-years of life in 1984 alone. The improvement in testicular cancer is primarily due to the development of curative chemotherapy for nonseminoma cases which has increased the five-year survival rate to over 80 percent.[14,15] Although testicular cancer is relatively rare, it causes death an average of 35.8 years before the normal life expectancy. Male childhood cancer victims died 63.6 years earlier than expected in 1984 with 68,257 person-years of life lost among 1,073 boys. Additional savings are seen for Hodgkin's disease, a 38 percent improvement; stomach cancer, an 11 percent improvement; and a 4 percent improvement for leukemia.

Discussion

The methodology used here differs from that used in previous investigations in that here we have used the life expectancy as the endpoint rather than age 65.[11–13] We believe that the life expectancy is a more appropriate endpoint for such an analysis because a large proportion of cancer deaths, about 65 percent, as well as 70 percent of non-cancer deaths occur over the age of 65 and today people remain productive and active well beyond the classical retirement age of 65. Further, we would argue that any death which is either preventable or avoidable, and which occurs before the end of the affected person's normal lifespan, is premature and hence should contribute to person-years of life lost. It should be noted that in 1984, a person 65 years old could expect to live for another 16.8 years.

The PYLL and AYLL are estimates of the differences between the actual age at death and the expected age at death considering all possible causes of death weighted by their occurrence in the population at large. These estimates do not depend on the date of diagnosis nor on the time between diagnosis and death (survival) and thus are free of some of the problems inherent in the interpretation of trends in incidence and survival rates due to diagnostic artifacts such as lead time bias.

Ideally we would like the PYLL to decrease over time. However, due to the aging of the population and to the increasing size of the population and therefore the greater numbers of persons at risk of death from cancer, it is possible for the PYLL to increase without an increase in the age-sex-specific risk of dying of cancer. The PYLL then estimates the actual impact of the cancer deaths on the population in terms of total person-years of life lost every year. As the impact of competing causes of death on the population lessens, the PYLL due to cancer will continue to become numerically larger in the United States for the reasons outlined above.

On the other hand, the AYLL (Average Years of Life Lost) estimates the joint effects of the changes in the cancer patients' ages at death and the change in the population life expectancy. The life expectancy at birth of the United States population increased by almost four years between 1970 and 1984, from 70.9 to 74.7, respectively. The expectation of remaining life also increased between 1970 and 1984 for all races combined, for each sex, and at every age with larger increases for younger persons and lesser increases for older persons. For making comparisons over time, we can artificially hold the overall life expectancy constant. In so doing, the comparison of two AYLLs shows the change in the average loss of expected life-years per cancer death.

It is important to note that the AYLL is based only on those people who die from cancer and therefore does not provide an estimate of either cure or prevention. For example, the number of people dying from testicular cancer decreased from 760 in 1970 to 401 to 1984 due to the advent of curative treatment but the AYLL increased because of different age and stage distributions between those who died and those who were cured. We also note that with no change in cancer patient survival, the AYLL would be expected to increase in accordance with the increasing life expectancy of the United States population. Therefore, we consider any changes in AYLL as a conservative (pessimistic) estimate of the curative life extension impact of cancer treatment.

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Breast Cancer Risk and Participation in Mammographic Screening

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Abstract: Within the context of an organized breast cancer screening program we conducted a prospective evaluation of the relation between breast cancer risk and participation in mammographic screening. The influence on participation of known breast cancer risk factors, as well as a summary risk label, (i.e. "high", or "moderate") were examined. The overall participation rate was 71 percent among 2,422 women, 50 to 79 years of age, invited to a centralized clinic. Multivariate analyses showed participation to be somewhat decreased among women with late menopause and definitely increased among women with any of the following factors: 1) increased age; 2) a family history of breast cancer; and 3) a previous breast biopsy. Women in the high-risk group were most likely to participate but the effect of the label was stronger among women ages 50 to 59 compared to women ages 60 to 79. The study results are generally consistent with previous findings that participants in screening programs have higher rates of breast cancer. The results also suggest the possibility that providing breast cancer risk information may encourage participation in screening. (*Am J Public Health* 1989; 79:1494–1498.)

Introduction

Self-selection bias has been a major concern in the assessment of cancer screening modalities.[1–3] The Health Insurance Plan (HIP) randomized trial of mammography demonstrated that breast cancer rates were higher among women who complied with an invitation to obtain a mammogram (participants) compared to those in the study population who did not comply with that invitation.[4] Several investigators have shown a higher prevalence of breast cancer symptoms among participants, but the influence of breast cancer risk factors on screening behavior has not been thoroughly evaluated.[5]

Investigators have examined the relationship between women's use of mammography and three risk factors: age, family history, and previous benign breast disease.[5–10] All but one of the studies were conducted retrospectively.[7] Participation appears to decrease with advancing age,[5–7] although one report showed increased participation,[8] and yet another found no association.[9] Two studies found no association between family history and participation.[5,6] A third study reported that participants in screening were more likely to have a family history of breast cancer, but this finding was based on very small numbers.[9] One study has examined the role of previous benign breast disease and showed an association in a negative direction with subsequent participation in screening, but the definition of this disorder was not explicit.[9]

The study reported here prospectively evaluates the association between participation in an organized screening program and the following six risk factors: age, family history of breast cancer, having a previous benign breast biopsy, nulliparity to age 30, early age at menarche, and late age at menopause. Previous work has suggested that perceived vulnerability to breast cancer is positively associated with participation in screening.[5,7,8] We, therefore, hypothesized that those factors which might be readily identified by a woman as increasing her risk of breast cancer (i.e., increased age, family history, and having needed a breast biopsy) would show positive associations with participation in screening. In contrast, we expected that nulliparity, early age at menarche, and late age at menopause would not be associated with participation in screening, as women are less likely to be aware that these factors increase their risk for breast cancer. In addition to the association between specific risk factors and participation in screening, this study also assessed the effect of being informed that one is at high risk vs moderate risk for breast cancer.

Methods

The study was conducted at Group Health Cooperative of Puget Sound (GHC), a 360,000-member closed panel health maintenance organization (HMO) that includes two hospitals and 21 primary care facilities in Western Washington state. Women GHC enrollees are slightly better educated and more likely to be in households with higher incomes than the general population of comparably aged women residing in Washington state.[11]

In March of 1985, Group Health Cooperative initiated a risk-based Breast Cancer Screening Program.[12,13] All female enrollees 40 years of age and older were mailed a two-page questionnaire which elicits information concerning breast cancer risk factors, medical and screening history, and selected lifestyle behaviors. Seven of 21 items on the questionnaire pertain to specific breast cancer risk factors. The response rate to this survey has been 85 percent.[13,14] Based on their personal constellation of risk factors, all women are assigned by the screening program to one of four risk categories: "high," "moderate," "borderline," or "no increased risk" which represent 18, 27, 12, and 43 percent of the population, respectively (see Appendix). Each woman who completes and returns this questionnaire is sent a follow-up letter, within two months, which indicates her risk category and a recommendation to: perform monthly breast self-examination, obtain a breast physical examination annually, and be seen in the breast cancer screening center for a mammogram every 1, 3, or 5 years according to risk level. Letters of invitation do not convey any risk factor information other than a woman's risk category. An automated data system maintains the survey data and documents all screening activity. These data served as the basis for the analysis of participation presented here.

The sample for this analysis consisted of women who had completed a risk factor questionnaire, were invited for a screening visit between March 1, 1985 and December 31, 1985, reported no mammogram during the one year prior to

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being surveyed, and were continuously enrolled at GHC since being invited to come in for screening. The study group was limited to women ages 50 to 79 who were in the "high" and "moderate" risk categories (Table 1). Of the 2,722 women in this group, 2,422 received invitations to the screening program during this period and constitute the study population. The non-study population consists of the 300 women from this group who did not receive invitations during the study period plus 4,498 women whose only risk factor was age. At the time of this study, these women were placed in a group designated as "no increased risk" and were not invited for a screening visit. This policy has subsequently changed.[13]

Participation was defined as scheduling and completing an appointment in the screening center by June 30, 1986. There was no fee for this visit which included instruction in breast self-examination and a physical examination of the breast, as well as the mammogram. Subjects had a minimum of six and maximum of 15 months opportunity to come in for screening following their invitation. At the time of this study, the wait time to obtain a mammogram was approximately one month. Analysis of overall program data shows that 93 percent of women who participate within one year do so within the first six months after being invited. Women received only one invitation and were not sent any reminders.

The relation between participation in screening and the following six factors was considered: age, family history of breast cancer, previous benign breast disease, menarche at 10 years of age or younger, menopause at 55 years of age or older, and nulliparity to age 30. A positive family history was defined as having one or more first or second degree family members with a history of breast cancer (i.e., mother, sister, daughter, grandmother, or aunt). Benign breast disease was defined as having reported one or more breast biopsies which were non-cancerous. In addition to considering the six individual risk factors, the effect on participation of being assigned a "high" vs "moderate" risk label was examined.

Bivariate contingency table analyses were carried out initially to assess the association between participation and each of the seven variables considered individually. Logistic regression was then used to estimate the effect of each variable controlling for the influence of all others. The logistic analysis was done in two steps. First, models were fit to test if there were any significant interactions between risk label and the other variables. Then, a final logistic model was fit containing the seven main effects plus any significant interactions.

TABLE 1—Risk Factor Comparison in Study vs Non-Study Women

Characteristics	Study[a] Women (n = 2,422)		Non-Study[a] Women (n = 4,798)	
	Number	Percent	Number	Percent
Age (years)				
60–79	1515	62.6	2715	56.6
50–59	907	37.4	2083	43.4
Risk Category[b]				
High	875	36.1	193	4.0
Moderate	1547	63.9	107	2.2
No Increased Risk[c]	0	0	4498	93.8
Family History Breast Cancer				
Yes	886	36.6	499	10.4
No	1536	63.4	4299	89.6
Previous Benign Breast Biopsy				
Yes	541	22.7	383	8.2
No	1841	77.3	4281	91.8
Nulliparity to Age 30				
Yes	1856	35.3	163	3.4
No	1566	64.7	4634	96.6
Menarche Age < 10				
Yes	166	7.0	41	0.9
No	2214	93.0	4661	99.1
Menopause Age ≥ 55				
Yes	427	17.6	128	3.2
No	1995	82.4	3822	96.8

[a]Among GHC women 50 years of age and older who completed and returned a risk factor questionnaire before December 31, 1985 and had not reported having a mammogram within one year. "Study Women" are those women who were invited for screening; "Non-Study Women" represent those who were not invited by December 31, 1985.
[b]There are no "Borderline" risk women in the age group studied (see Appendix).
[c]These women have none of the risk factors listed, except age.

Results

Since the study population was composed of women who were invited for screening because they were at increased risk for breast cancer, each woman necessarily had one or more risk factors for the disease. As expected, a higher proportion of study women had any given risk factor compared to non-study women (Table 1).

Table 2 shows the percentage of study women with each of the six risk factors according to their risk category. As expected, a larger proportion of high-risk women have any particular risk factor.

Of the 2,422 women in the study population, 71 percent came in to the screening center as requested by the letter of invitation. Table 3 shows the crude odds ratios (OR) for the association between breast cancer risk factors and participation in mammography. The strongest association is between the risk category and participation (OR = 2.59), followed by family history (OR = 1.83), previous biopsy (OR = 1.60), and age (OR = 1.45). Age is dichotomized in this bivariate analysis since participation varies little among women above age 60 when grouped into five-year intervals (73 percent to 75 percent). Three factors (age ≥ 55 at menopause, nulliparity to age 30, and age ≤ 10 at menarche) show no association with participation.

Table 4 shows odds ratios estimated from the logistic regression analysis. Since there is a significant interaction between age and risk designation, the final logistic model consists of the six risk factors, the risk designation, and this interaction term. Family history of breast cancer and previous benign breast biopsy continue to be associated with increased participation although the odds ratio is slightly less. Neither nulliparity to age 30 nor menarche at age 10 or

TABLE 2—Proportion of Women in Moderate and High-Risk Categories Who Have a Particular Risk Factor

Risk Factors	Risk Category[a]	
	Moderate (N = 1547)	High (N = 875)
Age 60–79 years	61%[b]	66%
Family History of Breast Cancer[c]	21%	63%
Previous Benign Breast Biopsy	15%	37%
Nulliparity to Age 30	31%	43%
Age ≤ 10 at Menarche	5%	10%
Age ≥ 55 at Menopause	14%	24%

[a]Risk category is the designation given by the screening program according to the algorithm in the Appendix.
[b]For example, 61 percent of moderate-risk women are 60 to 79 years of age compared to 66 percent of high-risk women.
[c]First and second degree family history result in different risk designations (see Appendix).

TABLE 3—Bivariate Analysis of Breast Cancer Risk and Participation in Mammographic Screening

Characteristics	% Participation	Unadjusted Odds Ratio (95% CI)[a]
Age (years)		
60–79	73.3	1.45
50–59	65.7	(1.21, 1.73)
Risk Category		
High	82.2	2.59
Moderate	64.1	(2.12, 3.15)
Family History of Breast Cancer		
Yes	78.2	1.83
No	66.2	(1.52, 2.22)
Previous Benign Breast Biopsy		
Yes	77.6	1.80
No	68.4	(1.50, 2.00)
Nulliparity to Age 30		
Yes	71.3	1.05
No	70.2	(.87, 1.26)
Age ≤ 10 at Menarche		
Yes	68.7	.90
No	70.7	(.64, 1.26)
Age ≥ 55 at Menopause		
Yes	66.4	.84
No	71.4	(.67, 1.05)

[a]95% confidence interval for odds ratio.
[b]Referent group for odds ratio is women without the risk factor.

TABLE 4—Results of Logistic Regression Analysis of Breast Cancer Risk and Participation in Mammographic Screening

Characteristics	Adjusted Odds Ratio[a]	(95% CI)
Family History Breast Cancer	1.35	(1.02, 1.79)
Previous Benign Breast Biopsy	1.36	(1.02, 1.81)
Nulliparity to Age 30	0.97	(0.75, 1.24)
Age ≤ 10 at Menarche	0.82	(0.55, 1.22)
Age ≥ 55 at Menopause	0.73	(0.55, 0.97)
Age and Risk Category[b]		
Moderate Risk Category		
Age 50–59	1.00	
Age 60–79	1.86	(1.49, 2.32)
High Risk Category		
Age 50–59	3.94	(2.61, 5.96)
Age 60–79	3.09	(2.21, 4.31)

[a]Odds ratios estimated from the logistic regression model including all six risk factors and a variable for the interaction between "age" and "risk category." Odds ratios are defined so that those without the characteristic are the reference group.
[b] Odds ratios are defined so that women ages 50–59 who are at "moderate" risk are the reference group.

younger show any association with participation. Controlling for other risk factors in the multivariate model increased the association between late menopause and participation.

The association of age with participation depends on whether the risk label was "moderate" or "high." Increasing age is associated with participation only among women with the "moderate" risk label. For women labeled high-risk, participation is essentially the same or even slightly less among older women (OR = 3.09/3.94 = 0.78).

As seen in the bivariate analysis, the high-risk label shows the strongest association with participation. However, the four odds ratios for the joint factors in Table 4 reveal that the association between the high-risk label and participation was more than two times as strong in younger (OR = 3.94) compared to older women (OR = 3.09/1.86 = 1.66, Table 4).

Discussion

The results of this study substantiate our hypothesis that risk factors recognizable by lay persons such as age, family history, previous benign breast biopsy, and the high-risk label are each positively associated with compliance with a mailed recommendation to obtain a screening mammogram. Since risk factor information was collected prior to screening visits, recall bias was avoided. The prospective observation of the entire population in the course of their usual care also eliminates any Hawthorne effect created in the process of selecting and observing a separate study population.

Performing the study in the context of a formal screening program also creates some potential limitations. First, all candidates for the study were respondents to the screening survey; the relation between risk and participation might be different for non-respondents. However, since 85 percent of women complete the risk factor questionnaire, this potential threat to generalizability is expected to be minimal. Second, as a result of the screening program's design, all study women had at least one risk factor in addition to age. In considering each individual risk factor, the referent group consisted of women without any factor but possessing "other" risk factors. While it is not possible to determine from these data whether the magnitude of the associations might differ substantially if the referent population included women whose only risk factor is age, it is likely that the associations would be stronger.

A final limitation is that the results may be biased due to residual confounding by two factors. The first is socioeconomic status (SES) for which no data were available. Since higher socioeconomic status has been associated with increased risk of breast cancer[15] and with increased participation in screening,[5,7] it must be considered a potential confounder. However, any effect of SES would be expected to be small since GHC is somewhat more homogeneous with respect to SES compared to the surrounding SMSA population.[11] Second, there may be residual confounding due to the presence of breast cancer symptoms. Participants in the HIP study were more likely to report pre-menstrual breast tenderness or having previously felt a breast lump.[5] However, when women call for an appointment at the GHC screening center, the policy is to ask about pain, lumps, or the presence of nipple discharge. Women with any of these symptoms are advised to see their primary care physician, so it is expected that the majority of screened women were asymptomatic. Symptomatic women who called for an appointment and were referred to their physician would have been misclassified as non-participants in this study since the program does not document these calls in its automated data base. This misclassification would weaken the observed associations.

Our data show that family history of breast cancer is significantly associated with participation even after controlling for other risk factors. French's work found that participants were more likely to have a family history of breast cancer than non-participants, but the difference was not statistically significant.[9] In the HIP study, there was no significant difference in "family history of cancer" between women who completed all four screening examinations compared to those who did not.[5] However, that study did not evaluate the relationship between family history of breast cancer and participation.

Our finding of increased participation among older women is not consistent with previous reports which show

associations in the opposite direction.[5–7] However, none of these studies included women beyond 64 years of age and only one was conducted prospectively.[7] The data presented here suggest women 60 to 79 years of age are more likely to participate than women 50 to 59 years of age. While the reason for lower participation in younger women is not clear, one possible explanation is that these women are more likely to be employed and therefore have conflicts during usual screening clinic hours.

Our finding of increased participation in older women is important because more women are living longer and the incidence of breast cancer increases greatly with advancing age. Celentano has pointed out that primary care providers often fail to offer cancer screening procedures to the elderly.[16] Others have suggested that cost is a barrier.[17,18] The relative importance of these two factors is not clear. With the advent of Medicare coverage for mammography every two years, it will be important to observe whether the use of mammography increases among older women in other populations. Our data suggest that senior women will use mammography if it is offered to them, and cost is not an issue.

No other studies to our knowledge have examined the association between participation and the three other risk factors studied here: nulliparity to age 30, early age at menarche, and late age at menopause. Our findings are not completely consistent with our hypothesis that these factors would be unassociated with participation. No associations exist in the bivariate analysis. However, after controlling for all other risk factors in the multivariate model, a negative association appears between late menopause and participation. The explanation for this is not clear, but one consideration is that there is confounding by some other factor such as estrogen use. This relation needs closer examination in future studies.

The increased participation among women with particular risk factors that is demonstrated in this study is consistent with the Health Belief Model[19,20] which suggests that perceived vulnerability to disease should influence the likelihood that a person will undertake preventive behaviors. We hypothesized that women with certain risk factors would be more likely to perceive themselves as vulnerable, and therefore participate in screening. While the increased participation has been demonstrated with our data, we did not directly evaluate women's perception of their vulnerability.

The risk assessment/feedback process used in Group Health Cooperative's Breast Cancer Screening Program is similar to Health Risk Appraisal (HRA) and may be a way to increase the use of mammography. The HRA process provides individuals with quantitative and/or qualitative disease risk estimates based on self-reported risk factors obtained through questionnaires.[21] Proponents of HRA argue that it is an effective strategy for facilitating positive health-related behavior. Our finding that women given a high-risk designation were most likely to participate suggests that this may be true. However, more needs to be learned about the effect of different risk labels, and the influence of these designations on other behaviors. Experience from hypertensive labeling suggests that there are potential negative effects such as increased depressive symptoms, and lower self-perceived health.[22]

In conclusion, overall our study found the expected positive associations between participation and those risk factors that could be easily identified by women. Self-selection for screening by women at increased risk has been identified as a major concern in the non-experimental evaluation of screening.[1–3] Our findings give substance to these concerns but also raise the possibility that informing women of all their risk factors or using a summary risk label may take advantage of the motivation behind self-selection and encourage participation.

APPENDIX
Risk Algorithm*

High-Risk Group	• Previous breast cancer • Mother with breast cancer • Age 50+ and 2 VRF listed below
Moderate-Risk Group	• Age 40–49 and 2 VRF listed below • Over age 50 and 1 VRF listed below
Borderline Risk	• Age 40–49 and 1 VRF listed below
No Increased Risk	• No VRF, any age
Variable Risk Factors (VRF)	• Previous cancer (other than breast) • First or Second degree relative (other than mother) with breast cancer • Menarche age 10 or under • Nulliparity • 1st pregnancy age ≥ 30 • Menopause age ≥ 55 • Previous benign breast disease

*Risk algorithm used in Group Health Cooperative's Breast Cancer Screening Program until April 1988.[13]

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RWJ Foundation Awards Grant to Study Child Abuse Responses in 50 States

The first 50-state survey of child abuse policies and regulations will be launched by the American Bar Association (ABA) with a grant from the nation's largest health care philanthropy. The $280,464 two-year grant has been awarded to the ABA Fund for Justice and Education from the Princeton-based Robert Wood Johnson Foundation. The ABA, in conjunction with the American Academy of Pediatrics, will develop recommendations about policies and procedures related to prevention, intervention, and investigations of child abuse.

Under the direction of Susan J. Wells, PhD, the ABA survey will gather and analyze information from child welfare agencies, physicians, coroners, police officials, prosecutors, and legislators in all 50 states to determine how state and local governments are currently responding to child abuse.

It is estimated that child deaths from abuse have increased 34 percent since 1985, resulting in about 5,000 deaths a year, said Leighton E. Cluff, MD, Foundation president. "But because states don't collect child abuse data uniformly, there is little information to guide policymakers and other officials working to prevent these deaths," he added. Cluff termed the ABA project "an important first step" in addressing the problem and in understanding the effect of interventions intended to prevent child abuse.

In addition to collecting data, the ABA will provide consultation to selected states that have expressed interest in revising their policies regarding child abuse.

The Robert Wood Johnson Foundation was established as a national philanthropy in 1972. Since then, it has awarded more than $996 million in grants to improve health care for such US populations as adolescents, children and mothers, the elderly, the homeless, the mentally ill, people with AIDS, and the uninsured. For further information, contact the RWJ Foundation, PO Box 2316, Princeton, NJ 08543-2316. Tel: (609) 452-8701.

Breast Cancer Screening by Mammography: Utilization and Associated Factors

JANE G. ZAPKA, ScD, ANNE M. STODDARD, ScD, MARY E. COSTANZA, MD, AND HARRY L. GREENE, MD

Abstract: The status of mammography screening experience and factors related to utilization were examined in six towns serviced by physician staffs at five hospitals. Data were collected via random digit dial telephone interview of a probability sample of 1184 women, aged 45–75 years. The results showed that 55% of the women reported ever having had a mammogram. Of those who had ever had a mammogram, 21% reported that the mammogram in the past year was their first one. Of those women who are over 50 and had ever had a mammogram, 57% reported one in the past year. Analyses demonstrated that a combination of demographic factors, certain beliefs and knowledge, having a regular physician, social interaction and media exposure are independently related to ever having a mammogram, and to having one in the past year. Despite anecdotal and empirical evidence that the proportion of women ever having had a mammogram has substantially increased in the past several years, increasing utilization among older and lower-income women provides a challenge for public health (*Am J Public Health* 1989; 79:1499–1502.)

Introduction

Among women, breast cancer leads all other cancers (28% in women), and is the second leading cause of cancer death.[1] Reductions in mortality from breast cancer depend on successful interventions aimed at early detection and treatment,[2–4] but the adoption of screening technologies by physicians and women has been slow.[5–7] However, there are recent indications that the utilization of screening technologies, notably mammography, has been increasing.[8] Targeted interventions are being called for by medical and public health authorities.[4,5] This study was undertaken to characterize the breast cancer screening experience, knowledge, and attitudes of a probability sample of urban women so as to provide a valid base for planning community interventions aimed at increasing the appropriate use of screening technologies.

Methods

A survey was conducted using the Waksburg random digit dial method[9] in two urban areas of eastern Massachusetts comprising six towns serviced by physician staffs of five community hospitals. A pretested structured 20-minute telephone interview was administered in late 1987. A woman was eligible for the study if she was between 45 and 75 years of age and had never had breast cancer. A minimum of 10 call-backs were made as needed to secure an interview, and a refusal conversion process was included. A response rate of 75.3 percent was achieved, yielding a sample of 1,184 women.

In addition to sociodemographic and health history items, the survey included items to measure knowledge (such as risk factor and incidence knowledge and knowledge that mammography is appropriate in the absence of symptoms), beliefs (such as perception of barriers to and benefits of having a mammogram, and susceptibility to breast cancer), enabling factors (insurance coverage, having a regular physician and usual location of care) and factors which reinforce or hinder screening behavior (encouragement to have screening by family, friends, and providers, discussion about screening with social network members and exposure to media messages about breast cancer screening). Summary variables were computed for items purported to measure personal and social factors and tests of reliability yielded Cronbach's alpha coefficients in the range of .56 to .71.*

We first looked at variables associated with ever having a mammogram in the total sample. Among women who had a previous mammogram, we subsequently looked at correlates of having a mammogram in the last year for those aged 50 and over. While recent study results suggest reduction in mortality from breast cancer with mammography in younger women,[10] the issue remains controversial. Therefore, women under age 50 were removed from this analysis since current guidelines do not consistently recommend annual screening for women under age 50.

For this exploratory study, forward stepwise logistic regression analysis[11] was used to select from among those bivariately associated with screening, a set of variables representing independent correlates of screening behavior. After main effect variables were entered into the model, selected two-way and higher order interactions were considered. The entry criterion was a chi-square p value of 0.05. Some interactions could not be tested due to sparse cells in the relevant multiway cross classifications.

Results

Selected characteristics of the sample are presented in Table 1. Fifty-five percent of the 1184 women surveyed reported ever having a mammogram. Among the 45 percent who had never had a mammogram, 10 percent had never heard of mammography. Twenty-five percent of the total sample reported previous medical problems with their breasts and these women were significantly more likely to have had multiple mammograms than those who reported never having had a problem. However, the majority of women (82 percent) reported having had their mammogram as a "check-up, for no particular problem." Women who had less than a high school education, or were Catholic or

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*Details on the survey items, factor analyses, and development of summary variables are available from the authors.

TABLE 1—Percent (and frequency) of Reported Breast Cancer Screening Behaviors by Selected Characteristics[a]

Characteristics	Total Sample n = 1184	Ever Had a Mammogram n = 650	Mammogram in Past Year[b] n = 290
Age (years)			
45–49	222	56.8 (126)	— —
50–54	224	60.7 (136)	32.7 (73)
55–59	200	61.0 (122)	30.0 (60)
60–64	197	55.3 (109)	33.5 (66)
65–69	165	45.0 (75)	27.9 (46)
70–75	152	46.7 (71)	29.6 (45)
Education			
Less than high school	215	38.6 (83)	21.5 (41)
High school graduate	513	55.8 (286)	29.3 (125)
Some college	255	59.2 (151)	35.8 (72)
College graduate	192	65.1 (125)	43.7 (52)
Employment			
Currently employed	598	57.3 (343)	31.0 (130)
Full-time homemaker	192	56.3 (108)	30.8 (49)
Retired	286	51.1 (146)	33.3 (93)
Other	100	48.0 (48)	22.0 (18)
Income			
Less than $15,000	333	40.5 (135)	22.9 (69)
$15,001–25,000	201	57.2 (115)	28.6 (48)
$25,001–35,000	163	58.3 (95)	32.0 (40)
More than $35,000	320	66.3 (212)	42.9 (90)
Religion			
Catholic	754	51.7 (390)	27.8 (167)
Protestant	283	56.9 (161)	31.4 (74)
Jewish	83	84.3 (70)	63.3 (38)
Marital status			
Married/living with	791	57.7 (457)	32.9 (203)
Separated/divorced	130	51.5 (67)	25.0 (22)
Widowed	211	46.9 (99)	25.8 (51)
Never married	43	48.8 (21)	42.4 (14)
Ever had medical problem with breast			
Yes	292	83.6 (244)	43.6 (98)
No	892	45.5 (406)	27.0 (192)

[a]Frequencies may not sum to total sample due to missing data in respective cross-tabulations.
[b]For women over 50 years of age.

Protestant (as compared to Jewish), were significantly less likely to have ever had a mammogram or had one in the past year. Income was directly related to ever having a mammogram; education and employment were highly correlated with income for the 86 percent of subjects who responded to the income question.

Given that getting a mammogram requires interaction with the medical care system, we were particularly interested in several variables related to sources and perceptions of medical care as well as insurance variables. Eighty-five percent of women reported that they had a regular physician. A majority (74 percent) of those who had a mammogram had had a clinical breast examination in the last year vs 44 percent of those who had never had a mammogram. Seven percent of the sample did not have any level of health insurance or entitlements. Seventy-one percent of those who ever had a mammogram said their insurance paid for it, 10 percent paid for it themselves, and 16 percent reported some combination.

Women who had never had a mammogram were asked why. Forty-five percent responded that they did not feel it was necessary and/or that they felt healthy, and 24 percent said their doctor had never recommended it. All women were asked if they would have a mammogram in the next year if their doctor recommended it, and 83 percent said they were very certain they would.

Factors Associated with Mammogram Experience

Table 2 presents the results of the main effects logistic regression analysis on ever having had a mammogram. There were significant negative interactions (data not shown) between being encouraged by a friend to have a mammogram and disagreeing with the statement that symptoms should precede a mammogram, and between being encouraged by a friend and discussing mammography with a friend. These interactions indicate that once one event has occurred there is no additional association with the other event. Variables which showed bivariate associations with ever having had a mammogram but which did not enter the model include knowledge of breast cancer risk factors, perceptions of susceptibility to breast cancer, perception of barriers to mammography, a belief that other health problems have priority, and education attainment. Women whose regular physician was an internist or gynecologist were more likely to report ever having a mammogram than women whose physician was a general or family practitioner (68 percent vs 41 percent); the latter women were no more likely to have had a mammogram than women with no regular doctor.

The women age 50 and over who had ever had a mammogram were divided into those who had one within the past year and those who had not. The main effects logistic regression model for this comparison is given in Table 3. Although having a mother or sister with breast cancer was positively associated with ever having a mammogram, having another relative (e.g., aunt) with breast cancer was negatively associated with having one in the last year. Variables which showed bivariate associations with mammography in the last year but which did not enter the model include a belief that mammography can detect cancer, history of breast problems, and employment status.

We were interested in the impact of insurance coverage on mammogram experience. Reliable and valid information on coverage was limited. A substantial number of subjects reported having more than one type of coverage. Since it was not possible to determine which plan provided coverage,

TABLE 2—Factors Associated with Ever Having a Mammogram: Main Effects Logistic Model (n = 840)

Variables	B	Odds Ratio	(95% CI)
Sociodemographic			
Income	—		
$15001–25000	.54	1.72	(1.08, 2.75)
$25001–35000	.03	1.03	(0.62, 1.70)
$35001+	.43	1.53	(1.00, 2.35)
Religion			
Catholic	−.09	0.91	(0.63, 1.33)
Jewish	1.14	3.14	(1.30, 7.60)
Health History			
History of breast problems (yes)	1.69	5.40	(3.47, 8.41)
Mother/sister with breast cancer (yes)	.95	2.59	(1.43, 4.68)
Knowledge			
Need symptom (disagree)	.76	2.15	(1.75, 2.63)
Attitude/Belief			
Benefits of mammography (agree)	.16	1.17	(1.08, 1.28)
Enabling			
Have a regular MD (yes)	.75	2.13	(1.30, 3.48)
Reinforcing			
Mammogram encouraged by a friend (yes)	.62	1.85	(1.20, 2.87)
Discuss mammography with friends (yes)	.71	2.04	(1.43, 2.89)
Constant	−2.38		

Hosmer-Lemeshow Goodness of fit: Chi-square = 5.78, df = 8, p = .67.

TABLE 3—Factors Associated with Having Had a Mammogram within the Past Year for Women Over Age 50: Main Effects Logistic Model (n = 346)

Variables	B	Odds Ratio	(95% CI)
Sociodemographic			
Income			
$15001–25000	−.67	0.51	(0.25, 1.06)
$25001–35000	−.34	0.71	(0.32, 1.61)
$35001+	.56	1.75	(0.78, 3.95)
Marital			
Separated/divorced	.41	1.50	(0.60, 3.79)
Widowed	−.70	0.50	(0.23, 1.08)
Never married	2.14	8.46	(1.25, 57.28)
Age (years)			
60–69	.99	2.69	(1.48, 4.86)
70–75	1.65	5.22	(1.88, 14.45)
Health History			
Mother/sister with breast cancer (yes)	.88	2.40	(1.06, 5.44)
Other blood relatives had breast cancer (yes)	−1.01	0.36	(0.17, 0.78)
Reason for mammogram (routine)	.84	2.31	(1.22, 4.38)
Knowledge			
Need symptom (disagree)	.37	1.45	(1.03, 2.04)
Know cost (yes)	.84	2.31	(1.09, 4.91)
Attitude/Belief			
Most women over 50 get a mammogram (agree)	.37	1.45	(1.09, 1.91)
Enabling			
Have regular MD (yes)	1.10	3.02	(1.09, 8.39)
Mammogram satisfaction (high)	.16	1.17	(1.01, 1.36)
Reinforcing			
Media exposure (greater)	.92	2.51	(1.11, 5.72)
Constant	−2.32		

Hosmer-Lemeshow Goodness of fit: Chi-square = 6.14 df = 8, p = .63.

women with more than one type of coverage were excluded and the screening behavior of those women who reported only one type of coverage was further examined. Given the possibility that a person's insurance plan changes over the years, analysis was limited only to mammogram experience in the past year. Bivariate analysis demonstrated that 34 percent of women who had commercial insurance, health maintenance organization (HMO) or Medicare plus commercial insurance had a mammogram in the past year compared to 15 percent of women covered by Medicare only, Medicaid only, or no insurance. For this set of subjects, the insurance variable did not significantly improve the multiple logistic model.

Discussion

The findings of this study support anecdotal and empirical evidence that the proportion of women ever having a mammogram has substantially increased in the last several years, particularly among White, middle and upper socioeconomic level, and younger women.[5,8,12] Further, we found that although a greater proportion of women over age 60 have never had a mammogram, among those who have ever had one, more older women than younger women did get one in the past year. A summary of data from 33 states[13] reported that 29 percent of US women 50 years of age or older reported having had a screening mammogram in the last year. The level of screening varies considerably by state, however, with 37 percent reported in Massachusetts.[14] For our sample of women ages 50–75 years, 31 percent reported a mammogram in the past year.

It is very difficult to determine the extent to which women have had mammographic examinations for screening rather than diagnostic purposes.[5] Our data demonstrate a positive correlation between previous breast problems and having had one mammogram or having multiple mammograms. However, 82 percent of the women reported that their most recent mammogram was for "check-up" purposes, not for a particular problem. Additionally, there is uncertainty in estimating the proportion of women who have been screened with mammography at some time in their lives as compared with the proportion who are routinely screened according to recommended age/frequency guidelines. Defining the reasons for mammography and documenting the prevalence of *regular* screening should be priority questions for prospective research on screening utilization.

The regression models reported here must be interpreted with caution. Stepwise forward selection of variables was used as a means of screening many bivariately associated variables to select those independently associated with screening behavior. Although this approach is suitable for screening it does not necessarily result in valid estimates of effect size.[15] The results of this analysis may be used to develop specific models which can then be explicitly estimated and tested in another data set.

Perception of barriers to mammography (exposure to radiation, pain, cost, and embarrassment) and to breast cancer screening have been suggested by theoretical models[16] and emphasized in previous studies.[17,18] Interestingly, none were significantly related to mammography behavior. Neither perception of severity, nor risk factor knowledge were associated. In contrast to the limited relationship of knowledge and belief factors, importance of having a regular physician, particularly a gynecologist and/or internist, is highlighted in this research.

Although the findings of this study cannot be generalized to minority or rural populations, they do highlight a particular challenge for public health to increase screening utilization behavior in older and lower-income women. Targets for education include their clinicians who need to initiate more effective discussion of screening requirements, motivation of public policy-makers to include coverage of screening mammograms to deal with any cost/access barrier, as well as education of women about the need to attend to breast health.

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Editor Search Committee Seeks New Editor for American Journal of Public Health

An Editor Search Committee is actively recruiting a replacement for Dr. Alfred Yankauer who has announced his retirement as Editor of this *Journal*, effective July 1, 1990. The six-member search committee welcomes and solicits nominations and recommendations from the APHA membership and leadership to assist them in identifying the best available individual for this important salaried position. Nominations should be submitted as soon as possible, as the committee will meet several times to consider suggestions, applications, and support materials of potential candidates.

Names of potential candidates, along with letters of endorsement and other support materials, should be sent to: Editor Search Committee, American Public Health Association, 1015 15th St., NW, Washington, DC 20005.

The following criteria will be considered by the search committee in selecting the Journal editor:

- Comprehensive knowledge and broad perspective of the field of public health, with an appreciative understanding of its many disciplines and solid grounding in the basic sciences of epidemiology and statistics;
- Professional accomplishment and identity with the public health field, including respected standing among peers, combined with an extensive network of professional contacts who will provide expertise in soliciting and evaluating materials for publication;
- Demonstrated research skills, with evidence (such as credited publication in peer review journals) of firm grounding in a field of scientific inquiry within public health;
- Demonstrated writing, reviewing, and editing skills, enabling authoritative advice to authors on the suitability of prepared manuscripts, facilitating informed consideration of reviewer assessments, and equipping the preparation of appropriate editorials as needed;
- Freedom to devote substantial time to editor duties—as much as needed, presumably at least half time;
- Working knowledge of APHA and sympathy to its advocacy goals;
- Easy access to Washington, DC;
- An academic base is deemed highly desirable, preferably in a school of public health or a university health sciences center.

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Nulliparity, Decade of First Birth, and Breast Cancer in Connecticut Cohorts, 1855 to 1945: An Ecological Study

ROBERT A. HAHN, PHD, MPH, AND SURESH H. MOOLGAVKAR, MD, PHD

Abstract: Risk of breast cancer increases with age at first birth, and is lower in women who bear their first children while young than in nulliparous women. While previous studies have investigated risk of breast cancer in birth cohorts by examining partial aspects of cohort childbearing, the present ecological study assesses total cohort childbearing risk in Connecticut women born between 1855 and 1945. In each cohort, the proportion of women nulliparous and first bearing children in their twenties, thirties, and forties are weighted by relative risks associated with these events as ascertained in previous studies. Summed cohort childbearing risks are compared to the incidence of breast cancer in women 40 years of age and older in the same cohorts. Changes in decade of first birth and nulliparity do not explain the changes in breast cancer incidence observed: while cohort childbearing risk has declined over the period examined, breast cancer incidence has increased in the same cohorts. Alternative explanations for cohort increases in breast cancer incidence are reviewed. (*Am J Public Health* 1989; 79:1503–1507.)

Introduction

In the United States, Europe, and elsewhere, breast cancer incidence and mortality reached a low point in women born at the turn of the twentieth century and have been rising steadily in cohorts born thereafter.[1,2] The predominance of cohort effects in accounting for these secular changes in breast cancer incidence and mortality was noted early by MacMahon,[3] and has been well established by the modeling methods of indirect standardization.[1,2,4,5] MacMahon hypothesized that these cohortwise changes in incidence and mortality might correspond to changing patterns of childbearing.[3]

With several exceptions,[6–8] most case-control studies[9–13] have demonstrated that a woman's risk of breast cancer increases with the age at which she completes her first full term pregnancy. The risk of breast cancer in women first bearing a child before age 18 is estimated to be one-third that of women first bearing after age 34. Nulliparity is associated with greater risk of breast cancer than bearing a first child up to age 34, after which the risk associated with first births exceeds that of nulliparity.[13] A large retrospective cohort study[14] further supports these results.

Several studies[7,15–20] have examined the association of changing population childbearing patterns with changing patterns of breast cancer incidence and mortality. Using summary vital records from England and Wales, Connecticut, and the United States, MacMahon[15] examined the association of cohort fertility and breast cancer incidence and mortality; findings contradicted the hypothesis that fertility was inversely associated with breast cancer rates. In contrast, Armstrong[16] examined proportions of women in the US bearing first children by certain ages and live birth rates by certain ages in England and Wales; he found that childbearing trends accounted at least in part for the cohort variation in breast cancer incidence (in Connecticut) and mortality (in the US and in England and Wales). Likewise, Blot[17] found parallels between proportions of US women nulliparous between ages 20–24 and breast cancer mortality in the same cohorts. Wigle[18] also demonstrated associations between marital and fertility characteristics of Canadian women and their rates of breast cancer incidence and mortality. On the other hand, based on correlations among rates of breast cancer mortality among single and married women in nine nations and regions within them, Hems and Stuart[19] claimed that international differences in breast cancer mortality were associated with diet but not with fertility. Most recently, White[20] predicted that delayed childbearing in cohorts of women born since 1945 will yield breast cancer incidence significantly greater than that of preceding cohorts, in which early childbearing was more common.

Other than White's predictive analysis, these studies have shortcomings of two sorts. Some[7,15] have examined risk factors tangential to those now recognized to be associated with rates of breast cancer, for example, cohort fertility by older ages, and proportions married at 20–24 and at 40–44 years. Further, all of these studies examine one or several aspects of cohort childbearing events, e.g., nulliparity between ages 20 and 24, first birth by certain ages, but not the overall cohort history of childbearing. Investigation of isolated elements of cohort childbearing may mask other cohort childbearing risk factors for breast cancer which either support or oppose the particular cohort risk factor investigated. For example, while one cohort may have apparently greater risk than another cohort because of high nulliparity in ages 20–24, it may also have lower risk than the other cohort because of a high rate of first births to women younger than 20 years. Apparent associations and non-associations between partial cohort childbearing events and breast cancer outcomes may thus be misleading.

The present study used vital records and Connecticut Tumor Registry data to examine the association between total cohort childbearing history—nulliparity and decade of first birth—and breast cancer incidence in the population of Connecticut women born between 1855 and 1945.

Methods

Information on cohort childbearing events was abstracted from the Connecticut Vital Statistics Report (1879–1945),[21] Vital Statistics of the United States (1946–1982),[22] and the US Census of Population (1880–1980).[23] Information on breast cancer incidence in Connecticut women, ages 20 to 79 years, from 1935 to 1982 was provided by the Connecticut Tumor Registry,[24] as reported in Stevens, *et al*,[2] and updated.

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Cohort Decades of Firth Birth and Nulliparity

Because Connecticut Vital Statistics Reports present information on women bearing their first children in time intervals of varying length, but commonly in 10-year age categories, e.g., between ages 20 and 29, the present investigation is constrained to a comparison of decades of first birth. Numbers of women bearing first children in each year were aggregated into 10-year groupings centered on decade years (in which Census data were gathered), e.g., 1885–1894 (with decade mid-year 1890), 1895–1940 (mid-year 1900), etc. Since Connecticut Vital Statistics Reports were first published only in 1879, information on first births at different ages in the 1880 "mid-year" was aggregated from 1879 to 1884.

Proportions of women bearing first children in their second, third, fourth, and fifth decades were calculated by dividing numbers of first births in each age group by the population of that group. Since vital registry data on first births for the first Census period (1880) were available only for six of the 10 years in one interval (1879–1984), the proportions of 2nd to 5th decade first births for this period were estimated by dividing the numbers of births by 6/10 of the appropriate Census figures for each age group.

Resultant proportions were reorganized according to the year of birth of the mothers. Thus, proportions of women who bore a first child in their second decade (i.e., ages 10–19) between 1879 and 1884 were associated with those who first bore in their third decades (i.e., 20–29) between 1885 and 1894, and with those who first bore in their fourth decades (i.e., 30–39) between 1895 and 1904, and so on; all of these women were born between 1855 and 1874, a 20-year span referred to by its mid-year as the cohort of 1865.

While a few women were reported to bear first children after age 50, for the purposes of this analysis it was assumed that a women who had not borne a first child by age 50 was nulliparous. Cohort nulliparity was calculated by subtracting from *one* the sum of proportions of cohort women who had borne a first child in their second through fifth decades. That is,

$$\text{Cohort Nulliparity} = 1 - (FB_2 + FB_3 + FB_4 + FB_5),$$

where FB_n = proportion of cohort women bearing a first child in their n^{th} decade.

Cohort Childbearing Breast Cancer Risk

Cohort proportions of women nulliparous or first bearing children in different decades were weighted by the relative risks ascribed to these childbearing events by the multination case-control study of MacMahon, *et al*:[10]

Age/First Birth:	Nulliparous	<20	20–24	25–29	30–34	>34
Risk	1.0	.48	.59	.76	.91	1.10

In order to make the relative risks of five-year age groups from the multi-nation[10] study commensurate with the decades of first birth in the present study, a mean of relative risks within decades was taken to estimate the relative risk for each decade. Since women older than 40 contributed less than 0.5 percent of cohort first births, their contribution to childbearing risk was no longer considered. The total nulliparity and first birth decade risk of breast cancer in cohorts was then estimated by summing the relative risks specific to each childbearing category, weighted by the proportions of each childbearing category in the cohort. That is,

$$\text{Childbearing Breast Cancer Risk} = FB_2 \times RR_2 + FB_3 + FB_4 \times RR_4 + \text{Proportion Nulliparous} \times RR_{\text{Nulliparity}},$$

where, as above,

FB_n = the proportion of cohort women bearing a first child in their n^{th} decade,

and where,

RR_n = the risk of breast cancer given this event, relative to nulliparity.

Breast Cancer Incidence and Standardized Incidence Ratios

Since the first birth "exposures" considered were those occurring to cohort females by age 40, only breast cancer incidence after this age was analyzed as a possible outcome. Incidence data for women older than 40 years were grouped into 10-year age categories, and arrayed by cohort to parallel available information on childbearing exposures. The number of cases divided by the corresponding sum of woman-years at risk gave the incidence of breast cancer in this group during this period.

To compare cohort experience of breast cancer incidence and changes of breast cancer incidence between cohorts, we computed an aggregate incidence measure for the cohort. Since age-specific data are missing in the youngest cohorts, the usual methods of direct and indirect standardization do not apply. Following the method of Breslow and Day,[1] we computed an internally standardized incidence ratio for each cohort. The method assumes that age effects are constant in all cohorts, and cohort effects constant for each age; both parameters are estimated for all ages and all cohorts by assuming their constancy in cells for which no data are available. It is assumed that the populations are large, and that disease events are rare and well characterized by the Poisson distribution. Further, the expected number of events (incident cases of breast cancer) in age group i and birth cohort j is assumed to be $E_{ij} = N_{ij} \times a_i \times b_j$, where N_{ij} is the population (woman years) at risk in age group i and cohort j, a_i is the effect of age group i, and b_j is the effect of birth cohort j. The parameter, b_j, can be thought of as an internally standardized incidence ratio. Maximum likelihood procedures are then used to estimate the parameters of the model. This method yields a summary incidence ratio for cohort data derived from cross-sectional studies.

Results

Cohort Childbearing Breast Cancer Risk

For all cohorts of Connecticut females born between mid-years 1865 and 1935, the largest proportion of women have borne a first child in their third decade; the second largest proportion are nulliparous, except for women born in mid-year 1935; the smallest proportions have borne first children in their 2nd or 4th decade (Table 1). Together, cohort proportions of women either nulliparous or first bearing children in their third decades account for between 72 percent and 83 percent of cohort women. Predominant cohort trends are the large, though irregular, increase in the proportion of women bearing their first children in their twenties and the correspondingly large decline in nulliparity.

Weighting of cohort childbearing proportions by the corresponding relative risks yielded a cohort childbearing breast cancer risk profile (Figure 1) indicating: a general declining trend from earlier to later cohorts, interrupted by a small increase from the cohort of 1865 to that of 1875, and a larger increase from the cohort of mid-year 1895 to that of 1905. These risk estimates correspond mainly to changing proportions of third decade first births and nulliparity. Similar

TABLE 1—Proportions of Connecticut Women Nulliparous and First Childbearing in Different Decades (2nd–4th) by Birth Cohort Mid-Year, 1865–1935

Cohort Mid-Year of Birth	Nulliparas	Woman's Age at First Childbirth		
		10–19	20–29	30–39
1865	0.33	0.11	0.46	0.09
1875	0.36	0.10	0.44	0.09
1885	0.28	0.10	0.51	0.10
1895	0.26	0.12	0.53	0.08
1905	0.30	0.13	0.43	0.14
1915	0.21	0.09	0.55	0.15
1925	0.13	0.09	0.70	0.08
1935	0.10	0.14	0.70	0.06

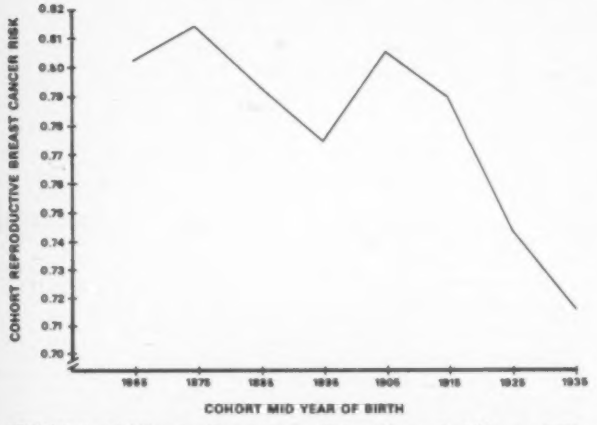

FIGURE 1—Childbearing Breast Cancer Risk in Connecticut Women, 1855–1945

breast cancer childbearing risk profiles (not shown here) derive from application of the relative risks from the large cohort study of Tulinius, *et al,*[14] and from the meta-analysis of White.[20]

Breast Cancer Incidence and Age-Adjusted Incidence Ratios

Incidence among women age 40 and older has increased steadily, with possible interruption in the cohort of 1885 for 70 year old women, of 1895 for 60 year old women, and of 1935 for 40 year old women (Figure 2). (The study of Stevens, *et al,*[2] indicated a trough in the standardized incidence ratio between Connecticut cohorts of 1885 and 1895 and a possible decline following the cohort of 1925.)

The standardized incidence ratio in women 40 years and older showed a general increase from the earliest to the last cohorts (Figure 3). Only from the cohort of 1905 to that of 1915 was there a slight decline in the incidence ratio, followed again by an increase—a more gradual increase than that preceding the decline. Points of inflection showed a rather different pattern from Figure 11 in Stevens, *et al,*[2] principally because Stevens' model incorporated year of event (that is, the interaction between age and cohort). In addition, whereas Stevens, *et al,* considered women 20 years and older in five-year age and cross-section categories, we considered only women 40 years and older in 10-year age categories and 10-year cross-sections. Our study also includes three years of more recent data.

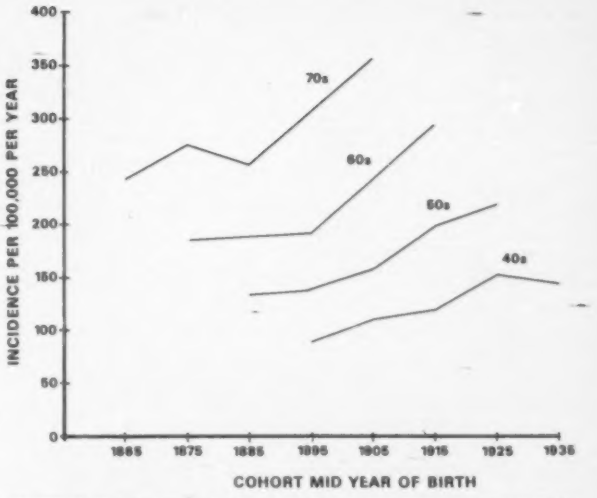

FIGURE 2—Breast Cancer Incidence in Connecticut Women Ages 40–79, by Mid-Cohort Year of Birth

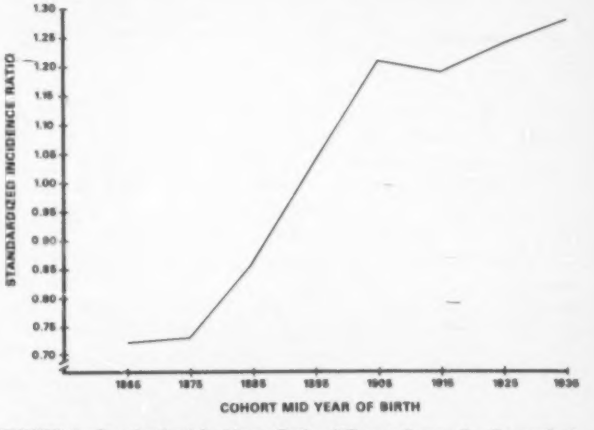

FIGURE 3—Standardized Incidence Ratio of Breast Cancer for Connecticut Women, 40 Years, by Mid-Year of Birth Cohort

Childbearing Breast Cancer Risk and the Standardized Incidence Ratio

Comparison of cohort childbearing breast cancer risk (Figure 1) and the standardized breast cancer incidence ratio for the same cohorts (Figure 3) indicated clearly opposing trends. While childbearing proportions of cohorts would have predicted a general fall in incidence, a generally increasing trend was observed in the standardized incidence ratio. Indeed, higher predicted childbearing breast cancer risk was associated with lower actual incidence ratios, and vice versa.

Comparison of childbearing breast cancer risk (Figure 1) with age-specific incidence rates (Figure 2) also showed a general opposition of trends.

To review the comparisons made in prior population based studies of this issue[7,15–20] with evidence from the present data, we compared the standardized incidence ratio with each of the childbearing proportions—nulliparity, 2nd, 3rd, and 4th decade first births. Discernible relations were contrary to those hypothesized. For example, the proportion of 4th decade first births was unrelated to the standardized

incidence ratio, and the proportion of nulliparas was inversely related.

Discussion

This ecological study examines the association of basic reproductive events—childbearing and decades of first birth—with subsequent risk of breast cancer for all Connecticut women born between 1855 and 1945. Predicted changes are contrary to those observed. While changing childbearing patterns predict a general decline of breast cancer incidence from the cohort of 1865 to that of 1935, the standardized breast cancer incidence ratio generally increases.

These findings are consistent with the large population-based cohort study of Tulinius, *et al*,[14] in which nulliparity and age at first birth were significantly associated with breast cancer incidence *within* cohorts, but predicted incidence changes *between* cohorts contrary to those observed. It may be noted that international differences in childbearing patterns, e.g., nulliparity and early first births, likewise cannot explain the large international differences in breast cancer incidence or mortality, for example, the 4- to 5-fold differences between the US and Japan.[25,26] In fact, breast cancer mortality among US males is also roughly four times that of Japanese males, suggesting that factors unrelated to gender and childbearing history are important in determining international differences.[27]

The conclusions of the present study do not contradict the association of nulliparity and age at first birth with breast cancer incidence. They suggest, however, that changes in breast cancer incidence among cohorts cannot be explained on the basis of changes in cohort childbearing. Cohortwise increases in breast cancer incidence, not only in Connecticut but elsewhere also,[2] have not been accounted for.

Several other recognized risk factors merit further consideration.

Age at Menarche and at Menopause: Early menarche and late menopause are recognized risk factors for breast cancer.[13] While age at menopause has remained relatively constant over the last century,[28] age at menarche has declined at a rate estimated at two months per decade,[29] thus suggesting an increase in breast cancer incidence.

Oophorectomy: Oophorectomy has been shown to be associated with subsequent decreased risk of breast cancer.[13] Armstrong[16] and others have attempted to show a negative association between rates of hysterectomy and breast cancer incidence. This comparison is misleading since it is not hysterectomy, but associated oophorectomy which affects breast cancer risk reduction. Changing rates of oophorectomy have not been examined for periods prior to 1960,[30] but this operation is probably rare and relatively recent, and thus is unlikely to be associated with the breast cancer changes considered here.

Oral Contraceptive Use: The effect of oral contraceptive use on subsequent breast cancer is a matter of controversy. Pike, *et al*,[31] have found that oral contraceptive use prior to first full term pregnancy increases the risk of breast cancer in young women (younger than 32 years). Vessey, *et al*,[32] and others[33] find no such effect. Despite their current prevalence, the recency of oral contraceptives precludes their association with the secular changes considered here.

First Trimester Abortion: The association of abortions prior to first full term pregnancy with increased risk of breast cancer in young women is also a matter of controversy[32,33] Documentation of abortion prior to the 1973 Supreme Court decision is problematic, but this procedure has probably become widespread only recently, and thus again is unlikely to be associated with the changes considered here.

Multiparity: While multiparity and age at birth beyond the first have been found to have less effect on breast cancer than age at first birth,[10,13] studies of high parity populations indicate a strong effect.[14,34,35] In the US, rates of parity of two or more have generally increased since 1900,[36] suggesting a decline in breast cancer rates.

Lactation: While the international studies of MacMahon, *et al*,[37] had shown that, after controlling for parity, the effects of lactation in reducing breast cancer risk disappeared, recent studies again indicate an association, but principally in premenopausal women.[38,39] It is not clear how long-term secular changes in lactation could be assessed.[40]

Diet and Body Weight: Diet and one of its correlates, body weight, have been associated with international differences and secular changes in breast cancer rates.[7,41] Historical records of national and per capita consumption of different food products may allow exploration of the association of nutritional changes and changes in breast cancer incidence.

Electricity and Uninterrupted Light: There is experimental evidence in the rat that exposure to electro-magnetic fields or light reduces pineal production of melatonin; in turn; decreased production of melatonin leads to increased ovarian estrogen and pituitary prolactin production. Stevens[42] has hypothesized that increased exposure to light and electromagnetic fields may increase the risk of breast cancer in humans. Thus, it is interesting to speculate that increasing electrification may be responsible for some part of the observed secular trend. However, it is highly unlikely that the effect of electrification would be seen as changes in risk by cohort.

Secular trends and international differences in this common disease remain largely unexplained. The present study provides further evidence that prominent childbearing events in cohorts predict changes in cohort breast cancer contrary to those which have occurred. Other risk factors must counteract these cohort childbearing trends. Efforts should be made to identify modifiable risk factors which might explain cohort increases in breast cancer incidence.

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NH Law Center Announces Plan to Publish Interdisciplinary Journal

Franklin Pierce Law Center is launching a new interdisciplinary quarterly journal, *Risk: Issues in Health & Safety*. Manuscripts dealing with *basic* problems in health and safety management are being sought for future issues. Of particular interest are research reports or essays addressing strategies for reducing net risk or focusing on the normative issues involved in determining the acceptability of risk—particularly ones spanning traditional categories of occupational, environmental, medical, and consumer injury.

Comments, book reviews, announcements concerning events of interest to a broad interdisciplinary audience, and letters are also welcome.

Those who wish to receive a complimentary copy of the first issue, to be published in late fall or early winter of 1989, may write to Carol Ruh, Franklin Pierce Law Center, 2 White Street, Concord, NH 03301. Annual institutional subscriptions are $50; individual subscriptions, $25.

Thomas G. Field, Jr., JD, Professor of Law and founding member of the faculty of Franklin Pierce Law Center, will serve as editor of the new journal. Those interested in submitting a manuscript may send them to him at the Law Center address, or telephone (603) 228-1541 for further information. FAX Telecopier: (603) 224-3342. The Law Center also publishes *Idea: The Journal of Law and Technology*.

Late-Stage Diagnosis of Breast Cancer in Women of Lower Socioeconomic Status: Public Health Implications

THOMAS A. FARLEY, MD, AND JOHN T. FLANNERY, BS

Abstract: To assess the success of breast cancer control activities in Connecticut, we examined data from the Connecticut Tumor Registry, determining differences in breast cancer stage at time of diagnosis over time and in selected subgroups. From 1982 to 1985, the percentage of women with cancer confined to the breast increased from 54.0 percent to 61.3 percent. During 1984 and 1985, lower socioeconomic status (SES) women with breast cancer were less likely than higher SES women to be diagnosed with early-stage disease (56.9 percent vs 62.7 percent). SES was estimated by census tract of residence. In the same years, the overall incidence of breast cancer was greater in higher SES women. Projections based on these incidence data found that lower SES women, as compared to higher SES women, had a higher rate of expected breast cancer deaths (24.6 vs 19.7 per 100,000), and a greater percentage of those deaths considered preventable by early detection (22 percent vs 11 percent). The rate of preventable deaths in lower SES women was 2.5 times as great as that for higher SES women (5.3 vs 2.1 per 100,000). Tumor registries can serve as useful surveillance systems to aid cancer control programs. Breast cancer early-detection programs should give special attention to lower SES women. (*Am J Public Health* 1989; 79:1508–1512.)

Introduction

The long-term survival of women with breast cancer is dependent on the stage of disease at the time of diagnosis.[1] Attempts to control breast cancer deaths have therefore relied on promoting early cancer detection and treatment. Prospective studies have demonstrated the success of screening programs in both detecting cancers in early stages and decreasing breast cancer mortality.[2–5] However, these studies have been in groups of women enrolled in health maintenance organizations or recruited for special projects. In Connecticut, women have primarily been encouraged to have breast cancer screening by their individual physicians and through educational programs of the American Cancer Society. The extent to which these efforts have been successful in the general population of the state is unclear.

The success of the breast cancer control programs should be measurable initially as an improvement in the percentage of all breast cancers diagnosed at an early stage and ultimately as a decrease in the incidence of late-stage disease. Tumor registries regularly contain information on cancer stage, and can consequently serve as surveillance systems to guide cancer control activities. We examined recent trends in breast cancer stage at the time of diagnosis in the Connecticut Tumor Registry. We then evaluated cancer stage in selected subgroups of the population, and projected estimates of the number of deaths preventable by earlier detection in women of various socioeconomic groups.

Methods

The Connecticut Tumor Registry has collected information on all Connecticut cases of invasive breast cancer and carcinoma-in-situ in a consistent manner since 1941.[6] The cancer stage is determined for each case by trained Medical Record Technicians at the Connecticut Department of Health Services (DHS) from detailed clinical, surgical, and histological reports. The staging system for breast cancer, unchanged since the registry began, can be summarized as follows: 1) *carcinoma-in-situ*; 2) *local*—invasive cancer localized to the breast; 3) *regional*—cancer in the breast with spread to regional lymph nodes or pectoral muscles; 4) *remote*—presence of distant metastases. The tumor registry has contained basic demographic information since its inception, but data on census tract of residence, which allow an estimation of socioeconomic status, have been included only since 1984.

The outcome measured was the distribution of cancer stage at the time of diagnosis. Comparisons of stage distribution were made by year from 1975 to 1985; for 1984 and 1985 comparisons were also made by race, place of residence, and socioeconomic status (SES), as estimated from census tract information. Census tract coding was not possible for 778 (17 percent) of 4,524 women with cancer diagnosed during those years. Data from these women were analyzed separately to test for potential bias the incomplete coding may have caused. We analyzed three different markers to identify census tract socioeconomic level (as determined in the 1980 census): 1) median household income; 2) percentage of persons below the poverty line; and 3) percentage of adults who have completed a high school education. These three variables were closely correlated, and the various definitions of census tract socioeconomic level led to identical conclusions; the data presented use the percentage of census tract adults who were high school graduates as the sole SES marker. Women were grouped into quartiles. The lowest SES quartile contained census tracts with 15–61 percent of adults who were high school graduates. The middle two quartiles were combined to form the middle SES group, which contained census tracts with 62–82 percent of adult high school graduates. The highest quartile contained census tracts with 83–97 percent of adult high school graduates. For analysis of Black women stratified by SES, the middle and higher SES groups were combined because of the small number of cancers in each stratum.

Population denominators for calculations of incidence trends from 1975 to 1985 were linear estimations based on 1970 and 1980 census figures for each age group. Incidence rates by SES were calculated using census tract population data from the 1980 census. Age-adjustment was done by the direct method, using the 1980 Connecticut population as the standard.

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The projected number of breast cancer-caused deaths in a cohort of women with breast cancer was derived according to the following general principle:

> Breast cancer-caused deaths = (Expected survivors in breast cancer cohort from actuarial estimates) − (Expected survivors in breast cancer cohort given presence of breast cancer)

The terms of this equation were calculated using: 1) 5-year survival rates by age and race from 1983 life tables; 2) 5-year relative survival rates for each stage established nationally[1] and in Connecticut; 3) estimates of the effect of SES on stage-specific 5-year relative survival rates from published studies;[7–9] and 4) mean annual numbers of breast cancer cases by age, race, and SES group during 1984–85 (see Appendix). "Nonpreventable" deaths represent similar estimates, assuming that the stage distribution in each age group achieved a goal as follows: *carcinoma-in-situ*—10 percent, *local*—60 percent, *regional*—25 percent, *remote*—5 percent of the total. This goal was felt to be reasonable based on published studies of breast cancer screening[5,10] and the current trend in Connecticut. "Preventable" deaths were defined as the difference between projected deaths and "nonpreventable" deaths. To help interpret these numbers of deaths, we calculated projected death rates by dividing them by the number of women in the population from which the cancer cases arose; population denominators were taken from the 1980 census. To study the longer-term impact of early detection, we projected 10-year death rates with similar methods.

We then assessed the reliability of these deaths rate estimates by varying the assumptions used, including: changing the program "goals", using different estimates of relative survival rates, and eliminating the differences in stage-adjusted survival by SES group. Changing these assumptions altered the magnitude of the projected death estimates, but caused only very small relative differences, and produced no difference in the conclusions regarding the SES group at highest risk. In general, increases in 5-year survival rates led to increases in the expected beneficial effect of early tumor detection, and increases in the ratio of projected preventable deaths in higher versus lower SES women. The data presented use 5-year survival rates established nationally (which include data from Connecticut)[1] and 10-year survival rates from the 1975–77 Connecticut breast cancer cohort.

The statistical significance of trends in disease stage was evaluated using extensions of the Mantel-Haenszel chi-square test for trends with multiple levels of exposure and multiple levels of disease.[11]

Results

Between 1975 and 1985, 21,131 breast cancers were diagnosed in Connecticut women. For 20,165 (95.4 percent) cancers, the available information was sufficient to accurately determine stage. In individual years, cancers of unknown stage represented between 3.9 percent and 6.0 percent of all cancers, with no increasing or decreasing trend. The trend of cancer stage among those cases in which information was complete is presented in Table 1. Between 1975 and 1981, there was little variation from year to year in stage at diagnosis, with no overall trend ($p = 0.29$). During this time, the percentage of cancers of known stage that were confined to the breast (*carcinoma-in-situ* and *local* stages) ranged from 52.2 percent to 55.9 percent. However, from 1982 to 1985 this percentage increased from 54.0 percent to 61.3 percent ($p = 0.0001$). Over these four years, the age-adjusted incidence of cancer in later stages (*regional* and *remote*) decreased slightly, from 5.06 to 5.02 cases per 10,000, while the breast cancer incidence overall increased from 11.6 to 13.6 and the incidence of disease confined to the breast increased from 5.9 to 8.0 cases per 10,000 (Figure 1).

During 1984 and 1985, the years in which information on SES was available, cancer stage was more closely and consistently related to SES than area of residence. There were no differences in the distribution of cancer stage among women residing in different counties. However, in cities of greater than 100,000 population (which have a disproportionately large share of lower SES women) only 56.7 percent of women presented with tumors confined to the breast, compared to 61.0 percent of women in smaller cities and towns ($p < 0.001$). Table 2 shows that women in the lower SES group, compared to women in the higher SES group, were less likely to have *carcinoma-in-situ* (6.2 percent vs 8.5 percent) or cancer confined to the breast (56.9 percent vs. 62.7 percent), and more likely to present with *remote* disease (8.9 percent vs 5.9 percent; $p = 0.001$ for trend). Middle SES group women had intermediate values. Black women, compared to White women, were less likely to have *carcinoma-in-situ* or cancer confined to the breast and more likely to have *remote* disease ($p = 0.007$ for trend). The differences in cancer stage by SES group were still present when cases were stratified by race ($p = 0.03$ for White women, $p = 0.04$ for Black women). There were no important differences between

TABLE 1—Percentage of Breast Cancers in Different Stages, Connecticut 1975–85

Year	Incidence*	Cancers of Known Stage				
		N	Ca-in-Situ	Local	Regional	Remote
1975	12.2	1756	3.9	50.7	37.3	8.1
1976	11.3	1641	3.3	52.4	35.6	8.6
1977	11.0	1651	3.4	52.5	35.2	8.9
1978	11.0	1641	3.0	50.7	35.2	11.1
1979	11.4	1716	4.0	51.2	38.2	6.6
1980	11.4	1761	4.0	49.9	38.5	7.6
1981	12.0	1867	3.7	48.5	38.7	9.1
1982	11.6	1814	4.9	49.1	38.5	7.5
1983	12.3	1973	4.5	50.4	37.1	7.9
1984	13.1	2125	6.3	53.1	33.1	7.4
1985	13.6	2220	7.4	53.9	31.5	7.2
Total		20,165				

*Cases per 10,000 women, including those of unknown stage, age-adjusted to 1980 Connecticut population.

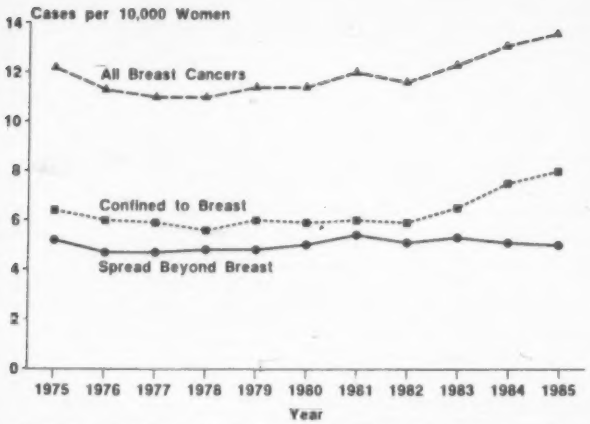

FIGURE 1—Incidence of Breast Cancer by Stage at Diagnosis, Connecticut, 1975–85

any of these subgroups in the percentage of cancers for which the stage was unknown.

The 778 women without census tract information were similar to women in known census tracts in the percentage that were Black or urban residents. Among the 684 of these women for whom the stage was known, the distribution of cancer stage was similar to that of women in known census tracts.

Population-based rates of the different cancer stages were calculated for each SES group. Lower SES women had lower crude and age-adjusted rates of breast cancer overall and of early-stage disease, but a slightly higher rate of metastatic disease (Table 3).

The numbers of cancer cases in each stage were then used to calculate projected breast cancer death rates for the different SES groups (Figure 2). These rates indicate the proportion of women annually diagnosed with fatal breast cancer ("fatal" meaning expected to die from breast cancer within five years from the time of diagnosis). To estimate the expected benefit of a moderately successful early detection program, these death rates were then separated into "preventable" and "nonpreventable" death rates as described. There were small differences among SES groups in the rates of "nonpreventable" deaths. However, lower SES women, compared to higher SES women, had a 25 percent higher projected breast cancer death rate (24.6 vs 19.7 per 100,000) and a greater percentage of those deaths classified as "preventable" (22 percent vs 11 percent). The preventable breast cancer death rate for lower SES women was 2.5 times as great as that for higher SES women (5.3 vs 2.1 per 100,000). A similar analysis projecting 10 years from the time of diagnosis produced parallel results; lower SES women had a 34 percent higher projected death rate (32.0 vs 23.8 per 100,000), and a greater percentage of those deaths "preventable" (20 percent vs 9 percent).

Discussion

The Connecticut Tumor Registry has for many years been an invaluable source of data on cancer incidence trends.[6] Recent advances in our understanding of cancer prevention allow us increasingly to use tumor registries like this as surveillance systems to aid in the design and evaluation of cancer control activities. As surveillance systems, tumor registries not only provide accessible information about the overall success or failure of control activities over time, but also identify subgroups of the population at unusually high risk of fatal disease.

Our analysis of the tumor registry data shows a shift toward early breast cancer detection in Connecticut in the 1980s, as indicated by an increase in the percentage of tumors diagnosed in early stages and a slight decrease in the incidence of later-stage disease. This shift represents an acceleration of the more gradual long-term trend toward earlier diagnosis of breast cancer present since the 1940s.[12,13] It is probable that this long-term trend is responsible (together with an improvement in stage-specific survival rates) for the stabilization of breast cancer mortality rates, in spite of a real increase in breast cancer incidence.[12–14]

No data are available regarding the frequency of screening mammograms or breast examinations in Connecticut. If breast cancer screening is responsible for the shift toward early-stage diagnosis, then we would hope to soon see a further decrease in the population-based incidence of *regional* and *remote* stage disease. Statewide surveillance of breast cancer should therefore periodically assess the incidence of disease by stage to monitor the success of early detection programs.

Both White and Black women in lower SES census tracts were more likely than race-matched women in higher SES census tracts to have later-stage disease at the time of breast cancer diagnosis. A definition of SES based on census tract information subjects the analysis to potential bias caused by an "ecologic fallacy." However, we would expect that the misclassification of women by this group attribute would tend to bias the data toward finding no differences by SES. In addition, our data show a consistent trend over three SES groups, are similar to the analysis of stage by race, and are similar to the findings of stage versus SES found by other authors.[7,8,15,16]

The relation between lower SES and late-stage diagnosis found by others has been noted in the process of correcting for the effect of stage on long-term survival rates. Rather than correcting for tumor stage, we used it as the outcome to measure the success of control activities. This allowed us to assess the effect of early detection programs independent of the effects of primary prevention and treatment programs.

Breast cancer screening programs may detect some early tumors which do not have the potential to progress to clinical cancer. Classifying these tumors as breast cancer would artificially increase both breast cancer incidence and the percentage of tumors diagnosed in early stages. This phenomenon may be partly responsible for the temporal trend and socioeconomic differences in early cancer rates. However, such benign tumors would not alter the population-based incidence of *regional* or *remote* cancer and would not have a significant impact on our projected breast cancer mortality rates.

Although lower SES women are more likely to present with later stage-disease and have poorer stage-adjusted 5-year survival rates[7,9] than higher SES women, they have a lower overall incidence of breast cancer than higher SES women. Therefore it is not immediately apparent which group should be a target of control programs. However, we estimate that these SES differences will lead to a 25 percent increase in deaths for lower SES women compared to higher SES women, with these deaths more than twice as likely to be preventable by early tumor detection.

We have had to make several assumptions in calculating these projected death rates. In particular, we have assumed

TABLE 2—Percentage of Breast Cancers in Different Stages by Socioeconomic Group and Race, Connecticut, 1984–85

Race	SES Group	Cancers of Known Stage				
		N	Ca-in-situ	Local	Regional	Remote
White	Lower	796	6.8	51.8	32.9	8.4
	Middle	1747	6.7	53.1	32.7	7.4
	Higher	897	8.2	54.0	31.9	5.9
Black	Lower	104	3*	44	40	13
	Middle	57	5*	58	26	12
	Higher	13	23*	62	8*	8*
All Women	Lower	907	6.2	50.7	34.2	8.9
	Middle	1822	6.6	53.1	32.6	7.7
	Higher	920	8.5	54.2	31.4	5.9
Total, known SES		3649	7.0	52.6	32.7	7.5

*Represents fewer than five cases.

TABLE 3—Crude (age-adjusted*) Incidence of Breast Cancer per 10,000 Women in Different Stages, 1984–85, by Socioeconomic Group

SES Group	Carcinoma-in-situ	Local	Regional	Remote	Total
Lower	0.65(0.65)	5.33(5.14)	3.60(3.51)	0.94(0.90)	10.8(10.4)
Middle	0.75(0.74)	5.98(5.88)	3.67(3.61)	0.87(0.85)	11.6(11.4)
Upper	1.06(1.04)	6.75(7.29)	3.91(4.08)	0.73(0.80)	12.7(13.5)

*1960 Connecticut population standard.

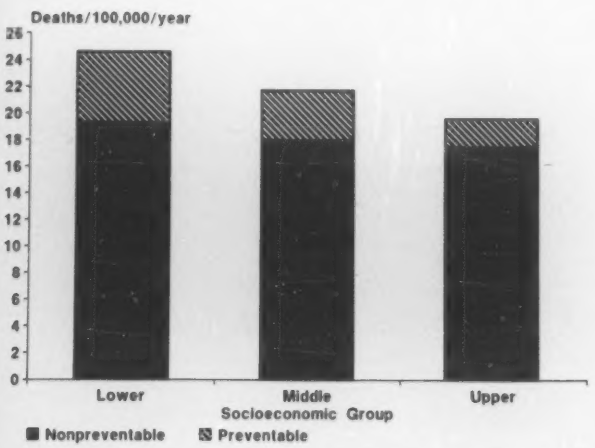

FIGURE 2—Projected Breast Cancer Death Rates for Cases Diagnosed in Connecticut during 1984–85

that within each SES group, long-term stage-specific cancer survival rates would remain unchanged after an early-detection program. This assumption is related to the effect of lead-time on cancer survival and mortality. A theoretical problem exists in assessing the benefit of screening programs if women who have cancers detected early do not have the expected improvements in long-term survival—if these women's deaths are neither prevented nor postponed, but simply occur further from diagnosis. However, over time, as cancers in Connecticut have been detected earlier, long-term survival rates for early-stage disease have actually increased (10-year rates are presently 99 percent for carcinoma-in-situ, 83 percent for cancer localized to the breast). Furthermore, two studies have demonstrated that screening has a beneficial long-term effect on mortality independent of its effect on diagnostic lead-time.[2,3,5] Therefore it is likely that the assumption of constant stage-specific survival rates is sufficiently valid to provide useful estimates regarding the benefit of early detection programs.

While the magnitude of our death rate projections depends on the numerical assumptions we have made regarding disease stage and survival, the trend toward a substantial increase in preventable deaths in lower SES women was present regardless of such assumptions. We believe these projections, combined with the established benefit of early tumor detection in decreasing cancer mortality,[2–5] provide strong justification for prevention programs to direct special attention to lower SES women.

It is probable that women of lower SES present with later-stage disease because of a combination of decreased access to medical care and a decreased awareness of or belief in the importance of early cancer detection. In the National Health Interview Survey, Black women had lower rates of utilization of breast examinations or mammography than White women, and among those >40 years old, were twice as likely as White women to have never heard of mammography.[17] Our data suggest that this difference may be due more to socioeconomic status than race. Data from neighboring Rhode Island indicate that among all women who had not had a mammogram in the past year, the most important reason for not having had the test was that they had believed it was unnecessary.[18] It is important to better define the precise reasons why women in lower SES groups appear less likely to use breast cancer screening techniques.

Connecticut has recently passed a law ensuring that all private and public medical insurance plans—including Medicare—pay for the cost of screening mammography. The Connecticut Department of Health Services is currently working with the American Cancer Society in the state to find ways

of better reaching lower SES women. We hope these efforts will improve early cancer detection and thereby contribute to the prevention of breast cancer morbidity and mortality.

APPENDIX

Estimated 5-Year Survival Rates by Cancer Stage

For each SES group, the number of projected breast cancer deaths (D) was calculated as the sum for each race of the following:

$$D = \sum_{s}\sum_{a} L_a \times C_{a,s} - \sum_{s} P_s \sum_{a} L_a \times C_{a,s}$$
$$= \sum_{s} (1 - P_s) \times (\sum_{a} L_a \times C_{a,s})$$

where s = stage, a = age-group, L_a = estimated 5-year actuarial survival for each age-group, P_s = estimated 5-year relative survival for each stage in that SES group (see below), and $C_{a,s}$ = number of actual cases of breast cancer in each age-group and stage. "Nonpreventable" deaths were calculated using the same equation except that the number of actual cases was replaced by the goal number of breast cancer cases in each stage.

	5-year Relative Survival by Cancer Stage				
SES Group	In situ	Local	Regional	Remote	All stages
High	.993	.912	.748	.190	.811
Middle	.989	.899	.685	.174	.782
Low	.986	.886	.622	.158	.754

The estimated 5-year relative survival rates were generated by adjustment of data from the Surveillance, Epidemiology, and End Results (SEER) program[1] with estimates of previously noted effects of SES on survival rates.[7–9] The rates used were as follows:

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Manual Being Developed to Assist Local Boards of Health in Policy-Making

To help improve the capabilities of local boards of health, a manual is being designed by the Association of North Carolina Boards of Health (ANCBH), under a $5,000.00 grant from the W. K. Kellogg Foundation. That financial support will enable ANCBH to review available information on public health policy-making and develop new resources specifically for boards of health in North Carolina, according to Vaughn Upshaw, Administrative Director of ANCBH in Pittsboro, NC. However, he added that the manual would be available to *all* boards of health in the US.

The manual is expected to offer a review of the state statutes in North Carolina, and also provide guidelines for board members to improve their assessment of community needs, policy-making skills, knowledge of agency operations, and advocacy for public health.

Local boards of health are empowered by the state to protect and promote public health. According to ANCBH President Ronald Tucker, "Growing concern for our environment and personal health increases the need for informed local officials." Few resources are available locally to boards of health on how to act effectively, he added. "Board members, health directors, health department staff, and the public will benefit from more knowledgeable and effective boards of health."

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Sentinel Health Event Surveillance: Skin Cancer of the Scrotum in New York State

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Abstract: Skin cancer of the scrotum is a disease that has been identified as a sentinel health event (occupational) (SHE(O)) that is necessarily occupationally related. The present paper examines the feasibility of using this disease in active cancer surveillance in New York State. After consulting cancer case reports, hospital records, death certificates, and city directories, we obtained occupational data for 17 of 22 cases of nonmelanoma skin cancer of the scrotum diagnosed between 1979 and 1984. Only three cases had occupations previously linked to scrotal cancer, while 11 of 19 cases had one or more possibly contributory medical conditions. The probable underreporting of scrotal cancer cases and incompleteness of occupational data may limit the usefulness of scrotal cancer as a sentinel health event (occupational) unless additional steps are taken. (*Am J Public Health* 1989; 79:1513–1515.)

Introduction

Cancer of the scrotum, which has been associated with exposures to numerous hydrocarbon products,[1] has been identified as a sentinel health event (occupational) (SHE(O)).[2] A SHE(O) is defined as:

> an unnecessary disease, disability, or untimely death which is occupationally related and whose occurrence may: 1) provide the impetus for epidemiologic or industrial hygiene studies, or 2) serve as a warning signal that materials substitution, engineering control, personal protection, or medical care may be required.[2]

SHE(O)s can be broken down into two broad categories: those which are necessarily occupationally related and would be unlikely to occur in the absence of occupational exposures, and those which may or may not be occupationally related. In the original SHE(O) paper,[2] scrotal cancer was identified as one of the three malignant neoplasm SHE(O)s that are necessarily occupationally related.

The Cancer Surveillance Program of the New York State Department of Health is assessing the feasibility of active surveillance using data from the State Cancer Registry to monitor SHE(O) cancers. This feasibility study has examined in detail the occurrence of necessarily occupationally related SHE(O) cancers in New York State and the information on individuals with these cancers obtainable from routine data sources. This paper presents the results of this examination for skin cancer of the scrotum.

Methods

The source of data was the New York State Cancer Registry, which receives reports from physicians, laboratories, and hospitals on all cases of cancer diagnosed in the state, as required by law. Cases are also obtained from death certificates, and through reciprocal interstate reporting agreements. Based on various quality control studies, cancer reporting to the Registry is estimated to be over 95 percent complete for the state.[3] At the time the Registry case listing for this analysis was obtained, case reporting was complete through 1984.

Since it is the epithelial cancers of the scrotum that have been identified with occupational exposures, analysis was confined to these morphologic types. Reporting to the Cancer Registry of nonmelanoma skin cancers of the scrotum, which account for the majority of epithelial scrotal cancers, began in 1979. The present analysis includes cases reported from 1979 through 1984.

Cancer Registry data were supplemented by an examination of hospital records for the cases of nonmelanoma skin cancer of the scrotum identified through the Registry. Hospital records were reviewed for information on smoking history, occupational exposures, course of illness, and any prior or concurrent medical conditions that may have been related to the development of scrotal cancer. Information on occupation was also sought from city directories and from death certificates when appropriate.

Incidence rates for scrotal cancer were computed as average annual rates for the period 1979 through 1984. Rates were age-adjusted to the 1960 United States population to allow for the comparison of rates in different populations.

Results

The New York State Cancer Registry identified 30 incident cases of epithelial skin cancer of the scrotum in New York State residents between 1979 and 1984. This corresponds to an average annual age-adjusted incidence of 0.56 per million males. The epithelial cancers included 22 cases of nonmelanoma skin cancer, all of which were the squamous cell type, and eight other epithelial types such as adenocarcinoma and unspecified carcinoma.

Occupational History

The cancer case report requests information on employer, industry and usual occupation, but only eight of 22 nonmelanoma skin cancer cases had an occupation listed on the case report. These occupations tended to be in the managerial and professional, or service categories. Hospital records, obtained for 19 of the 22, confirmed any occupations listed on the cancer case reports and provided occupations for five additional cases—all in the skilled, semi-skilled, or unskilled labor category. Death certificates supplied occupational information for three more cases, and a city directory provided occupation information for another case. After consulting these sources, five occupations remained unknown. These results are summarized in Table 1.

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TABLE 1—Occupational Categories of Males Diagnosed with Nonmelanoma Skin Cancer of the Scrotum, New York State, 1979–84, by Source of Occupational Information

Occupational Category[1]	Cancer Case Report	Hospital Records	Death Certificate	City Directory	Total
Managerial or professional	4	0	0	1	5
Skilled, semi-skilled or unskilled workers[2]	1	5	1	0	7
Service occupations	2	0	0	0	2
Technical, sales and support	1	0	1	0	2
Farm occupations	0	0	1	0	1
Total	8	5	3	1	17

NOTES: 1. Occupational categories derived from summary groupings found in the *1980 Census of Population: Alphabetical Index of Industries and Occupations*, US Department of Commerce, 1982.

2. Includes US Census categories Precision Production, Craft and Repair Occupations, and Operators, Fabricators and Laborers.

Five additional occupations remained unknown.

Three of the persons classified as skilled, semi-skilled, or unskilled workers had occupations that could have put them at elevated risk of scrotal cancer. These included a bridge painter who had reported occupational exposures to graphite and kerosene, a machinist who may have been exposed to carcinogenic cutting oils, and a mechanic for a heating equipment business who had possible exposure to soots and tars. Each of these substances has been linked with scrotal cancer.[1,4] None of the other occupations presented any apparent opportunities for relevant exposures to occur.

Medical Risk Factors

Medical records showed that eight of the 19 squamous cell skin cancer cases with available medical records had a prior history of cancer. Five of these prior cancers were skin cancers (two squamous cell, one malignant melanoma, one unspecified skin, and one mycosis fungoides). One other person had a history of psoriasis, which was treated with coal tar. The patient with mycosis fungoides had undergone PUVA (psoralen-ultraviolet-light) treatments, a regimen often used to treat psoriasis and linked to squamous cell cancers.[5] Other conditions which may have had significance in the development of scrotal cancer are a prior injury to the urinary tract, undescended testis, and "very poor hygiene." The latter condition has been identified as a risk factor for scrotal cancer from the days of Percivall Pott,[6,7] up to a recent case series.[8] There were 11 persons in all who may have had one or more possibly contributory medical conditions.

Other Geographic Areas

The age-adjusted rate (0.56 epithelial cases per million man-years) in New York State for 1979–84 compares with age-adjusted rates of 1.32 per million for Connecticut during the 45-year period 1935–79, and 0.97 per million in Connecticut for 1975–79.[5] More recent data from Connecticut* show an age-adjusted rate of 1.28 per million males for all cancers of the scrotum for the period 1978–84. If epithelial cancers still account for 75 percent of all scrotal cancers reported in Connecticut, then the epithelial scrotal cancer rate is roughly 0.95 per million males, nearly twice that observed in New York.

The age-adjusted incidence rate of 0.52 per million males observed for New York City was compared with that for Metropolitan Detroit, a northern industrial urban area with a similar proportion of black males. The Detroit figures for 1979–84** give an average annual age-adjusted rate of 1.14 cases per million males, which is again about twice that for New York City.

In New York, none of the 30 epithelial cancers of the scrotum reported through 1984 was the (less serious) basal cell carcinoma. Nine of 71 confirmed epithelial cancers in Connecticut from 1935 through 1979 were basal cell, and three of 16 epithelial cancers in Metropolitan Detroit from 1979 through 1986 were basal.

Discussion

Incidence rates of epithelial cancers of the scrotum in New York State and New York City are approximately half those of the Connecticut and Detroit SEER‡ areas, respectively. This may indicate some degree of underreporting, but the extent to which this deficit may also reflect true differences in occupational exposures is not known. The predominance of the squamous cell type also supports the possibility of differential underreporting of the less serious cases.

Skin cancer of the scrotum has been characterized as a cancer whose occurrence is unlikely in the absence of occupational exposures.[2] In the present study, evidence of a possible occupational origin was found for only three of the 17 cases for whom occupational data were available. In contrast, 11 of the 19 persons for whom hospital records were available had prior or co-existing medical conditions that might be related to their scrotal cancers.

One possible interpretation of these findings is that occupational cancer of the scrotum in New York State is at such a low level that "background" or nonoccupational cases predominate. However, the possibility of significant underreporting, particularly of uncomplicated cases, makes it conceivable that an occupationally related spatial or temporal clustering may not be reflected in Registry data.

A related issue is that of the completeness and quality of the occupational data that are available. Less than half of cancer case reports had occupation listed, and those occupations that were listed tended to be of the type where unusual exposures generally do not occur. For the remainder of cases, it was necessary to consult several sources of varying accessibility and completeness. Hospital records provided occupational information for more patients than the cancer case reports, but had to be sent for specially, a process often taking several weeks. New York State death certificates are generally good sources of occupational information, but their usefulness is limited for nonfatal diseases such as skin cancer of the scrotum. It was also not feasible to obtain death certificates for New York City residents, who made up 40 percent of the cases. City directories can provide occupational information, but are not a consistent source since complete listings are not available for all areas. After con-

*Flannery JT: Chief, Connecticut Tumor Registry, personal communication, 1988.

**Schwartz A: Michigan Cancer Foundation, personal communication, 1988.

‡SEER: Surveillance, Epidemiology, and End Results, the population-based cancer surveillance system established by the National Cancer Institute in 1970.

sulting all these sources, nearly one fourth of the occupations were still unknown. Furthermore, the occupations that were ascertained represent the usual or the most recent occupation; other pertinent occupations may not be recorded.

Steps may be taken to improve the representativeness and completeness of data on scrotal cancer cases and their occupations collected in the future. One would be to remind all physicians of their duty to report nonmelanoma skin cancers of specified sites, and of the proper format and reportable items. A second step might be to send a reminder to all hospital tumor registrars regarding the importance of completing the occupation section on the cancer case report. Obtaining more complete occupational histories would require contacting cases or their next-of-kin directly, often requiring more extensive resources than generally available for surveillance activities.

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The Homeless Families Program—A National RWJ/HUD Initiative

A multi-million dollar public/private program for homeless families has been announced as a cooperative national initiative of the Robert Wood Johnson Foundation and the US Department of Housing and Urban Development (HUD).

The Homeless Families Program is intended to help communities develop the infrastructure necessary to strengthen the potential of homeless parents and their children to function successfully. The Johnson Foundation will make as many as eight two-year grants of up to $300,000 each to eligible cities (population 250,000 or more) or the counties in which these cities are located; Public Housing Authorities (PHAs) in jurisdictions selected under the competition will also be eligible for a share of the Section 8 certificates HUD will set aside for this purpose. The grants and housing certificates will support community-wide projects aimed at providing comprehensive health and supportive services and housing assistance to homeless families suffering from complex, often chronic, problems, including health and mental health problems, inadequate education or job training, and lack of day care and other services.

The goal of the Homeless Families Program is to show that even in the most dysfunctional families, appropriately designed health and support services, combined with suitable housing, can help parents become healthy and hopeful, and avoid becoming permanent members of the nation's underclass. The program also seeks to promote the long-term expansion of permanent housing options for these families.

Interested applicants should send a letter of interest to: **Dr. James O'Connell, Director, Homeless Families Program, Massachusetts General Hospital, 32 Fruit Street, Boston, MA 02114.** Limit the letter to two typed, double-spaced pages. Identify the parties to be involved in developing the application, documenting local government support, and generally outlining the project to be proposed. A contact person should be identified to serve as principal liaison during the application process. Application materials will be sent upon receipt of the letter. **The deadline for receipt of applications is December 22, 1989.** Grant recipients will be announced May 1990. The Robert Wood Johnson Foundation is at College Road, PO Box 2316, Princeton, NJ 08543-2316.

Carcinogenicity of Dark Liquor

KENNETH J. ROTHMAN, DRPH, CRISTINA I. CANN, AND MARVIN P. FRIED, MD

Abstract: To investigate whether the non-alcohol content of distilled alcoholic beverages affects the carcinogenicity of the beverage, we conducted an epidemiologic study of laryngeal and hypopharyngeal cancer. We interviewed 384 cases (or spouses, for deceased cases), and compared their responses with those of 876 controls. We classed distilled liquors as dark or light, a rough division according to content of potentially carcinogenic compounds in the beverages. The relative effect on hypopharyngeal cancer risk was much stronger for those who reported high consumption of dark liquor (relative risk = 4.4, 90% confidence interval = 2.9, 6.8) than for those reporting comparable consumption of light liquor (relative risk = 1.3, 90% CI = 0.8, 2.1). For laryngeal cancer, consumption of dark liquor had a smaller effect, and there was little distinction between the effects of dark and light liquor. The data appear consistent with the theory that the non-alcoholic content of distilled alcoholic beverages is a determinant of cancer risk, and that alcoholic beverages act topically rather than systemically in their carcinogenic action. (*Am J Public Health* 1989; 79:1516–1520.)

Introduction

Alcohol consumption is carcinogenic to most of the tissues in the upper aerodigestive tract, including the larynx, pharynx, and mouth.[1–6] Little is known, however, about the mechanism for alcohol carcinogenicity. The effect of alcohol on cancer risk appears greater for tobacco smokers than for non-smokers,[7] and some have questioned whether alcohol consumption is carcinogenic among nonsmokers.[8] Theories about the mechanism of alcohol carcinogenesis include the possibility that alcohol has a topical action, acting as a solvent for carcinogenic compounds, possibly enhancing their penetration through mucosa[8,9]; that alcohol consumption alters the nutritional balance and consequently leads to reduced consumption of dietary preventives that act through a central mechanism on epithelial cells[8,10,11]; and that alcohol directly affects metabolism in a way that increases the susceptibility of certain mucosal cells to cancer.[8] If alcohol acts as a solvent, its carcinogenic effect should be greater among smokers than among non-smokers. In addition, alcoholic beverages that come with already dissolved carcinogens should be more carcinogenic than alcoholic beverages free of such ingredients. This latter effect should be more easily measured among non-smokers, since smokers may inhale enough carcinogens in smoke to overwhelm the possibly smaller effect of carcinogenic substances in the beverages themselves.

Various carcinogens or potential carcinogens have been detected in alcoholic beverages. Beer, for example, has been reported to contain nitrosodimethylamine.[12] Scotch whisky is made by exposing sprouted barley to the smoke of burning peat,[13,14] and many spirits, including bourbon, dark rum, and rye are aged in charred oak barrels.[14] Cognac is aged in limousin oak barrels from which the beverage acquires a high tannin content.[14] Tannin is reportedly carcinogenic in both rats and mice following subcutaneous injection.[15] Despite the presence of these carcinogens in alcoholic beverages, it is uncertain whether the type of alcoholic beverage is an important determinant of cancer risk. For this investigation, we restricted our attention to distilled alcoholic beverages, which have the highest concentrations of alcohol, and evaluated the comparative carcinogenic effects of dark liquor versus light liquor. We classed all whiskeys, dark rum, and cognac as dark liquors, and vodka, gin, and light rum as light liquors. Dark liquors as a group contain much higher concentrations of organic compounds that include higher alcohols, esters, and aldehydes and other congeners.[16] Light liquors are highly distilled alcoholic beverages that are comparatively free of additional chemicals. Many vodkas, for example, are nearly pure ethanol solutions.[16]

Methods

The study population comprised all residents of the Boston Standard Metropolitan Statistical Area during the period January 1, 1982 through December 31, 1984. We prospectively identified cases of laryngeal and hypopharyngeal cancer that were diagnosed in the study population during the study period with the cooperation of 119 otolaryngologists in the area. We would have liked to include cases of mouth cancer in the study, but the otolaryngologists who were our major source for case ascertainment did not see in their practices a high enough proportion of the mouth cancer cases occurring in Greater Boston, whereas they saw the great majority of cases of laryngeal and hypopharyngeal cancer. Before the study, we wrote to all otolaryngologists and head and neck surgeons in Greater Boston and surrounding communities (as far as Worcester); during the study period we continued to approach new practitioners for their cooperation. In all we asked 214 otolaryngologists and head and neck surgeons to cooperate, of whom 119 agreed. Most of the physicians who did not cooperate, some of whom were retired or semi-retired, saw few cancer patients, so that the proportion of potentially eligible cases who were ascertained through physicians' practices is far greater than the proportion of cooperating physicians. We contacted all cooperating physicians monthly to learn of any newly diagnosed eligible cases. All cases had to have a histologically confirmed diagnosis of cancer of the larynx or hypopharynx. We sought written permission from the responsible physician to contact each eligible case.

As an independent source of case ascertainment, we included all cases of laryngeal and hypopharyngeal cancer recorded by the Massachusetts Cancer Registry. The Massachusetts Cancer Registry is a population-based registry to which all hospitals and clinics are required to report all cancer cases, excluding certain skin carcinomas. (At the time that we began this study, the Registry was new and was not an

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adequate source by itself for case ascertainment.) For each case identified by the Registry, we approached the responsible physician and obtained written permission to contact the patient, if the patient had not been identified previously.

For each case identified, we randomly selected as control subjects up to three people from the listing of residents of the same town as the case. These listings are published by each town under state law; with them we were able to ascertain conveniently a geographically representative series of controls that had roughly the same age and sex distribution as the cases. The process called for randomly identifying a place in the town book, and from that place identifying the next resident of the same sex born in either the same calendar year as the case subject or one year earlier or later. This step was repeated for each control subject. The matching criterion for year of birth was relaxed slightly for cases 70 years of age or over. To be eligible, controls had to be alive and still residing in the same town as the case at the time that we attempted to contact them. Many of the potential control subjects that we identified, upon investigation, were not alive; in these instances we continued to pick names from the town listing until we reached a maximum of three controls per case. (Toward the end of data collection, to avoid prolonging the study, we did not seek a full set of three controls for each case.)

All cases and controls received an introductory letter that stated in general terms that we were conducting a health study and desired to arrange an interview. The subjects were then contacted, usually by telephone, to arrange a personal interview conducted by trained study personnel. Those subjects who agreed were interviewed at home using a completely structured interview form. The interviewers were not told that the specific focus of the study concerned alcoholic beverages. The interview covered demographic data, medical history, occupational history, tobacco intake, certain dietary factors, drugs, mouthwashes and gargles, and other items. We obtained detailed data about timing, intensity, and type of exposure to alcoholic beverages and tobacco products. Nearly all interviews were conducted in English; three were in Spanish. For cases who had died or were unable to communicate owing to recent laryngeal surgery, interviews were conducted with the help of a spouse or with the spouse directly. One interview was conducted with the mother of a case, with whom the subject lived all his life except during military service.

For the stratified analyses, the estimation of relative risk (RR) across several strata was performed using maximum likelihood techniques to estimate the common odds ratio.[17]

Results

We identified 679 eligible cases, of whom 425 were ascertained directly from the cooperating physicians, and 254 were ascertained later through the Massachusetts Cancer Registry. We attempted to contact 625 of these cases, and could locate 604. No contact was attempted for 29 because the responsible physician was not cooperating with the study; for 20 because the physician's office requested that we not include the patient in the study; and for five cases, ascertained through the Veterans Administration, because there was no response to several requests from the responsible physician for consent by the patient to include him or her in the study. Of the 604 cases located, 42 were too ill for us to interview. An additional 128 patients had died before we contacted them. Of these 128, we obtained an interview with the spouses of 27, but 71 left no surviving spouse, 12 spouses were not available or did not respond, 10 spouses refused to be interviewed, and eight cases were identified through the Veterans Administration, which did not permit us to contact the surviving spouse. An additional 77 cases refused to participate, eight because of language problems. The total number of cases or spouses who were interviewed was 384; 18 of these interviews were with spouses of living cases who could not communicate readily, and one was with the mother of a case. The "response rate" among cases may be calculated in various ways. Of the 679 originally identified cases, only 57 percent are included among those interviewed, but this group represents 82 percent of those for whom an interview was possible (604 located cases minus 42 who were too ill, 83 who died with no spouse to contact, eight for whom we were not allowed to contact the spouse, and eight with language difficulties).

We identified 2,312 potential control subjects. We received no response to our initial letter addressed to 721 of these people. Many of these, however, no longer lived at the listed address when we attempted contact, or had died. The total, therefore, includes the names of an uncertain number of subjects who were not actually eligible for inclusion as a control. We were able to locate 1,591 of the potential controls; of these, 27 did not participate because of language problems, and 588 did not participate for various other reasons. We interviewed 883 controls, of whom seven gave interviews that we considered unreliable and did not use. The proportion of located controls whom we interviewed (55 percent) is lower than we would have liked, and reflects the increasing difficulty of obtaining cooperation for general population studies.[18]

The most important risk factors to consider as potential confounding factors in a study of laryngeal and hypopharyngeal cancer are age, sex, alcohol consumption, and tobacco use. Total alcohol consumption may be regarded as a possible confounder of the non-alcoholic component of dark or light distilled liquor consumption. Tobacco use may not only confound, but also modify an alcohol effect. Occupational risk factors for these cancers have also been reported, but these risk factors affect only small proportions of the population, and should not be strongly related to type of alcoholic beverage consumed, so that confounding from occupational risk factors was not a concern in this analysis. Socioeconomic status is not a strong risk factor conditional on total alcohol consumption and tobacco use.

We first examined the relation between cancer occurrence and the maximum amount of either dark or light liquor consumed weekly at any time. For analysis, we categorized subjects into three levels of maximum consumption (one drink per week or less, two-six drinks per week, and seven or more drinks per week), and compared those in the highest category with those in the lowest. The crude RR for drinking seven or more drinks per week of light liquor, relative to one or less, was estimated to be 1.7, with a 90 percent confidence interval (CI) of 1.3, 2.3. Controlling sex and age by stratification and calculating the maximum likelihood estimate of a common odds ratio indicated that despite the matching (which can introduce confounding) there was no confounding by these factors. We defined tobacco use in terms of pack-years of cigarette smoking (one pack-year equals 7,305 cigarettes smoked), or pack-year equivalents, based on comparable amounts of tobacco consumed, for cigar, pipe and cigarillo smokers and users of smokeless tobacco. We considered one cigar the equivalent of five cigarettes; one

pipeful, one plug, or one cigarillo the equivalent of two cigarettes. For these analyses we divided total tobacco use into two categories, those who reported less than 25 pack-years ("light" smokers) and those who had reported at least 25 pack-years ("heavy" smokers). Finer stratification of smoking produced intolerably small numbers within strata, and the consistently heavy smoking of laryngeal cancer cases precluded using a category boundary less than 25 pack-years. This stratification diminished the point estimate for light liquor consumption slightly, to 1.5. We then controlled simultaneously for tobacco and for the total amount of alcohol consumed; we estimated the total lifetime consumption of ethanol, including beer, wine, and other sources, and we then categorized the total into three levels: fewer than 1,000 drinks, 1,000–14,999 drinks and 15,000 or more drinks. One drink was equivalent to one shot (1½ ounces) of distilled liquor, 12 ounces of beer, and five ounces of wine. Control of tobacco and total alcohol consumption reduced the effect of light liquor consumption, comparing the highest level with the lowest, to an estimated RR of 1.1 with a 90% CI 0.8, 1.5.

The crude RR of consuming seven or more drinks per week of dark liquor, relative to one drink per week or less, was estimated to be 2.6, 90% CI 2.0, 3.2. This value was essentially unchanged after controlling for age and sex by stratification. Controlling for pack-years of tobacco use, however, reduced the point estimate to 1.9, 90% CI 1.5, 2.4. Controlling additionally for total lifetime alcohol consumption reduced the effect estimate to a RR of 1.3, 90% CI 1.0, 1.7.

The difference in effect estimates for the maximum rate of consumption of dark versus light liquor is relatively modest; nevertheless, even after control of confounding by tobacco and total alcohol consumption, there was some indication of a possible carcinogenic effect for dark liquor that did not exist for light liquor.

Cases were classified according to site of tumor into three major categories, hypopharyngeal (84 cases), supraglottic (125) and glottic (175), and the above analyses were repeated for each anatomic site separately. There were no cases of subglottic cancer among the 384 cases interviewed. These results are summarized in Table 1. The effects of dark liquor and light liquor appear similar for the supraglottis and glottis, but dark liquor appears to increase the risk of hypopharyngeal cancer whereas light liquor does not.

To determine whether the effect of dark liquor varied according to level of tobacco use, we examined the effects of drinking various quantities of light and dark liquor separately for light and heavy tobacco users. The distribution of cases for each type of cancer, and controls, is given by the maximum weekly intake of dark liquor and by category of tobacco use in Table 2. There is an increasing pattern of risk for greater maximum weekly consumption of dark liquor for both tobacco-use categories. The trend is again concentrated among hypopharyngeal cases (although it is unstable owing to the small number of cases among those with fewer than 25 pack-years of tobacco use). The joint effect of tobacco use and heavy consumption of dark liquor appears substantially greater than additive for the hypopharyngeal cases. This observation is based on a comparison of the highest versus lowest category of liquor consumption for each of the tobacco categories: the difference is 16.7 in the high-use category and 5.0 in the low-use category. The small number of cases in the latter category affects this comparison also, making the evidence for interaction in these data tentative.

We then conducted multivariate logistic analyses to examine the effects of dark and light liquor consumption in the same analysis. Conditional logistic models, which control for the matching variables of town, age and sex, gave results similar to the corresponding unconditional logistic models,[17] and are not reported. These analyses provided the efficiency to evaluate three levels of consumption of dark and light liquor simultaneously. We categorized subjects according to total number of drinks of dark or light liquor consumed during the subject's lifetime, which we calculated from the detailed histories that we obtained. We used categories of fewer than 200, 200–4,999, and 5,000 or more drinks; those who consumed fewer than 200 drinks formed the referent category. Although the total number of drinks is a function of age, age was found not be confounding, nor was sex, and these variables were therefore omitted from the regression models presented here. The model in Table 3, with terms for two levels of dark and light liquor consumption (in addition to the implied referent category), indicates an association with cancer risk of heavy consumption of dark liquor, and a smaller association for heavy consumption of light liquor. The comparable model restricted to hypopharyngeal cases (Table 4) shows a much stronger effect of heavy consumption of dark liquor and no material association for light liquor consumption. When terms for total alcohol consumption are added to the models in Tables 3 and 4, the effect of heavy consumption of dark liquor is smaller, but some effect

TABLE 1—Relative Risk Estimates for Consuming Seven or More Drinks per Week of Light and Dark Liquor, Relative to Consuming One Drink or Less, with 90% CI, for Three Anatomic Sites, Controlling for Tobacco Use and Total Alcohol Consumption

	Light Liquor		Dark Liquor	
Anatomic Site	RR	90% CI	RR	90% CI
Hypopharynx	1.0	0.6, 1.7	1.6	1.0, 2.5
Supraglottis	1.2	0.7, 1.8	1.2	0.8, 1.8
Glottis	1.0	0.7, 1.6	1.2	0.8, 1.8
All Sites Combined	1.1	0.8, 1.5	1.3	1.0, 1.7

TABLE 2—Distribution of Subjects by Maximum Weekly Consumption of Dark Liquor and by Lifetime Tobacco Use

	Tobacco Consumption					
	Fewer than 25 pack-yrs			25 or more pack-yrs		
	Dark Liquor (drinks/week)			Dark Liquor (drinks/week)		
Subjects	<1	2–6	7+	<1	2–6	7+
Controls	253	53	28	298	88	156
Hypopharynx Cases	3	0	2	25	10	44
Supraglottis Cases	1	2	1	61	16	44
Glottis Cases	20	4	1	68	22	60
RR (all cases combined)	1.0	1.2[a]	1.5[a]	5.4[a]	5.8[a]	10.0[a]
				1.0	1.1[b]	1.8[b]
RR (hypopharynx cases)	1.0	0.0[a]	6.0[a]	7.1[a]	9.6[a]	23.8[a]
				1.0	1.4[b]	3.4[b]
RR (larynx cases)	1.0	1.4[a]	0.9[a]	5.2[a]	5.2[a]	8.0[a]
				1.0	1.0[b]	1.5[b]

[a]Risk ratio with the low-tobacco, low-alcohol category as the referent.
[b]Risk ratio with the high-tobacco, low alcohol category as the referent.

TABLE 3—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.343	0.193	1.41	1.03, 1.94
Dark Liquor (5,000+ drinks)	0.854	0.141	2.35	1.86, 2.96
Light Liquor (200–4,999 drinks)	−0.680	0.234	0.51	0.34, 0.74
Light Liquor (5,000+ drinks)	0.462	0.174	1.59	1.19, 2.11

remains, whereas there is no effect of heavy consumption of light liquor conditional on total ethanol consumption. Table 5 gives the model with total alcohol consumption for hypopharyngeal cases.

We examined the influence of dark liquor consumption for various time periods before the cancer was diagnosed on hypopharyngeal cancer risk. These analyses indicated that consumption of dark liquor within the 10 years preceding diagnosis was much less strongly related to occurrence than was consumption in earlier decades.

Discussion

Methodologically the greatest concern in this study is the response rate, especially that of controls. Only 55 percent of the located controls were interviewed, and an unknown number of eligible controls were not located, in part because many of our subjects came from neighborhoods with high population turnover. The concern is that the subjects actually interviewed could have had exposure histories that differ from those not interviewed, possibly because the exposure is directly or indirectly related to the sequence of steps leading to inclusion in the study. Were the study focusing on total alcohol consumption, this concern would be intensified, since it is easy to image that the amount of alcohol consumed could affect the likelihood of participation. For the exposure of interest, however, which is the type of distilled alcoholic beverage consumed, we conjecture that selection bias from non-participation is a less serious problem. An important bias would have to involve marked selective participation among controls for consumers of dark liquor relative to light liquor or vice versa, independently of the total amount of alcohol consumed. Although there may be social class differences among liquor types that might introduce an association between beverage preference and the decision to participate in the study, the magnitude of these associations would have to be very strong to introduce a worrisome degree of bias into the comparisons reported here.

TABLE 4—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis (Hypopharyngeal Cases Only)

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.396	0.407	1.49	0.76, 2.90
Dark Liquor (5,000+ drinks)	1.483	0.2631	4.40	2.85, 6.78
Light Liquor (200–4,999 drinks)	−0.385	0.405	0.68	0.35, 1.32
Light Liquor (5,000+ drinks)	0.237	0.311	1.27	0.76, 2.12

TABLE 5—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis Controlling Alcohol Consumption from All Sources (Hypopharyngeal Cases Only)

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.019	0.422	1.02	0.51, 2.04
Dark Liquor (5,000+ drinks)	0.559	0.284	1.75	1.10, 2.79
Light Liquor (200–4,999 drinks)	−0.559	0.405	0.57	0.29, 1.11
Light Liquor (5,000+ drinks)	−0.136	0.308	0.87	0.53, 1.45
Total Alcohol (1,000–14,999 drinks)	1.522	0.671	4.58	1.52, 13.8
Total Alcohol (15,000+ drinks)	2.635	0.632	13.9	4.93, 39.4

Another potential concern is the use of surrogate interviews for 12 percent of cases. Cases who were deceased were included if a spouse could be interviewed. We also interviewed the spouses of 18 cases who had difficulty communicating. Spouse interviews presumably vary in accuracy depending on the information sought. The recent history of tobacco use, alcohol use, and other factors of interest is likely to be more accurate than early use. Alcohol consumption, which sometimes occurs without complete spousal awareness, may be particularly susceptible to error in spousal interviews. Here again our focus on type of beverage more than on amount mitigates the potential bias.

We used no surrogates for controls, since controls had to be alive to be eligible. Technically, since controls were matched individually to cases, controls who were alive at the time that the case was diagnosed should be eligible, regardless of whether they died before we attempted to contact them. Our decision not to include such individuals was motivated by convenience; it presents little problem if we can assume that there is no important departure from a dynamic equilibrium in the prevalence of type of distilled alcoholic beverage consumed.

Because all the exposure information in this study was reported by the subjects, there is presumably a substantial amount of misclassification. Some of the misclassification stems from faulty recall, and some from simplifying assumptions that we could not avoid in constructing summary measures of exposure. To the extent that such misclassification is comparable for cases and controls, the effect estimates in this analysis are underestimates of the actual effects.

The carcinogenic effect of alcoholic beverages is already well established.[19–21] When controlling for total alcohol consumption, which permits the evaluation of the effect of the non-alcohol component of alcoholic beverages, the additional cancer risk estimated for heavy consumption of dark liquor is modest.

What is important to note in these findings, however, is the divergent pattern of risks for upper airway cancer experienced by heavy consumers of dark liquor and heavy consumers of light liquor among hypopharyngeal cases. The site localization and the differences in effects between light and dark liquor are predicted by the theory that the carci-

nogenic action of alcohol is mainly topical, rather than systemic, and that the role of alcohol is to act as a solvent for other carcinogens, increasing the penetration of these carcinogens into epithelial tissue. The interactive effect with tobacco use that was observed is also consistent with this theory. These findings, on the other hand, are difficult to reconcile with alternative theories of alcohol carcinogenesis that suggest that ethanol increases carcinogenic risk through central rather than topical mechanisms.[8]

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'Children and Hospitals Week' Designated for March 25–31, 1990

A public awareness campaign focusing on the special needs of children and families in health care settings will be held March 25–31, 1990 throughout the United States. Designated "Children and Hospitals Week," the nationwide activity will feature health fairs, hospital open houses, preparation programs, "Teddy Bear" Clinics, library and school exhibits, educational conferences, and hundreds of other events. The theme for the week is "Commitment to Caring."

Communities and organizations who wish to participate in the Children and Hospitals Week can send for a free Planning Guide, which includes ideas for activities, publicity, and fund raising. The planning guide is available from:

Association for the Care of Children's Health (ACCH)
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ACCH is a multidisciplinary organization devoted exclusively to the psychosocial well-being of children and families in health care settings.

Prevention of Childhood Injuries: Evaluation of the Statewide Childhood Injury Prevention Program (SCIPP)

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Abstract: We evaluated the effectiveness of a community-based injury prevention program designed to reduce the incidence of burns, falls in the home, motor vehicle occupant injuries, and poisonings and suffocations among children ages 0–5 years. Between September 1980 and June 1982, we implemented five injury prevention projects concurrently in nine Massachusetts cities and town; five sites, matched on selected demographic characteristics, were control communities. An estimated 42 percent of households with children ages 0–5 years were exposed to one or more of the interventions over the two-year period in the nine communities. Participation in safety programs increased three-fold in the intervention communities and two-fold in the control communities.

Safety knowledge and practices increased in both intervention and control communities. Households that reported participatory exposure to the interventions had higher safety knowledge and behavior scores than those that received other community exposure or no exposure to intervention activities.

We found a distinct reduction in motor vehicle occupant injuries among children ages 0–5 years in the intervention compared with control communities, associated with participatory exposure of about 55 percent of households with children ages 0–5 years. We have no evidence that the coordinated intervention programs reduced the other target injuries—although exposure to prevention messages was associated with safety behaviors for burns and poisonings. (*Am J Public Health* 1989; 79:1521–1527).

Introduction

Injuries are the leading cause of death and a major cause of serious morbidity and disability among children and youth.[1] Yet, there have been relatively few efforts to carry out controlled community prevention trials. Schlesinger, *et al*, evaluated a health education program for families with children less than 7 years of age in a study community using a matched community as a control, but found no reduction in medically attended injuries.[2] The other population-based intervention trials reported have focused on the prevention of specific injury types, such as burns[3,4] and pedestrian injuries.[5,6] We evaluated the effectiveness of the Statewide Childhood Injury Prevention Program (SCIPP)—a community-based injury prevention program designed to reduce the incidence of burns, falls in the home, motor vehicle occupant injuries, poisonings, and suffocations among children ages 0–5 years. Our rationale was that by combining the safety interventions available at the time we could reduce childhood injury rates.

Methods

Study Design

The investigation involved concurrent implementation of five injury prevention projects in nine Massachusetts cities and towns with a total population of 139,810 persons and selection of five control communities with a population of 146,866, matched on demographic characteristics to the intervention communities. The injury prevention projects commenced in September 1980 and continued for a 22-month period, through June 1982. A surveillance system to collect data on incident cases of childhood injuries was established in September 1979, one year before the intervention began, and was maintained throughout the study period to August 1982.[7] Telephone surveys of attitudes, knowledge, self-reported safety practices, and exposure to injury prevention projects were completed before the beginning of the interventions in August 1980 and again after their completion in August 1982.

Intervention Methods

Five intervention programs were selected:

- injury counseling for the parents of young children by pediatricians using the developmentally based Framingham Safety Surveys[8];
- school and community burn prevention education using the Project Burn Prevention curricula[4];
- household injury hazard identification and control through systematic home safety inspections by specially trained local board of health staff[9];
- community-wide promotion of the Massachusetts Poison Control System's telephone information service and public education about poison prevention[10,11]; and
- promotion of child automobile restraint use, particularly for children in preschools and infants leaving maternity hospitals.

The primary target injuries, target age groups, and components for each project appear in Appendix A. The principal focus was families with children ages 0–5 years. Implementation of the projects occurred at health and educational institutions that already served children and their families in the study communities. The five prevention programs were integrated in the same study communities to reinforce each other's preventive messages.

Site Selection

The site selection process involved comparison of data on all 351 cities and towns in Massachusetts for: population

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size and density, age composition, education level, family income, housing characteristics, hospital usage patterns, and characteristics of local pediatricians, school districts, and boards of health,[12–16] and informal information supplied by an advisory committee. The sites were grouped into two comparable sets of intervention and control communities. Details have been published previously.[7]

Measurements

The unit of intervention of this study was the community. Therefore, we evaluated the program's effects on the level of safety knowledge and practices and on the incidence of childhood injuries in the study and control communities with counts of project participants, pre- and post-intervention telephone surveys, and a hospital-based injury surveillance system.

Injury Prevention Project Activities: Reports from each intervention project provided counts of the numbers of persons and households reached and materials distributed. We also conducted a post-intervention telephone survey in August 1982 to estimate exposure to interventions over the preceding two years.

Telephone Surveys: We conducted telephone surveys in both intervention and control communities with use of a random digit dial methodology[17]: pre-intervention in August 1980 (1200 respondents) and post-intervention in August 1982 (914 respondents). The primary criterion for selection into the sample was that at least one child 19 years old or younger lived in the household. The surveys included 95 items that assessed a household's exposure to an intervention (e.g., home visited by a housing inspector), and self-reported safety behavior and knowledge.

In addition, prevention behaviors were measured. We grouped responses to behavior-related questions on the survey to form an index of prevention behavior for each of three injury causes: poisonings (14 items), burns (12 items), and falls (6 items). A committee of injury prevention professionals assigned weights to items within each index and adjusted the weights to produce a scale score of 0–100 points[18] (Appendix B). We used the safety scores as outcome measures. A question concerning the use of auto passenger restraints by children was analyzed as a separate variable.

We used parental responses to questions about exposure to each injury prevention intervention to classify households into one of three hierarchical levels: 1) no exposure; 2) community exposure, i.e., the family received general injury prevention information, such as pamphlets, from a community source; or 3) participatory exposure, i.e., the family received specific intervention materials, direct person-to-person counseling, or modifications of a household hazard from at least one SCIPP-funded or related project. We classified households by their greatest degree of exposure to any one intervention. Participatory exposures could have been provided by our own projects or those of other community agencies.

Injury Surveillance: The sampling plan and operational characteristics of the injury surveillance system have been described elsewhere.[7] Briefly, we defined cases as children and adolescents (ages 0–19 years) who resided in one of the 14 communities and who had an injury that required medical treatment at an emergency room, admission to a hospital, or resulted in death. We recorded injury cases at 23 hospitals that provided care for an estimated 93 percent of all pediatric injuries in the population.

The five target injuries for intervention efforts were motor vehicle occupants; poisonings due to medicinals and household products and plants; burns; falls from stairs, ladders, other building structures and furniture; and suffocation from objects.

Statistical Analysis

Changes in Knowledge and Practices: We assessed differences between pre- and post-intervention telephone survey responses for the intervention and control communities by means of the safety scores for burns, poisonings, and falls in the home and the responses to the motor vehicle restraint behavior question. Since the two surveys consisted of different respondents, we conducted a two-factor analysis of covariance (ANCOVA) with socioeconomic status (SES) as the covariate, using the SAS PC computer statistical package.[19] Green's SES score constituted the measure of SES in this analysis.[20] The two factors were the community effect (control vs intervention sites) and the time effect (pre- vs post-intervention). We construed an interaction effect between the two main effects as evidence of differential patterns of safety by community type and time of measurement. A statistically significant interaction would need further investigation with use of post hoc comparisons to determine whether the pattern of interaction revealed an impact of the prevention programs in the study communities.

Effects of Exposure on Knowledge and Practices: We used the nature of exposure (no exposure, community exposure, and participatory exposure) as the main factor in a one-factor ANCOVA with SES as the covariate; the safety indices for poison, burns, and falls in the home were the dependent variables. For motor vehicle occupant behavior, we evaluated the association between nature of exposure (exposed or not exposed) and response to the survey question about child use of auto restraints by chi-square and logistic regression that adjusted for SES.

Changes in Injury Incidence: We compared age-specific injury rates in the pre-intervention period (approximately one year) to the during-intervention period (approximately 22 months) in the control and intervention communities. The post-intervention period was too brief to analyze. We computed Cochran-Mantel-Haenszel (CMH)[21] statistics to test the association between the rate changes (pre- to during intervention) and community type (control or intervention) with control for SES. The measure of SES used in this analysis was median family income of the community, which we stratified into three levels to achieve groups of roughly similar size: less than $20,000, $20,000–25,000, and more than $25,000. Within each combination of age group and injury type, we established a 2 × 2 contingency table.

Results

Exposure to Interventions

During the 22-month period of intensive intervention in the nine intervention communities, direct counts of participation showed that an estimated 42 percent of households with children ages 0–5 years participated in the activities of the SCIPP prevention projects. Pediatric counseling, which included a strong child auto seat component, reached 21 percent of households with children ages 0–5 years; child auto passenger safety programs reached 17 percent; Project Burn Prevention in the community reached 10 percent; home hazard inspection reached 5 percent; and the poison control system reached 1 percent.

We obtained completed telephone surveys for 1,200 households in the pre-intervention survey and 914 in the post-intervention survey; in all we reached the mothers of 4,351 children ages 0–19 years, about 5 percent of all children in the 14 communities. Success in completion of surveys for eligible telephone numbers varied from 59 percent in the pre-intervention survey, when we made only five call-backs, to 85 percent in the post-survey when we made up to 10 call-backs, but did not differ between intervention and control communities. The refusal rate for eligible households was 9.8 percent in the pre-intervention survey and 6.0 percent in the post-survey. The demographic characteristics of the interviewee samples were similar. For the purposes of this evaluation, we used only those households with children ages 0–5 years.

Table 1 shows that even prior to the beginning of the program, households in both study and control communities were exposed to childhood safety messages by a variety of means. We found no significant differences in pre-intervention safety exposures between intervention and control communities.

Over the course of the intervention, the level of participatory exposure for households with children 0–5 years increased three-fold in the intervention communities and two-fold in the controls. Overall, intervention communities' had a higher percentage of participatory exposure than control communities, 62.4 percent vs 38.2 percent (95% confidence intervals of difference 13.9, 34.5). Similar differences hold for participatory exposure in all five target injuries (Table 1).

Changes in Safety Knowledge and Practices

The SES-adjusted mean safety scores for burns- and falls-related behaviors increased from the pre- to post-intervention survey to the same extent in both the intervention and control communities (Table 2) The poisoning safety index showed some effect of exposure. We found no interaction effects for any of these indices.

Effects of Exposure to Interventions on Knowledge and Practices

We found an association between level of participation and safety behaviors for burns and poisoning but not for falls (Table 2).

For the auto restraint use variable, we evaluated only two exposure groups (exposed and non-exposed). Reported use of child auto restraints was 22.2 percent–higher in households that had exposure to prevention programs than in households not so exposed (Table 2). To hold the effects of SES constant, we computed the logistic regression of the probability of reporting restraint use on exposure status and SES. The coefficient for exposure status was 0.47 ($\chi^2 = 31.2$, $p = 0.0001$). With employment of methods commonly used in econometrics to calculate the first derivatives of the logistic regression function,[22] the difference between exposed and non-exposed households in reported child restraint use adjusted for SES reduces to 11.6 percent.

Changes in Injury Incidence

Figure 1 shows the pattern of injury incidence rates for target injuries sustained by children ages 0–5 years in the intervention and control communities over the three-year period. At every point, the incidence rate for intervention communities is lower than that for controls. This difference reflects the difference in SES between the intervention and control communities and disappears with adjustment of the rates for SES.

The crude incidence rates for specific target injuries for children ages 0–5 years appear in Table 3. The overall incidence for all target injuries increased in both intervention and control communities from the pre- to the during-intervention period. In the intervention communities, however, the incidence of motor vehicle occupant injuries decreased 54 percent, while rates rose in the control communities. With control of SES, the odds in favor of a motor vehicle occupant injury in the pre-intervention period versus the during-intervention for the intervention communities was 2.78 times as large as that for the control communities.

Discussion

The principal positive finding of this study is a distinct reduction in motor vehicle occupant injuries among children

TABLE 1—Exposure of Households with Children Ages 0–5 Years to any Injury Prevention Program as Measured by Responses to Pre- and Post-Intervention Telephone Surveys

		Community Type and Time			
		Intervention Communities		Control Communities	
Target Injury	Exposure	Pre-	Post-	Pre-	Post-
		Number			
		230	184	256	157
		Percent			
Motor Vehicle Occupant	Participatory	19.1	55.0	14.1	34.4
	None	80.9	44.9	86.0	65.6
Burns	Participatory	13.0	58.7	13.3	29.3
	Community	10.4	13.8	7.4	22.9
	None	76.5	27.5	79.3	47.8
Falls	Participatory	12.2	54.5	10.2	28.7
	None	87.8	45.5	89.8	71.3
Poisonings	Participatory	13.5	55.6	13.3	32.5
	Community	77.0	41.3	75.8	60.5
	None	9.6	3.6	10.9	7.0
Any Target Injury*	Participatory	20.4	62.4	19.9	38.2
	Community	71.7	34.4	70.7	57.3
	None	7.8	3.2	9.4	4.5

*Unduplicated count of exposure to any one of the intervention programs.

TABLE 2—Changes in Safety Knowledge and Behaviors for Households with Children Ages 0–5 by Community Type and Time and by Level of Exposure to Intervention

Target Knowledge Behaviors	By Community Type and Time				By Level of Exposure in Intervention and Control Combination		
	Intervention Communities		Control Communities				
	Pre-	Post-	Pre-	Post-	Participatory	Community	None
	Percent Using Restraints						
Child auto restraint use[b]	49.1	65.0	49.6	63.3	70.9	—	48.7
	Adjusted Mean Safety Scores[a]						
Burns	44.6	49.2	43.7	46.8	49.6	46.8	43.8
Falls[b]	28.4	30.3	29.7	30.7	31.2	—	29.2
Poisonings	31.9	34.3	27.2	30.5	36.0	29.4	23.8

a) Mean safety score adjusted for SES; possible score is 0–100.
b) Single level of exposure to falls and child auto restraint interventions.

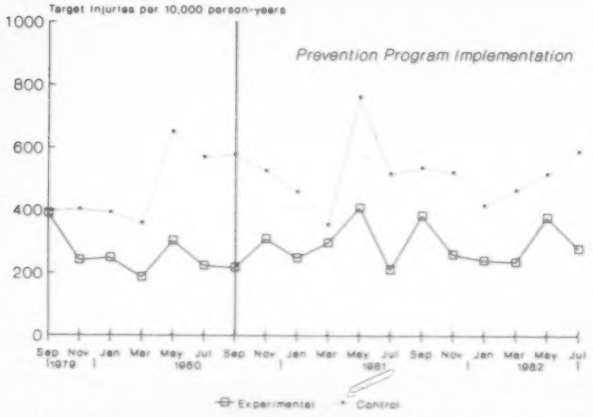

FIGURE 1—Injury Incidence Rates for Target Injuries for Children Ages 0–5 Years in Experimental and Control Communities by 60-Day Periods before and during Injury Prevention Program Implementation

ages 0–5 years in the intervention compared with control communities. This finding is associated with evidence that about 55 percent of households with children ages 0–5 years participated in the interventions. Because many factors may have influenced the use of child auto restraints, however, the observed decrease in motor vehicle occupant injuries cannot be attributed to SCIPP alone. We have no evidence that the coordinated intervention programs reduced the other target injuries—although exposure to prevention messages was associated with safety behaviors for burns and poisonings. These findings suggest three major issues: the design of injury prevention evaluations, the relationship between injury incidence and preventive efforts that alter safety knowledge and behaviors, and the efficacy of efforts to reduce childhood motor vehicle occupant injuries.

The study was designed with the community as the level of intervention and analysis. Because it was impractical to randomize communities, we attempted to match communities on socioeconomic and demographic characteristics with use of the most currently available census data, 1970; injury morbidity rates were unavailable. By 1980, however, the control communities differed demographically from the intervention communities, and had higher proportions of poor and Hispanic households. This demographic change was reflected in higher baseline injury rates in the control communities, a finding which is in itself important. We addressed this problem by controlling for SES in all analyses.

Exposure to injury prevention messages occurred from sources other than our project. The media promoted childhood safety as did the pediatric and public health literature[23–25]; schools introduced safety curricula into their health education programs; the Massachusetts Legislature was debating a child auto restraint bill which, no doubt, led some motivated families to purchase these devices. This exposure of control sites introduced an element of misclassification into the comparison of intervention and control communities. It would have been impossible, however, to have eliminated these sources; estimating the magnitude of their effect on the findings is indeed difficult.

Evaluation of the effect of an intervention depends on the case definition of an injury and the method of ascertainment. We used an emergency room visit as the definition of the least severe injury. It is possible that some of our prevention messages may have altered the level of injury severity and use of the emergency room. For example, knowledge of the use of cold water following a scald burn might have reduced sufficiently the severity of burns so as to avoid hospitalization. Moreover, differential access to treatment by area of residence and socioeconomic status may have influenced ascertainment of cases.

High baseline levels of safety behaviors influenced our ability to show community effects. For example, our pre-intervention survey found that 73 percent and 69 percent of households in study and control communities, respectively, had smoke detectors. At the end of the study period, this level had changed to 82 percent and 84 percent, respectively. Given the low incidence of burns associated with house fires, the study did not have adequate power to evaluate this change. In other cases, the level of exposure to an intervention may have been too low to demonstrate an effect. For example, household inspection and correction of hazards is a highly efficacious intervention,[9] but we reached only 5 percent of all target households in this trial. These design considerations emphasize the importance of using injury morbidity rates by cause at the local level to influence the selection of community sites, target injuries, and intervention strategies.

Finally, the study design did not permit us to disaggregate the changes in knowledge and behavior by the type of intervention strategy. We cannot comment on the relative efficacy of patient counseling by pediatricians, school edu-

TABLE 3—Changes in Target Injury Rates for Children Ages 0–5 Years in Intervention and Control Communities

Target Injuries	Injury Rates[a]				Mantel-Haenszel Odds Ratio[b]	
	Intervention		Control			
	Pre-	During	Pre-	During	OR	95% CI[b]
Motor Vehicle Occupant	46.54	21.54	44.53	60.77	2.78	1.66,4.66
Burns	52.56	59.68	75.01	106.03	1.26	0.84,1.90
Falls	127.07	175.02	252.03	262.44	0.78	0.61,1.00
Poisonings	31.76	36.14	92.71	92.71	0.95	0.57,1.58
Total target[c]	262.80	297.11	477.42	523.96	0.99	0.83,1.19

a) Injury Rates: per 10,000 Children per year using 1980 US census data.
b) Controlling for SES.
c)Total target injuries include a small number of suffocations on objects other than food.

cational curricula, community promotion, or home inspection as the technique to best influence behavior. Different intervention techniques will likely have different effects, and these need further study.

We do know that there were several components to the efforts to reduce motor vehicle occupant injuries. These included promotion by pediatricians of infant and child auto restraint utilization, an in-hospital training program for health professionals and car seat loan programs, establishment of a Child Auto Passenger Safety Resource Center at the state health department with community presentations for parents and providers at preschools and other settings, and statewide advocacy for a mandatory child restraint law which was passed in December 1981. Participation in these activities combined with media attention may have increased safety behavior in the intervention communities as compared to the non-intervention sites. There is good evidence from studies of cardiovascular disease control programs that combined mass media and comprehensive community-based interventions can succeed in improving a community's health status.[26–28]

There is good reason to believe that the prevention technologies promoted through our educational efforts were of differing levels of efficacy. Thus, while we may have exposed our population to all the injury-specific messages equally, we observed a protective effect on auto occupant injuries, primarily, because the child auto restraint device is a highly efficacious technology, and auto crashes occur with sufficient frequency so that one can measure an effect.

Length of exposure to injury prevention efforts may be important to the success of community efforts such as SCIPP. We maintained the interventions and surveillance system for only 22 months because of limited resources. The benefits of changed safety knowledge and practices may manifest themselves more slowly and over a longer time frame. Schelp, in a community-based intervention in Sweden, showed decreases in injury rates after four years of activity.[29] In retrospect, our study was grossly underfunded given its ambitious nature.

Finally, we must acknowledge constraints in implementing "text-book" interventions in a real world setting. We cannot understate the logistics and difficulty of managing a set of complex interventions in several communities simultaneously. Our efforts to persuade local health agencies and community boards to take more of a lead role were thwarted by the unavailability of local morbidity data, a lack of financial incentives, and a lack of media attention to the injury problem. It may be that child auto injury was the only well-recognized injury issue.

In designing our project, we defined childhood injuries broadly and selected prevention methods from those available to us in 1979, when we had little evaluation data available. For example, the results of the evaluation of Project Burn Prevention were not available until 1982, when our intervention was nearly completed.[4] Our target injuries were limited by the availability of these interventions. Were we to redesign the SCIPP intervention today, we would look carefully at all the effective interventions discussed by Rivara[30] and others[31,*] and select only those that have strong evidence of efficacy. Future interventions must include the active participation of local public health agencies, a media component to increase the awareness of both the public and professionals, and evaluation over a longer time period. One must convince these local agencies of the importance of the injury problem, obtain their commitment of resources, and assure that the requisite local morbidity data are available for targeting injuries which are prominent in the community and for evaluation of the interventions.

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APPENDIX A

Injury Prevention Intervention

Intervention	Objectives	Targets	Components
Child Auto Passenger Safety Project	• To increase utilization of infant and child auto restraints • To reduce motor vehicle occupant injuries to children ages 0–5 years.	• Parents of newborns and young children • Health providers with access to maternity patients • Hospitals	• Survey of current hospital practices • In-hospital training of maternity and child birth educators • Resource Center for technical assistance • Promote car seat loan programs • Outreach to preschools • Observational study of restraint usage • Passage of mandatory child restraint law
Home Injury Prevention Project	• To reduce the number of hazards in the home environment • To incorporate injury prevention into traditional role of health agents • To reduce burns, poisonings, falls, and product-related injuries	• Families with children under age 5 years • High-risk housing • Landlords and homeowners	• Systematic enforcement of State Sanitary Code • Safety counseling to hazardous household practices • Distribution and installation of safety devices • Follow-up evaluation
Pediatric Accident Prevention Project	• To provide a developmentally-oriented, focused counseling system for pediatric primary care settings • To increase awareness and safety practices in parents of young children • To reduce burns, poisonings, motor vehicle injuries, falls, choking, and drowning	• Parents of children under age 5 years • Pediatricians • Well-baby clinics • Visiting nurses	• Age appropriate safety surveys • Counseling by health providers • Free safety supplies • Follow-up home assessment/evaluation
Poison Control System	• To promote appropriate treatment of the poisoned patient • To promote awareness and proper storage of poisonous substances	• Parents and older siblings of children under age 5 years • Hospital staff • Pharmacists Other retailers	• Promote emergency consultation care of poisoned patient • Educate health professionals • Promote poison information hotline • Promote availability of Ipecac • Mail surveys and site visits to pharmacies and other retailers • Free distribution of Ipecac displays, phone stickers and pamphlets
Project Burn Prevention	• To institute a burn prevention curriculum in schools • To increase awareness of burn hazards, escape from house fires, and clothing ignition • To reduce all causes of burns	• Parents of young children under age 5 years • School-age (4–18) children enrolled in public and private schools • Community groups	• Poster contest for grades K–2 • Train teachers in curriculum use • Free multi-media kits • Train volunteers • Promotion through libraries, fire & police, day care centers, civic organizations and youth groups • Follow-up evaluation

APPENDIX B

Telephone Survey Questions and Weights Used to Calculate Safety Indices for Burns, Poisonings, and Falls

Telephone Survey Questions	Weight
Poison Safety Index	
1. Store drugs/pain relievers on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	2.6
2. Store drugs/pain relievers on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	20.8
3. Store drain cleaners on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	5.2
4. Store drain cleaners on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	22.0
5. Store drain cleans in the original container?	4.2
6. Store liquid bleach on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	2.0
7. Store liquid bleach on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	2.0
8. Never left bleach in a measuring cup?	2.2
9. Use kindergard latches on the cabinets?	2.8
10. The first response to poisoning is call poison control center for advice?	15.4
11. The first response to poisoning is call doctor/nurse for advice?	6.8
12. Have ipecac Syrup at home?	9.6
13. Have poison phone sticker posted and the number is 232-2120 or 1-800-682-9211?	2.4
14. Have poison phone sticker posted and the number *is other than* 232-2120 or 1-800-682-9211?	2.0
Total:	100.0
Burn Safety Index	
1. None of the cord of the electrical appliance hangs below the counter?	3.5
2. The best thing to put on burn is cool water?	8.6
3. Have a smoke detector in the building?	18.4
4. Have smoke detector installed in the bedroom hallway, or the basement, or all rooms?	14.6
5. Family has fire escape plan?	4.4
6. Family has tried fire escape plan?	5.6
7. Have safety plugs on electric outlets (yes/no question)?	2.0
8. Have safety plugs on *all* the outlets?	2.2
9. Have safety plugs on *some* of the outlets?	2.1
10. Do not have additional heat source at home?	3.7
11. The hot water temperature at home is 125 or below?	32.6
12. The fuses at home do not blow frequently?	2.0
Total:	99.7
Fall Safety Index	
1. Do not have broken window?	3.3
2. There are screens or barriers on window?	7.8
3. Have safety gates at either the top or bottom of the stairs?	26.3
4. Have safety gates at both the top and bottom of the stairs?	45.0
5. All the stairways in the building have railings?	14.3
6. None of the railings are unsteady?	3.3
Total:	100.0

Human Cryptosporidiosis Associated with an Epizootic in Calves

John S. Reif, DVM, MSc(Med), Lynne Wimmer, DVM, John A. Smith, DVM, MS, David A. Dargatz, DVM, MS, and John M. Cheney, DVM, MS

Abstract: An outbreak of human cryptosporidiosis occurred among previously healthy persons in a veterinary medical teaching hospital. Human illness began after admission of calves from a farm which had been experiencing an epizootic of neonatal diarrhea due to *Cryptosporidium*. The clinical syndrome in humans was characterized by watery diarrhea, abdominal cramping, flatulence, and headache. Cryptosporidiosis was confirmed by zinc sulfate flotation of fecal specimens in four persons, three of whom had been responsible for the care and treatment of infected calves. A fourth patient had washed her husband's soiled clothing and appeared to have been infected indirectly through fomite contamination. Among 112 persons surveyed, 26 (23.2 percent) had a diarrheal illness during the outbreak and nine met the case definition of a diarrheal illness lasting at least three days. These persons were more likely to have had contact with a calf with diarrhea than were 52 referents who did not become ill during the outbreak. (*Am J Public Health* 1989; 79:1528–1530.)

Introduction

Cryptosporidium is an intestinal protozoan parasite, recognized relatively recently as a human pathogen,[1] despite its original identification over 70 years earlier.[2] Following the description of the first human case of cryptosporidiosis in 1976, the disease was reported rarely, and primarily in immunocompromised persons.[3] Since 1982, the number of reported cases has increased dramatically.[4] Initially this increase was noted in patients with acquired immunodeficiency syndrome[5] but recent reports indicate that cryptosporidiosis also occurs in immunologically normal persons.[6–8] In immunocompetent persons, cryptosporidiosis is manifested as an acute, self-limiting diarrheal illness lasting five to six days.[4,6–8] Cases of cryptosporidiosis have been described in travelers,[9] residents of day care centers,[10] animal handlers,[8,11] veterinary students,[12–14] and following consumption of contaminated community water supplies[15] or surface waters.[16] A previous description of an outbreak of cryptosporidiosis among five veterinary medical students[13] was ascribed to contact with experimentally infected calves. Recently, infection of at least 10 students who had had contact with 1-to-3-day-old calves in a laboratory setting was described.[14] We report here an outbreak of cryptosporidiosis among previously healthy veterinary students associated with a spontaneous epizootic of bovine cryptosporidiosis in a clinical setting.

Methods

Outbreak in Humans

The index case was a 34-year-old female veterinary student whose duties included nursing care, sample collection, and treatment of calves with diarrhea which were subsequently identified as infected with cryptosporidia. On April 6, 1987, one week after her initial contact with the calves, she complained of watery diarrhea, abdominal cramps, nausea, fever (greater than 99°F), headache, vomiting and flatulence; she was ill for two weeks. She received medical attention and was found to have cryptosporidiosis after parasitologic examination of her stools.

During the next two weeks, several additional students and faculty members complained of gastrointestinal symptoms. A large proportion of these persons were associated with the food animal services. When cryptosporidia were identified in calves hospitalized in the isolation unit, a potential relationship between the animal and human diarrheal illnesses became apparent.

An investigation was initiated on April 17, 1987. A questionnaire distributed to all students and food animal hospital staff included items on recent gastrointestinal illness, and exposure to potential risk factors in the hospital and at home. Dates of potential exposure to calves in the isolation unit were determined from student duty rosters. Persons experiencing diarrheal illness and others with food animal contact were asked to submit stool samples.

Parasitologic examination for cryptosporidia oocysts consisted of fecal flotation by centrifugation of zinc-sulfate (sp. gr. 1.18) to which aqueous iodine had been added.[17] Cover slips from these flotations were examined by light and phase contrast microscopy. Fecal smears were examined after methanol fixation and Ziehl-Nielsen staining.[18] Analysis of risk for specific exposures was accomplished by calculation of odds ratios (OR) for persons experiencing a diarrheal illness, using those who remained clinically normal as the comparison group.[19] Confidence intervals (95% CI) for these risk estimates were obtained by exact testing.[20]

Epizootic in Calves

An outbreak of diarrheal illness in beef calves occurred on a farm in northern Colorado. Virtually all of the 91 calves born after February 1987 developed postnatal diarrhea. Newborn calves continued to develop diarrhea during the first week of life as the calving season progressed. Due to the continuing nature of the diarrheal disorder, four newborn calves with severe diarrhea and dehydration were admitted to the Veterinary Teaching Hospital on March 23, 1987 and placed in the isolation unit. Additional affected calves were admitted weekly until June 6. On April 16, 1987 a fecal flotation performed on one of the inpatient calves revealed the presence of cryptosporidia oocysts. Upon testing of additional inpatient calves, 6/6 (100 percent) were positive for *Cryptosporidium*. Among 20 calves tested which remained on the farm, 17 (85 percent) were positive. Other potential enteric pathogens identified in calves from this farm included

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Campylobacter, *E. coli*, bovine coronavirus, and bovine rotavirus.

Results

Diarrhea occurring between March 16 and April 26, 1987 was reported by 26 of 112 questionnaire respondents (Table 1). With the exception of one unconfirmed illness, all cases were clustered in the three-week period between April 6 and April 26, 1987. In order to attempt to exclude short-lived illness due to viral and bacterial enteric pathogens and dietary upset, a case definition of diarrheal illness lasting at least three days was adopted. Nine respondents (7.4 percent) met this criterion and were defined as cases in subsequent analyses. Cryptosporidiosis was confirmed by identifying oocysts in stool specimens from four persons among six who reported diarrhea. Conversely, cryptosporidia were not detected in any of nine other individuals without a history of recent diarrhea.

Fourth year students had more frequent direct contact with diarrheic calves, adult cattle, and other animals than third year students during the outbreak. Three of the confirmed cases were fourth year students who had responsibility for therapy of the diarrheic calves in the isolation unit between April 1 and the date of diagnosis. The fourth was a female hospital employee whose spouse was a student. She denied contact with calves in the isolation unit. However, her husband had contact with the calves during the weeks of April 6 and 13 and she had washed his fecally contaminated clothing. She became ill during the week of April 13, complaining of a mucoid and watery stool, abdominal cramps, fever, nausea, vomiting, and flatulence.

A watery stool was reported by all patients. Abdominal cramping, flatulence, and headache were predominant features of the clinical disorder. The symptoms reported by persons with parasitologically confirmed cryptosporidiosis were generally similar to those reported by the unconfirmed cases.* The mean duration of illness in confirmed cases was seven days; the illness lasted over two weeks in one person.

The association between personal and occupational risk factors and the occurrence of gastrointestinal illness during the outbreak was examined.* The reference group comprised all fourth year student and staff respondents without a history of gastrointestinal illness (N = 52). Odds ratios of 13.2 and 11.8 were found for contact with a calf with diarrhea and having taken a calf's temperature, while intubating a calf for anesthesia carried no excess risk. The exact confidence intervals around these risk estimates were wide due to the small number of persons affected but did not include one. Contact with dogs, cats, or foals with diarrhea did not increase risk.

No association was found between human gastrointestinal illness during the outbreak and having a child in a day care center, or consumption of untreated surface water. The community is served by a municipal water system which uses complete conventional treatment consisting of chlorination, alum and polyelectrolyte coagulation and flocculation and rapid sand filtration through multimedia filters. Cases were not more likely than members of the comparison group to have had another illness, a history of frequent diarrhea or blood transfusion, or to have taken oral antibiotics during the previous month. A high proportion of both affected and unaffected students claimed to be under physical or psychological stress.

*Complete data available on request to authors.

TABLE 1—Frequency of Symptoms, Duration of Illness and Isolation of *Cryptosporidium* during Outbreak of Gastrointestinal Illness in a Veterinary Teaching Hospital, March 16 to April 26, 1987

	Fourth Year Students	Third Year Students	Hospital Personnel	Total
Persons Responding	45	47	20	112
Persons with Diarrhea	10	13	3	26
Persons with Symptoms ≥3 Days	8	0	2	9
Confirmed Cases	3	0	1	4

Discussion

The outbreak of cryptosporidiosis reported here reemphasizes the importance of this organism as a cause of gastrointestinal illness in previously healthy, immunocompetent persons.[6–8,15,16,21,22] Travelers, residents of day care centers, homosexual males, and children in developing countries constitute important risk groups for this cosmopolitan parasite.[23]

In the current outbreak, calves served as the source of human infection. This occurred despite the facts that veterinary students have frequent contact with diarrheic patients of many species, and that *Cryptosporidium* is reported to display little, if any, host specificity.[4] However, it is not clear if *Cryptosporidium* oocysts from animals other than cattle are infectious for humans. Previous reports of cryptosporidiosis in veterinary medical students have also involved contact with calves. In one, a student who provided supportive care of two calves with cryptosporidiosis became ill,[12] and in another, five students caring for experimentally infected newborn calves were affected.[13] In a recent report,[14] *Cryptosporidium* was detected in 10 of 20 students who had contact with 1-to-3-day-old calves in a student laboratory. The incubation period ranged from five to 14 days after exposure and the diarrhea lasted 30 hours to 16 days. Two infected students had only nausea or headache, suggesting that unrecognized infections may occur.

Reports of cryptosporidiosis in research workers[11,24] and animal handlers[8,24] with direct contact with feces from experimentally and naturally infected calves add to the evidence that the young calf is an important source of human infection. The reason for the consistent role of the calf as a source of cryptosporidiosis for humans is not clear, but may be related to the high prevalence of infection among calves,[25] and shedding of large numbers of oocysts for prolonged periods.[26] The preeminence of diarrheic calves as a source of transmission for zoonotic cryptosporidiosis dictates the necessity for exposed persons to adopt stringent hygienic precautions including protective clothing when working in an environment potentially contaminated with *Cryptosporidium*.

Fomites appear to constitute a hazard for the indirect transmission of *Cryptosporidium*. In the current outbreak, one confirmed human case had contact with clothing soiled with fecal material from infected calves as the likely source of infection. The oocysts of *Cryptosporidium* are resistant to desiccation and several disinfectants[27] and are thus likely to persist in the environment. Evidence that waterborne transmission occurs supports this hypothesis.[15,16]

The outbreak began approximately two weeks after the first calves were admitted, but the status of shedding of *Cryptosporidium* by specific animals was not determined until after the onset of the epidemic in humans. Instituting hygienic measures and awareness of the existence of an epidemic of gastrointestinal illness among students with contact with calves may have contributed to the cessation of clinical cases after the week of April 20, despite the fact that calves continued to be admitted to the hospital from the farm in question.

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Public Health Service Implementation Plan for Responding to Nursing Shortage

The US Public Health Service has developed an implementation plan in response to the recommendations of the Secretary's Commission on Nursing, according to O. Marie Henry, RN, DrPH, PHS Chief Nurse Officer. Reporting recently to the Advisory Council on Nurses Education of the Health Resources and Services Administration, in Rockville, MD, Dr. Henry indicated that the plan is intended to coordinate activities and resources among PHS agencies and establish a focus within the US Department of Health and Human Services for responding to the nursing shortage.

In general, the plan continues current activities in such areas as research, financial assistance, and program development, within available resources, and also establishes new initiatives. The plan is organized around nine major objectives in the general categories of resource development, maintenance, and utilization, Dr. Henry said.

Copies of the 16-page "Public Health Service Implementation Plan for the Secretary's Commission on Nursing Final Report" are available from the Office of the USPHS Chief Nurse Officer, Room 14A-40, 5600 Fishers Lane, Rockville, MD 20857. Tel: (301) 443-0577.

Polymedicine Use among Community Resident Older Women: How Much of a Problem?

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Abstract: In a community-based study of older women describing the prevalence of specific medication combinations, the risk of deleterious consequence from polymedicine was concentrated in a minority of older women. There were few prevalent medication combinations, and many of the more common combinations were unavoidable. These results suggest that broad, community-based drug education programs may be less effective than those targeting medical providers and multi-drug users. (*Am J Public Health* 1989; 79:1537–1540.)

Introduction

As a group, the elderly suffer a larger burden of illness than younger adults, and consume disproportionately more medication;[1–3] community-based surveys report that about 70 percent of those over 65 years old take at least one medication.[4,5] Estimates of the average number of drugs taken at any one time varies considerably, from about 1.5 drugs to more than four drugs, depending on the age distribution of the sample, and whether those reporting no drug use were included in the average.[4,5] Moreover, age-related physiologic changes may increase the likelihood of adverse drug effects. Hence, older persons are thought to be at increased risk for the deleterious consequences of "polymedicine."[6,7] The size of this risk depends on the prevalence of specific medication combinations, yet little is known about the prevalence of specific medication combinations in the older community population, and none of the available information is specific to women. Women typically use more drugs than their male counterparts, although this association wanes somewhat with age.[8] However, women may be more susceptible than men to adverse drug reactions as they age because they experience larger declines in lean body mass.[9,10]

The purpose of this paper is to describe the prevalence of specific medication combinations in a community resident population of older White women.

Methods

Sampling Design

Study participants included 807 White women aged 65 and older living in a stable, relatively homogeneous urban community in a 20 census tract area in Northeast Baltimore. During 1984, a random sample of 6,540 addresses was enumerated from this community. The enumeration effort had a 95 percent response rate and yielded 1,203 homes occupied by at least one woman over age 65. If a household contained more than one woman over age 65, one was randomly selected to participate. Of the 1,203 eligible subjects, 807 (68 percent) agreed to participate. Details of this survey are available elsewhere.[11]

Prescription Drug Use

We asked respondents if they had taken any prescription medications during the past month and examined the original containers for all medications reported. Name, date of prescription, dose, and reason for taking each medication (using the information on the medication label) were recorded. Bottles were available for 91.6 percent of all prescriptions. Respondents provided information on other cases and, when necessary, dispensing pharmacies were contacted for clarification. Each drug was assigned the appropriate therapeutic drug code, using the American Medical Association's drug classification system.[12] The therapeutic codes were condensed into 10 drug categories. A number of drug categories with very low prevalence in this community dwelling population were omitted from the analysis. These included anti-malarial agents, anti-neoplastic agents, dermatologic compounds, and chelating agents.

Non-Prescription Drug Use

The interviewer also asked respondents if they had taken any non-prescription medications during the week prior to the interview. If the respondent answered affirmatively, the interviewer read the respondent a list of types of non-prescription drugs, including vitamins, laxatives, over-the-counter pain relievers, antacids, sleeping pills, and insulin, asking the respondent which ones she had taken.

Results

The 807 women in this sample report a mean age of 72.7 years, with a range of 65 to 99 years. Nearly 6 percent are over 85 years old. On average the respondents have completed 9.9 years of schooling, although 40 percent had

TABLE 1—Number of Prescription Medications Reported

Number of Medications	Percent of Respondents	
	Prescription	Non-Prescription
0	26.0	23.3
1	21.1	34.0
2	19.8	23.5
3	12.8	12.8
4	9.0	4.4
5	5.2	1.5
6 or more	6.1	0.5

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TABLE 2—Drug Use by Therapeutic Category and Subcategory

Drug Category	Percent Reporting Use
Cardiovascular renal agents	55.8
Digitalis glycosides	11.3
Antiarrhythmic agents	7.0
Antianginal agents	15.0
Agents for peripheral and cerebral vascular disorders	2.5
Antihypertensive agents	19.9
Diuretics	36.4
Blood formation, volume, coagulation agents	2.7
Drugs for deficiency anemias	2.4
Anticoagulants	0.4
Homeostatic, nutrient agents	10.8
Agents for hyperlipidemia	0.4
Vitamins/minerals	1.2
Replenishers and regulators of water, electrolytes and nutrients	9.1
Agents affecting calcium metabolism	0.2
Drugs used for pain relief	19.5
Strong analgesics	2.4
Mild analgesics	3.6
Agents used to treat migraine	0.1
Agents used to treat gout	1.3
Antirheumatic agents	14.0
Central nervous system drugs	13.8
Sedatives and hypnotics	3.8
Antianxiety agents	6.8
Antipsychotic and antimanic agents	0.7
Anticonvulsants	0.9
Antidepressants	3.8
Hormones	14.6
Adrenal corticosteroids	2.3
Estrogens, progestogens	2.4
Anterior pituitary and hypothalamic agents	0.1
Agents for hyperglycemia	5.7
Agents for thyroid disease	4.6
Respiratory/allergic disorder drugs	5.8
Bronchodialators and antiasthmatics	2.4
Nasal decongestants	0.4
Antitussive agents	0.1
Antihistamines	2.7
Cold remedies	0.6
Antimicrobial agents	5.2
Penicillins	1.2
Cephalosporins	0.6
Macrolides, lincomycin and clindamycin	0.6
Tetracyclines	0.5
Sulfonamides	0.6
Nitrofurans	0.5
Antifungal agents	0.4
Drugs used in ophthalmology	3.7
Agents used to treat glaucoma	2.6
Mydriatics and cycloplegics	0.1
Ocular anti-inflammatory agents	0.9
Gastrointestinal agents	8.7
Agents used to treat peptic ulcer	2.2
Antidiarrheal agents	1.5
Laxatives	0.4
Antiemetics	0.2
Miscellaneous	3.7

completed less than eight years of schooling and 10 percent had completed at least some college.

Prescription Drug Use

On average, the women in this sample report using 2.02 prescription drugs each, with a range of 0 to 11 drugs. The distribution of prescription medication use is presented in Table 1. Over one-fourth report using no prescription medications, while nearly 20 percent report using four or more prescription medications. Table 2 breaks down drug use by therapeutic category, and by class within category.

TABLE 3—Percent of Respondents Reporting Use of Each Category of Non-Prescription Drug

Drug Category	Percent Reporting Use
Vitamins	31.5
Laxatives	18.5
Pain relievers	31.0
Antacids	20.0
Sleeping pills	2.4
Insulin	1.2
Others	18.5

About 9 percent of all prescription medications had label instructions to use "as needed," including: 36 percent of the allergy/respiratory disorder drugs, 20 percent of the gastrointestinal agents, 27 percent of the central nervous system agents, and 21 percent of the drugs used for pain.

Use of Non-Prescription Drugs

The mean number of non-prescription drugs used in the week prior to the interview was 1.4 with a range of 0 to 7 (see Table 1). Twenty-three percent of the sample report taking no non-prescription drugs during the week prior to the interview, while 19 percent report taking three or more non-prescription drugs. Table 3 reports the percent of respondents reporting use of each category of non-prescription drug.

Analysis of Drug Combinations

To describe the potential extent of polymedicine in this sample, we ascertained the number of respondents reporting the use of each possible two-drug category combination by cross-tabulating all the drug category variables. The chi-square test with Yates' correction for 2×2 tables was applied to differences between observed and expected values. Tables 4 thru 6 report the results of this analysis. It is important to remember that the number of cases reported for any drug combination is bounded by the prevalence of the least frequently occurring drug in the combination.

Table 4 presents observed and expected frequencies for combinations of two prescription drugs. Of the 45 potential combinations, eight (18 per cent) occurred with significantly greater than expected frequency. It is noteworthy that about one in eight respondents (109/807) report taking the combination of cardiovascular renal agents and drugs for pain. This is not surprising since 44 percent of the women reported having high blood pressure, and 60 percent reported having arthritis, the two most common indications for use of cardiovascular renal agents and pain medications, respectively, in this sample.

Table 5 reports the observed and expected frequency of respondents reporting two-way combinations of one prescription drug and one non-prescription drug. Of the 60 combinations, five (about 8 percent) occurred with greater than expected frequency. Four combinations of cardiovascular-renal agents and non-prescription drugs have prevalence rates in this sample of over 10 percent. More than 25 percent of the sample (219/807) report using both cardiovascular renal agents and over-the-counter pain relievers. The combinations of cardiovascular renal agents with vitamins, laxatives, and antacids each occur in at least 10 percent of the sample.

Table 6 performs an analysis for two drug combinations of non-prescription drugs. Of the 15 potential combinations,

TABLE 4—Observed and Expected ( ) Frequency of Respondents Reporting Each Possible Two-way Combination of Prescription Drug Category

		2	3	4	5	6	7	8	9	10
1. Cardiovascular Renal Agents	Obs.	16	76**	109**	77**	75	29	24	20	47
	(Exp.)	(12)	(49)	(88)	(62)	(66)	(26)	(23)	(17)	(39)
2. Blood Formation Volume, Coagulation Agents	Obs.		2	8	5	2	2	2	1	4
	(Exp.)		(2)	(4)	(3)	(3)	(1)	(1)	(1)	(2)
3. Hemeostatic and Nutrient Agents	Obs.			22	17	16	6	6	3	12
	(Exp.)			(17)	(12)	(13)	(5)	(5)	(3)	(8)
4. Drugs Used for Pain	Obs.				36**	35**	18**	10	8	15
	(Exp.)				(21)	(23)	(9)	(8)	(6)	(14)
5. Central Nervous System Drugs	Obs.					18	9	9	6	24**
	(Exp.)					(16)	(7)	(6)	(4)	(10)
6. Hormones and Agents Affecting Hormonal Mechanisms	Obs.						9	4	7	8
	(Exp.)						(7)	(6)	(4)	(10)
7. Respiratory and Allergic Disorder Drugs	Obs.							8*	3	7
	(Exp.)							(3)	(2)	(4)
8. Anti-microbials	Obs.								0	6
	(Exp.)								(2)	(4)
9. Opthalmologic Agents	Obs.									4
	(Exp.)									(3)
10. Gastrointestinal Agents	Obs.									—
	(Exp.)									—

*p < .05
**p < .01

four (26 percent) occurred with greater than expected frequency. Two combinations of non-prescription drugs occur in more than 10 percent of the sample: vitamins and laxatives, and over-the-counter pain relievers and antacids. The latter was also reported with greater than expected frequency.

Discussion

This analysis indicates that there is a wide range of drug taking patterns among older women living in the community. While half the sample reported taking no or only one prescription medication, 20 percent reported using four or more prescription drugs. The use of non-prescription medications is equally varied. Hence, community dwelling older women are not homogeneous in their "risk" for the potentially deleterious effects of polymedicine.

An analysis of medication combinations demonstrates few prevalent combinations in this sample of community dwellers. In addition, many of the more prevalent combinations may be therapeutically unavoidable. For example, the combination of cardiovascular renal agents and pain relievers (prescription and non-prescription) occurs with high fre-

TABLE 5—Observed and Expected () Frequency of Respondents Reporting Two-way Combinations of Prescription and Non-Prescription Drugs

		Vitamins	Laxatives	OTC-Pain Relievers	Antacids	Sleeping Pills	Insulin
Cardiovascular Renal Agents	Obs.	123	84	219	96	14	9
	(Exp.)	(142)	(83)	(210)	(90)	(11)	(6)
Blood Formation Volume, Coagulation Agents	Obs.	9	6	14	5	0	0
	(Exp.)	(7)	(4)	(10)	(4)	(1)	(0)
Hemeostatic and Nutrient Agents	Obs.	26	22	40	22	4	0
	(Exp.)	(27)	(16)	(41)	(17)	(2)	(1)
Drugs Used for Pain	Obs.	54	42*	71	42	7	1
	(Exp.)	(50)	(29)	(73)	(31)	(4)	(2)
Central Nervous System Drugs	Obs.	47*	24	60	35*	6	3
	(Exp.)	(35)	(20)	(52)	(22)	(3)	(1)
Hormones and Agents Affecting Hormonal Mechanisms	Obs.	40	24	33	20	32*	4
	(Exp.)	(37)	(22)	(37)	(21)	(27)	(3)
Respiratory and Allergic Disorder Drugs	Obs.	15	9	23	8	1	1
	(Exp.)	(15)	(9)	(22)	(9)	(1)	(1)
Anti-microbials	Obs.	13	8	19	11	1	1
	(Exp.)	(13)	(8)	(20)	(8)	(1)	(1)
Opthalmologic Agents	Obs.	10	10	15	6	0	1
	(Exp.)	(9)	(5)	(15)	(6)	(1)	(0)
Gastrointestinal Agents	Obs.	19	16	30	32*	3	0
	(Exp.)	(22)	(13)	(33)	(14)	(2)	(9)

*p < .05
**p < .01

TABLE 6—Observed and Expected ( ) Frequency of Respondents Reporting Two-way Combinations of Non-Prescription Drugs

		Laxatives	OTC-Pain Relievers	Antacids	Sleeping Pills	Insulin
Vitamins	Obs.	59*	131	64	10	3
	(Exp.)	(47)	(119)	(51)	(6)	(3)
Laxatives	Obs.		80	45**	9*	2
	(Exp.)		(69)	(30)	(4)	(2)
OTC Pain	Obs.			97**	13	6
Relievers	(Exp.)			(75)	(9)	(5)
Antacids	Obs.				5	1
	(Exp.)				(4)	(2)
Sleeping	Obs.					1
Pills	(Exp.)					(0)
Insulin	Obs.					—
	(Exp.)					—

*$p < .05$
**$p < .01$

quency simply because many older women suffer from both hypertension/heart disease and arthritis. Other frequently observed combinations of medications result from the need to temper side effects—the co-occurrence of cardiovascular renal agents and hemeostatic/nutrient agents represents the need to add potassium supplements to diuretic therapy. Similarly, the frequent joint occurrence of over-the-counter pain relievers and antacids may reflect self-medication of the gastrointestinal side effects of aspirin.

Some of the more dangerous combinations fortunately occur with relatively low frequency. Some combinations of central nervous system agents and pain medications could be potentially dangerous. Overall, this combination occurs in about 4.5 percent of our subjects. In addition, six persons using a central nervous system agent and seven persons using a pain medication reported self-medicating with non-prescription sleeping pills.

Several caveats are in order. This sample derives from a community-based population of White women. While representative of a sizable portion of the older population, they can be expected to differ in important ways from other elderly. Treatment-based populations, both inpatient and outpatient, would overrepresent high medication users relative to this sample. Men and persons of other races may also differ from White women in their use of medications. Furthermore, these analyses use prevalence data. It is likely that our reports of drug use underestimate the incidence of drugs used for acute conditions.

Because relatively few older women report using many drugs, this analysis suggests that the risk of deleterious effects from polymedicine is not significant for the majority of community resident older women. A large minority, about 20 percent, are however, at substantial risk. Educational interventions directed toward providers which aim at encouraging appropriate prescribing and monitoring practices when treating older adults are already underway in many areas of the country. Given that this analysis suggests that the risk of deleterious effects of polymedicine is concentrated in a minority of older women, educational interventions should be directed at these community-dwelling, high medication users as well.

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Public Health Briefs

Factors Affecting Place of Death of Hospice and Non-Hospice Cancer Patients

CAROL MCMILLEN MOINPOUR, PHD, AND LINCOLN POLISSAR, PHD

Abstract: We identified factors associated with death at home for 28,828 hospice and non-hospice cancer patients in 13 counties of western Washington State. Hospice participation was found to be the variable most strongly associated with death at home. Admission to hospice appears to override the tendency for certain subgroups of patients, such as the extreme elderly and those diagnosed close to death, to die in an institutional setting. These findings are discussed with respect to the problem of selection bias. (*Am J Public Health* 1989; 79:1549–1551.)

Introduction

Death at home has been associated with availability of hospice programs[1,2] and whether or not the hospice is hospital-based or home care-based.[1,3–5] In addition, variables such as case mix, availability of caregiver support, and federal policies associated with the new prospective payment system and the Medicare hospice benefit are suspected of influencing length of stay in hospitals and hospice[4–14] and may affect place of death. A number of these variables were addressed in this analysis of factors associated with death at home for hospice and non-hospice cancer patients. Hospice participation was predicted to be a major determinant of death at home.

Methods

The study population included all 28,828 residents of 13 Washington State counties who died during 1980–85 with cancer as a primary cause of death, and whose diagnosis was made prior to death. Among this population, 6,762 were admitted to one of the hospices in the area. Cancer diagnosis information was obtained from the Fred Hutchinson Cancer Research Center Cancer Surveillance System and hospice data were obtained through the cooperation of 21 hospices.

We selected two types of independent variables: 1) previously established association with place of death and/or traditional adjustment or controlling variables (age at death, sex, race, marital status, rural/urban nature of county of residence, stage and site of cancer at diagnosis, presence of a contributory cause of death on the death certificate, time from diagnosis to death, and year of death); 2) policy-related variables (type of ownership of the hospital involved in continuing care of the patient, whether or not the hospital was affiliated with or had its own hospice program, whether or not the hospital was affiliated with or had its own home care program, and type of hospice program utilized).

The effect of independent variables on the relation between hospice participation and death at home was measured by a statistic derived from the relative risk.[15] In each stratum of a variable, the "scaled" relative risk was calculated as the stratum-specific relative risk for home death divided by the overall relative risk pooled across all strata. A variable that does not modify the hospice effect on place of death will have a scaled relative risk of 1.0 for all strata.

Logistic regression analyses were based on 26,500 patients with known values for all relevant variables. The dependent variable was coded as dying at home versus dying elsewhere. Independent variables in the logistic regression were modeled as dummy variables.[16]

Results

Hospice patients were 2.8 times as likely to die at home as non-hospice patients, the strongest association with place of death of any variable studied. Patients in hospice Home Health Based 1 and 2 programs were more likely to die at home than other hospice patients (Table 1). Inpatient hospice participants had a frequency of home death that was closer to the frequency for non-hospice than for hospice patients. The association of place of death with hospice participation yielded a p value $<.0005$ whether hospice was categorized as

TABLE 1—Place of Death, among Cancer Patients, by Type of Hospice or Non-Hospice Care

	Place of Death (percent)				
Type of Care	At Home	Hospital	Nursing Home	Other	N
Non-Hospice	16.3	63.6	19.1	1.0	22066
Hospice*	45.3	42.3	10.8	1.6	6762
HH-Based 1	53.2	36.8	8.2	1.8	3794
HH-Based 2	41.3	45.7	11.6	1.2	1642
Inpatient	25.1	56.7	16.7	1.6	1013
Community-Based/Volunteer Intensive	35.8	44.1	18.6	1.3	313
Total N					28828

*HHB1: General home and hospice home care delivered by a Medicare Certified Home Health Agency.
HHB2: Hospice home care only, delivered by a Medicare Certified Home Health Agency.
Inpatient: Hospice care delivered in a hospital setting.
Community-Based/Volunteer Intensive: Hospice care delivered by organizations supported by donations and volunteer services.

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TABLE 2—Percent of Hospice and Non-Hospice Patients Dying at Home; Relative Risk and Scaled Relative Risk for Dying at Home Associated with Hospice By Patient Demographic and Medical Variables (N = 26,828)

Variables	Hospice % (N)	Non-Hospice % (N)	RR	Scaled RR
Age at Death (years)				
0–19	69.2 (26)	25.9 (189)	2.7	1.0
20–64	44.3 (2488)	17.9 (7892)	2.5	.9
65–74	45.0 (2397)	16.4 (6580)	2.7	1.0
75–84	46.0 (1494)	15.4 (5064)	2.7	1.1
85+	49.2 (356)	12.0 (2351)	4.1	1.5
Unknown (N = 1)				
Marital Status				
Single	37.9 (264)	14.2 (1405)	2.7	1.0
Married	47.6 (4400)	18.2 (12694)	2.6	1.0
Divorced	36.4 (623)	13.7 (2159)	2.7	1.0
Widowed	43.4 (1470)	13.6 (5778)	3.2	1.2
Unknown (N = 35)				
Residence				
Rural	39.7 (398)	20.1 (2326)	2.0	.7
Semi-Urban	55.0 (762)	18.7 (3939)	2.9	1.0
Urban	44.4 (5602)	15.1 (15801)	2.9	1.0
Unknown (N=0)				
Time from Diagnosis to Death				
<30 Days	50.0 (70)	5.1 (1228)	9.8	3.6
>30 Days	45.1 (6598)	17.0 (19970)	2.7	1.0
Unknown (962)				
Year of Death				
1980–82	40.4 (2142)	15.4 (11567)	2.6	1.0
1983–85	47.5 (4620)	17.3 (10499)	2.7	1.0
Unknown (N = 0)				
Hospital Ownership				
Local Govt	44.3 (784)	16.4 (3188)	2.7	1.0
Non-Profit	44.8 (5080)	16.8 (14691)	2.7	1.0
For-Profit	45.8 (341)	11.5 (1017)	4.0	1.4
Federal Govt	42.0 (150)	11.3 (1294)	3.7	1.4
Unknown (2283)				

TABLE 3—Relative Risks, Coefficients, and Standard Errors for Death at Home, Linear Logistic Model (n = 26,500)

Variables	Relative Risk	Coefficient	Standard Error
Intercept	–	−1.62	.04
Age at Death (years)			
<65	1.1	0.12	.05
65–74*	1.0	0	–
75–84	1.0	−0.04	.05
85+	0.8	−0.25	.08
Hospice Participation			
Non-Hospice*	1.0	0	–
Home Health-Based 1	5.1	1.63	.06
Home Health-Based 2	3.6	1.28	.07
Inpatient	1.6	0.48	.09
Community-Based	1.9	0.66	.14
Marital Status			
Currently Married*	1.0	0	–
Single	0.7	−0.34	.07
Divorced/Widowed	0.8	−0.27	.04
County Location			
Urban*	1.0	0	–
Semi Urban	1.3	0.24	.04
Rural	1.4	0.32	.05
Time from Diagnosis to Death			
<30 Days	0.3	−1.36	.14
>30 Days*	1.0	0	–
Year of Death			
1980–1982*	1.0	0	–
1983–1985	1.2	0.14	.03
Hospital Ownership			
Non-Profit*	1.0	0	–
Local Government	0.9	−0.07	.04
For-Profit	0.9	−0.09	.08
Federal Government	0.6	−0.46	.08
Interactions			
Hospice × Time from Diagnosis-Death less than 30 days	6.0	1.80	.29
Hospice × Age at Death			
Hospice × <65	0.9	−0.09	.08
Hospice × 75–84	1.1	0.12	.09
Hospice × 85+	1.6	0.49	.14

*Reference Category for Dummy Variables

dichotomous (yes/no), as a five-category variable as in Table 1, or when limited to the four categories of hospice.

The variables that had both a statistically significant direct association with place of death (home versus other) and a significant interaction with hospice in affecting place of death are shown in Table 2. All interactions were significant with $p \leq .005$ except for year of death ($p = .02$).

The positive association of hospice with death at home was stronger for the very old (age 85+), for those not living in rural counties, for those diagnosed close to the time of death (the highest scaled relative risk), and for patients using for-profit and federal government hospitals instead of non-profit and local government hospitals. There was only a minor variation in the hospice association with place of death across categories of marital status and year of death.

A logistic regression model for place of death (home versus other) was fit including all variables from Table 2. The interactions of each of the variables with hospice participation (yes/no) were included in exploratory analyses. Initially 13 constructs (all levels of a variable or levels of interaction) were included. Constructs with a p-value of .05/13 = <.004 were retained in the model.

The final model is presented in Table 3. The main effects and interactions all had p-values below .0001. The pattern of main effect relative risks closely followed those in Table 2. Among the interactions, only that of hospice with age and with time from diagnosis to death remained significant. As in Table 2, the positive association of hospice with death at home modifies the relation between place of death and the two variables, age at death and time from diagnosis to death. Patients age 85 and older, and those diagnosed close to death were more likely to die at home than would be predicted by age at death, time from diagnosis to death, and hospice alone.

Discussion

Hospice participation is clearly the variable most strongly related to whether or not a cancer patient will die at home. It appears that once a patient is admitted to a hospice program, the strong philosophy supporting home deaths overrides the likelihood for patients with certain characteristics to die in hospitals or nursing homes.

Because this is an observational study, it is impossible to determine if the strong association of hospice with home death is causal or if it is due to the selection of hospice by people who prefer to die at home. It is likely that at least some of the association is a causal effect of hospice itself.

The relative risk of death at home for hospice patients varies across type of hospice program. These data are consistent with the results of the National Hospice Study[1,3] where patients cared for by hospices not controlling hospital

beds were almost twice as likely to die at home. It is still the case that, relative to non-hospice patients, hospice patients have a much greater likelihood of dying at home. Further, the effect of hospice also seems to be to counteract factors such as age that reduce the likelihood of death at home.

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The Impact of Media Coverage of Nancy Reagan's Experience on Breast Cancer Screening

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Abstract: In surveys of random samples of women 50 years of age and older residing in two communities in Long Island, New York, both within-subject (cohort) and independent-sample comparisons were made before and after the media announcements of Nancy Reagan's breast cancer. Knowledge of lifetime risk of breast cancer increased significantly only in the cohort comparison, while self-perception of risk did not increase. Small proportions of women surveyed, however, reportedly were influenced to contact a health professional (6–8 percent) and to have their first mammogram (1.5–2 percent) which they attributed directly to Mrs. Reagan's experience with breast cancer. (*Am J Public Health* 1989; 79:1551–1552.)

Introduction

While the breast cancer experiences of public personalities could have an effect on awareness and use of cancer screening tests, no population-based studies have been published on the response to such media events. Black, *et al*,[1] found no evidence for improvement in stage distribution of breast lesions treated in two hospitals in New York City after the public announcements of breast cancer in the wives of the US President and Vice President in 1974. On October 15, 1987, Nancy Reagan's breast cancer was announced and in October–November newspapers and both local and network television reported that the cancer had been detected at an early stage by mammography. We examined the short-term effect of the news about Nancy Reagan's breast cancer experience on the breast cancer awareness and screening practices of women on Long Island.

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Methods

As part of a community-based study,[2] a random sample of 254 female licensed drivers 50 years of age and older residing in two non-adjacent townships on Long Island were mailed a survey in September–October 1987 (time-1), with a $2 token prepayment. This survey included questions about past screening behavior (mammography, breast physicals, and self-examination), concerns about mammography, knowledge of breast cancer risk, and demographic characteristics. After three mailings (including certified mail) the final response rate to the time-1 survey was 68 percent (N = 183), excluding those who had died or moved out of the area. The 173 who had responded prior to the announcement of Mrs. Reagan's surgery in the media were sent a follow-up survey in November–December (time-2). The 142 women responding to both surveys comprise the cohort group. Surveys were also sent in November-December to an independent sample of women randomly selected from the same two Long Island communities (time-2), with a response rate of 71 percent (N = 205). To assess the changes in responses from time-1 to time-2, chi-square was used for the independent samples and a matched-pair or McNemar chi-square test was used for the cohort.

Results

There were no significant differences between these

groups of women in age, education, or income (Table 1). The follow-up surveys at time-2 directly asked women whether they had heard about Nancy Reagan's experience with breast cancer and to what extent their screening behavior had been influenced by that experience. Some 85–88 percent of the women surveyed at time-2 had been exposed to the news of her breast cancer in the month after the event through both newspapers and TV coverage, and the majority of women (57–62 percent) also heard about it on the radio. About 8 percent of the cohort respondents and 6 percent of the independent sample (time-2) respondents reportedly contacted a doctor or other health professional as a result of Mrs. Reagan's experience; none contacted either the American Cancer Society or a hospital.

In the period between the two surveys, 17 of the 142 women in the cohort had mammograms, three of whom had never had a mammogram previously. Among these 17 women, 12 said that their decision was not influenced "at all" by Nancy Reagan's experience, three were "somewhat" influenced, and only one was influenced "a great deal." The latter four included the three women (or 2.1 percent of the 142) who had their first mammogram in this period.

Among the independent sample time-2 respondents, 17 women had a mammogram in the October to December period; five of these women had never had a mammogram previously. Of these 17 women, three who said that their decision was influenced by Mrs. Reagan's experience had never had a mammogram previously (3 of 205 or 1.5 percent).

Changes in proportions of correct responses to knowledge questions were slight but in the direction of improved knowledge. In the cohort at time-2, the proportion of correct responses to the question regarding lifetime risk of breast cancer had increased significantly (Table 2) but there was no increase in the proportion of cohort respondents who considered it "very likely" or "somewhat likely" that they would get breast cancer.

TABLE 1—Selected Characteristics of Cohort and Independent Samples

Characteristics	Cohort (N = 142)	Independent Samples Time-1 (N = 183)	Independent Samples Time-2 (N = 205)
Mean Age (years)	62	63	63
Education Level Completed			
≤High school	63 (45.3)	82 (45.8)	99 (48.8)
1–3 years of college	37 (26.6)	47 (26.3)	57 (28.1)
College graduate+	39 (28.1)	50 (27.9)	47 (23.1)
Annual family income			
≤$15,000	27 (20.3)	39 (24.2)	45 (24.1)
$15,000+	106 (79.7)	122 (75.8)	142 (75.9)

TABLE 2—Percent Changes in Response from Time-1 to Time-2

Variables	Cohort	Independent Samples
Correct answer to what proportion of women will get breast cancer at some time in their lives	+9.2%[a] (p = .049)	+3.5% (p = .412)
Perceives self at risk for getting breast cancer	−5.6%[b] (p = .136)	−1.6% (p = .711)

NOTE: P values in the cohort sample are probabilities (two-tailed) obtained by using the normal approximation to the binomial for analysis of discordant pairs.[9]
[a]McNemar chi-square = 3.89, p < .05.
[b]McNemar chi-square = 2.23, p > .10.

Discussion

This study examined the population-level response to the media attention paid to a public figure's experience with breast cancer. Almost all women over the age of 50 had heard about Nancy Reagan's experience through the media and a very small proportion of the defined population sample was influenced in terms of a behavioral change—i.e., seeing a health professional or having their first mammogram. The slight increase in knowledge of risk was not accompanied by an increased sense of personal susceptibility to breast cancer.

Data from the 1987 Behavioral Risk Factor Surveillance System (BRFSS) analyzed by the Centers for Disease Control[3] showed an absolute increase of 12 percent in screening mammography use between the first two months of 1987 and the last two months of 1987 among women who had a medical checkup in that year. The CDC noted the temporal association with Mrs. Reagan's breast cancer but could not attribute the increase in mammography to this event because the effects of the media coverage of the Nancy Reagan news could not be distinguished from the media attention paid to other cancer control promotions in the same year.

According to the Health Belief Model, a media event can serve as a cue or a trigger for an individual to take a preventive health action but the effectiveness of the external cue depends on a complex host of variables including the perceived benefits of an action, the structural barriers to taking an action, as well as the intensity and duration of the cue.[4] On the basis of communication research, the rapid diminution of the effect of episodic information,[5–7] even if widely diffused (as with Mrs. Reagan's experience), may be overcome by community-based interventions[8] that maintain any changes in knowledge, attitudes, and behavior and that help in removing barriers to screening. The small increase in numbers of women contacting a health professional, and the direct attribution of the adoption of first-time use of mammography to the news of Nancy Reagan's breast cancer by a small number of women found in this study, may provide impetus for further work on the integration of such experiences with intervention programs to encourage preventive health behaviors.

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Is Testicular Cancer Incidence in Blacks Increasing?

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Abstract: Using data collected by the Surveillance, Epidemiology and End Results Program (SEER) from 1973–84, we found no evidence of an increase in the incidence of testicular cancer in Blacks, in contrast to an increase of nearly 33 percent in Whites. (*Am J Public Health* 1989; 79:1553–1554.)

Introduction

In 1985, Sagalowsky and Admire,[1] on the basis of treating four testicular cancer cases in Blacks in the early 1980s compared to none in the previous seven years, hypothesized that the incidence rate of testicular cancer was increasing in Blacks during the 1970s and early 80s. Epidemiologic studies in the past have reported no increase in testicular cancer in Blacks but definite increases in Whites.[2–6] We sought to update earlier studies.

Methods

Data were obtained from the Surveillance, Epidemiology and End Results (SEER) Program of the National Cancer Institute.[7] There were 4,788 cases of testicular cancer in Whites and Blacks identified between January 1, 1973 and December 31, 1984 by the SEER registries, of which greater than 99 percent were histologically confirmed. Testicular tumors were classified into three broad histologic categories using the International Classification of Diseases for Oncology (ICD-O)[8]: seminoma (9060-9064), non-seminoma germ cell (9070-9073, 9080-9083 and 9100-9102) and all other tumors. Population estimates based on US Bureau of the Census estimates were provided by the SEER Program. All rates presented are standardized to the age distribution of the US population in 1980 and expressed per million men per year. Poisson regression was used to evaluate whether the linear slope of the incidence rates by year of diagnosis differed from zero.[9]

Results

Table 1 shows the incidence rates of testicular cancer by year, age, histology and race during 1973–84. In Black men, with a total of 103 cases, there was no evidence of any increase in rate during the interval, for the individual histological categories, for all categories combined, or by age. In contrast, during the same time period, the incidence of testicular cancer in White men increased nearly 33 percent, primarily in age groups below 50 years and in seminomas.

Discussion

In Blacks, no increase in the incidence of testicular cancer was apparent during 1973–84. Data on testicular cancer in US Blacks for earlier decades are limited, and it

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TABLE 1—Incidence* of Testicular Cancer in Blacks and Whites by Age and Histology: Surveillance, Epidemiology and End Results Program, 1973–84

Characteristics	Time Period					Regression Coefficient (SE)**
	1973–75	1976–78	1979–81	1982–84	1973–84	
Histologic Type†						
Black						
All	8.9 (19)††	10.4 (29)	9.0 (28)	8.3 (27)	9.2 (103)	−0.054 (0.088)
Seminoma	6.5 (13)	4.0 (11)	4.0 (12)	5.7 (17)	5.0 (53)	−0.066 (0.119)
Other Germ Cell	1.5 (4)	3.0 (9)	4.3 (14)	2.1 (8)	2.8 (35)	0.100 (0.163)
Other	0.9 (2)	3.4 (9)	0.7 (2)	0.5 (2)	1.4 (15)	−0.316 (0.231)
White						
All	38.3 (857)	42.0 (1087)	47.5 (1289)	51.1 (1452)	45.3 (4685)	0.098 (0.013)
Seminoma	18.7 (420)	20.0 (519)	22.6 (614)	25.9 (741)	22.0 (2294)	0.111 (0.019)
Other Germ Cell	15.9 (358)	17.3 (452)	19.3 (533)	19.9 (567)	18.2 (1910)	0.078 (0.021)
Other	3.7 (79)	4.8 (116)	5.5 (142)	5.4 (144)	4.9 (481)	0.112 (0.041)
Age (years)						
Black						
20–34	17.7 (10)	15.9 (12)	25.5 (22)	16.4 (16)	19.0 (60)	0.019 (0.119)
35–49	8.6 (3)	21.6 (9)	4.4 (2)	11.8 (6)	11.6 (20)	−0.081 (0.202)
50–64	10.8 (3)	6.1 (2)	5.9 (2)	5.8 (2)	7.0 (9)	−0.206 (0.305)
White						
20–34	77.7 (433)	86.2 (579)	104.1 (751)	107.3 (813)	95.1 (2576)	0.113 (0.018)
35–49	54.2 (211)	63.6 (274)	65.1 (293)	75.8 (362)	65.2 (1140)	0.103 (0.027)
50–64	34.6 (121)	28.0 (108)	24.8 (97)	35.5 (138)	30.6 (464)	0.014 (0.043)

*Rate per million men per year.
**Poisson regression coefficient and standard error for linear slope of incidence by year of diagnosis.
†Rates standardized to the age distribution of the 1980 US population.
††Number of cases shown in parentheses.

remains uncertain whether there has been an increase over a longer time period. All of this contrasts to the clear steady rise of testicular cancer incidence in US White men, both during 1973–84 and earlier.

The explanation of this discrepancy in trends lies either in a difference in susceptibility to the agent(s) responsible for the increase seen in Whites and/or to a lower level of exposure to these agents. The similarity of the White:Black incidence ratio for testicular cancer in different populations supports the hypothesis that there are at least some racial differences in susceptibility. In South Africa's Cape district, the rate ratio was 3.3 (Whites compared to "coloured")[10] while the ratio in the US is around 4.5[4–6] Further, the incidence of testicular cancer in Blacks is very similar in different areas of the world.[11–15] However, the presence of such a difference in susceptibility between the races does not preclude the possibility that Whites have been increasingly exposed to a greater extent than Blacks in recent decades to some, as yet unidentified, exposure that predisposes to testicular cancer.

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Self-Report and Medical Record Report Agreement of Selected Medical Conditions in the Elderly

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Abstract: This study assessed agreement between self- and medical record report of medical conditions in an elderly population. Medical charts of 120 participants in a screening program were abstracted, and the questionnaire report of eight major conditions was compared with the medical record. There was substantial or moderate agreement between self-report and medical record report for each condition, although strength of agreement varied by condition. Self-report by elderly individuals compares favorably with medical record report of medical conditions. (*Am J Public Health* 1989; 79:1554–1556.)

Introduction

Information on medical conditions is frequently gathered by questionnaire because a standardized medical examination would be impractical and expensive, but the accuracy of self-reported information is seldom assessed. This is of particular concern in elderly persons, who may be more likely to misreport information than younger individuals. The purpose of this study is to evaluate the agreement between self-report and medical record report of selected medical conditions in elderly individuals.

Methods

Population

Individuals in this study have participated for at least three years in the Florida Geriatric Research Program, a health-screening program located in Dunedin, Florida. This free program is open to all ambulatory residents of Dunedin over 65 years of age; over 45 percent of the age-eligible residents have been enrolled. The program began in July 1975 and screens 2,200 persons each year.

Participants are seen annually for a clinic visit, where they are given a brief medical examination and complete a detailed questionnaire ascertaining previous and present illnesses. Detailed descriptions of this population have been published.[1]

For this analysis, a 10 percent random sample of participants who had their annual clinic visit between July 1, 1985 and December 31, 1985 was selected. These individuals

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were sent a letter requesting permission to review their medical records.

Medical Record Review

Mease Health Care, Inc. (MHC), a private hospital with a large outpatient clinic, serves the Dunedin community. All medical records (both inpatient and outpatient) of MHC were requested for sampled individuals. One of us (TLB) then reviewed each medical record and noted the occurrence and date of first diagnosis of the following conditions: angina pectoris, cancer, cataract, diabetes, fracture, hypertension, myocardial infarction, and stroke. Any non-definitive diagnosis (i.e., possible stroke) was also noted.

Questionnaire Responses

The wording of the questions used is presented in Table 1. Except for cataracts, possible responses to each question are: Yes, No, or Unknown. Possible responses to the question on cataracts are: One Eye, Both Eyes, or None.

Analysis

The medical record diagnosis for each condition (yes or no) was compared to the self-report (yes or no) obtained by questionnaires administered at all previous program visits. Medical record diagnoses after the date of completion of the questionnaires were ignored. We did not assume the medical record was more correct than the self-report. The percent agreement between the two reports, the kappa statistic, and z-scores for kappa were calculated.[2]

Results

In this study, 120 of the 122 randomly selected individuals consented to a medical chart review. The two participants not providing consent had died between their clinic visit and the request for permission to review. Of these 120 persons, 115 had received care at MHC and theoretically had charts available for evaluation; charts were located and reviewed for 68 women and 39 men (total = 107). At their first visit, participants ranged in age from 65 to 90 years ($\bar{x}$ = 71 years). Of the 170 medical diagnoses noted in the medical records, 62 percent were diagnosed after 1975 and 38 percent prior to 1975.

The proportion with a positive response on medical record or self-report, the percent agreement between self-report and medical record report, and kappa are presented (Table 2). Angina pectoris, cataract, fracture, hypertension, and myocardial infarction were somewhat more likely to be noted in the self-report, whereas cancer and stroke were slightly more often noted in the medical record.

TABLE 1—Questionnaire Items Used in Self-Report of Selected Medical Conditions in the Elderly

Condition	Wording of Question
Angina	Have you ever been told by a doctor that you have angina?
Cancer	Have you ever been told that you had cancer and this was confirmed?
Cataract	Have you been told of cataracts in the *following*?
Diabetes	Do you have diabetes mellitus?
Fracture	Have you had any broken bones within the last five years?
Hypertension	Have you ever been told that you have hypertension or high blood pressure?
Myocardial Infarction	Have you had a heart attack, thrombosis, coronary, or myocardial infarction?
Stroke	Have you ever had a stroke?

TABLE 2—Percent Positive Responses by Self-Report and Medical Record Report, Percent Agreement Between Self-Report Medical Record Report and Kappa: Selected Conditions

Condition	N	Positive Report by % Self	% Medical Record	% Agreement	Kappa
Angina	103*	26.2	15.5	85	.57
Cancer (Any)	107	24.3	31.8	89	.72
Cataracts	105*	50.5	34.3	76	.53
Diabetes	107	16.8	16.8	98	.93
Fracture	103*	22.3	13.6	91	.71
Hypertension	107	46.7	32.7	86	.71
Myocardial infarction	107	12.1	8.4	94	.70
Stroke	107	5.6	7.5	98	.85

*Four persons were excluded from these analyses either because a non-definitive diagnosis was listed in the medical chart or unknown was checked on the questionnaire.

The percent agreement between self-report and medical record report ranged from 76 percent for cataract to 98 percent for stroke and diabetes. The overall agreement was 89 percent. There were no differences in agreement by sex or recency of condition. All kappa values had p-values <.001.

Discussion

Public health researchers are concerned about the validity of self-reported information.[3,4] In this study, agreement is high between the self-report and medical record report of common medical conditions. Kappa scores for six of the eight conditions are >0.6, indicating substantial agreement.[5] Kappa values between .40 to .60, seen for the remaining two conditions, are evidence of moderate agreement.[5]

There are several limitations to this study. First, participants were not a representative sample, but volunteers who came to a screening program and may be different from non-participants in their interest and knowledge of health-related matters. Over 45 percent of the age-eligible residents of Dunedin were enrolled, however, and the self-reported prevalence of these conditions is very similar to the prevalence reported in three other community studies of elderly persons.[6]

A second limitation is that the respondents had all participated in this screening program, and participation may have affected their knowledge about their health status. However, elderly individuals in general are more knowledgeable about their health than are younger persons[7]; 80 percent of older Americans visit physicians at least once a year, with the average physician visits = 4.8/year.[8] Whether and to what degree participation in this program would enhance participants' knowledge over and above what they ascertain from physician visits is unknown.

An additional limitation is that the medical records were from the one major hospital/clinic in Dunedin. Participants may have had conditions diagnosed in other medical locations and this information may not be in the reviewed chart. The effect of this type of error would be to lower the agreement between the two sources.

Although overall agreement between self-report and medical record report is good to excellent, there is variability in the percent agreement among the medical conditions. This variability in prevalence by ascertainment source has been reported previously in younger persons,[4,9–13] and probably

results from errors at each source. Self-reported information can be wrong for numerous reasons including a misunderstanding of the diagnosis presented, or the forgetfulness of the individual reporting. Likewise, medical records are not necessarily an accurate source of information.[14–21] Serious non-reporting or misreporting of diagnoses in medical charts has been found in several studies.[16–18,20]

Given the potential sources of error with both methods of ascertaining information, it is impossible to know which assessment comes closest to truth. For most important conditions this is probably not an issue, since self-report is significantly correlated with medical record report.

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Geographic Variation in the Occurrence of Hip Fractures among the Elderly White US Population

W. Edward Bacon, PhD, MPH, Gordon S. Smith, MBChB, MPH, and Susan P. Baker, MPH

Abstract: Geographic variations in hip fracture rates across the nine US Census Divisions were examined using data for elderly Whites from the 1979–85 National Hospital Discharge Survey. Rates varied considerably and were highest in the West North Central Division where rates for females were 50 percent higher than the US rate. Reasons for the geographic variations in hip fracture rates are unknown but do not appear to be closely related to latitude or fall-related deaths. (*Am J Public Health* 1989; 79:1556–1558.)

Introduction

Fractures of the hip result in over 250,000 hospital admissions each year and, in the elderly, represent 25 percent of all injury admissions.[1] A number of risk factors have been identified for osteoporotic hip fractures including increasing age, White race, and female sex.[2–5] Incidence rates for hip fracture also vary considerably from one country to another and a high rate has been reported for the United States in comparison with other countries.[2,3,6] This study explores geographic variations in hip fractures within the US using data from the National Hospital Discharge Survey (NHDS).

Methods

The NHDS collects data annually on a representative sample of discharges from nonfederal short-stay hospitals in the US.[1] The data set for this study consisted of all sample discharges from 1979 to 1985 among Whites, age 65 years or older in whom hip fracture (ICD-9-CM codes 820.0 to 820.9) was one of the discharge diagnoses. Blacks and other races were excluded from the analysis because of the small number of sample cases. Cases for which race was not stated were considered White based on previous demographic analysis.[4] All discharges whose hospitalization involved late effects of hip fracture (ICD-9-CM code 905.3) or complications of a previous fracture (ICD-9-CM code 733.8) or who were transferred to another short-stay hospital were excluded to minimize multiple counting of the same fracture. The final sample contained 6,701 discharges.

Geographic comparisons were limited to the nine US Census Divisions[7] because of the design characteristics of the

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NHDS.[8] Based on the sample, the projected total numbers of hip fracture patients discharged each year were computed by hospital Census Division using the NHDS weighting factors.[8] Age/sex-specific rates were derived from the mean annual number of discharges and Census estimates of the White civilian population as of July 1, 1982 for male and female age groups 65–74 years, 75–84 years, and 85 years and older. Age/sex-adjusted rates were based on the above age/sex-specific rates and the 1982 US White population as the standard. Standard errors were based on the 1982 NHDS,[9] were adjusted to account for aggregating multiple years of data,[8] and assumed the standard population was not subject to sampling error.

Results

Age/sex-adjusted hip fracture rates per 1,000 population and 95% confidence intervals are shown for the nine US Census Divisions and for the US in Figure 1. Comparisons of all pairs of rates among the nine Census Divisions using the Bonferroni Inequality[10] (comparison wise p = .0014) indicated the rate in the West North Central Division was significantly higher than the rates in all other Divisions except the Mountain and Pacific Divisions.

Age/sex-specific rates (Table 1) were generally higher in the West North Central Division for each age and for both sexes compared to other Divisions. For females, the West North Central rates were at least 45 percent higher than the corresponding US rates in each of the three age groups. Only for males age 75–84 were discharge rates higher in other Divisions (Mountain and Pacific). The highest rates also occurred in the West North Central Division in each of the seven years from 1979 to 1985, the time period over which the data were aggregated.

Discussion

Although hip fractures typically result in hospitalization, hospital discharges cannot be assumed to equal hip fracture incidence—particularly because of multiple counting of cases due to transfers from one hospital to another, and readmissions for additional treatment of old fractures.[11–13] To minimize this potential bias, all transfers to another short-stay hospital and all hip fracture cases with a secondary diagnosis of malunion or late effects were excluded. The data base does contain refracture cases which may represent as many as 15 percent of the discharges.[5,14–16] However, there is no reason to believe that refracture rates would vary greatly by geographic area and, as

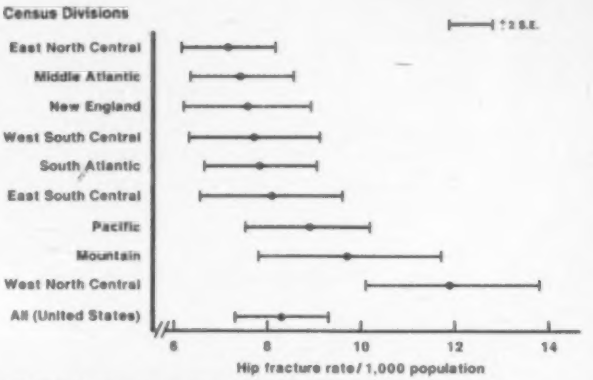

FIGURE 1—Mean Annual Hospital Discharge Rates for Hip Fractures per 1,000 White, Elderly, Civilian Population by Census Division
New England: ME, NH, VT, MA, RI, CT
Middle Atlantic: NY, NJ, PA
South Atlantic: DE, MD, DC, WV, VA, NC, SC, GA, FL
East North Central: MI, WI, IL, IN, OH
East South Central: KY, TN, MS, AL
West North Central: MN, ND, SD, NE, IA, MO, KS
West South Central: AR, OK, LA, TX
Mountain: MY, WY, ID, CO, UT, NV, NM, AZ
Pacific: WA, OR, CA, AK, HI

new events, should be included in estimates of fracture incidence.

Factors such as listing old hip fractures as secondary diagnoses, admitting patients with old hip fractures for arthroplasty, and admitting patients residing outside the Census Division of the hospitals could vary by Census Division and thus are potential sources of bias. Analyses of these variables showed little geographic variation, however.

To corroborate the findings using a different data source, discharge data from a 20 percent sample of all inpatient bills were obtained from the 1985 Health Care Financing Administration (HCFA) MEDPAR file. Hip fracture rates for aged Medicare beneficiaries were also highest in the West North Central Division compared to other Divisions.

This study indicates that discharge rates for hip fracture in elderly Whites is higher in the West North Central region of the US than in other Census Divisions for reasons which are not clear. Several factors have been associated with an increased risk of osteoporotic fractures[2,3,5,16–19] but many, such as diet and exercise, would seem less prevalent in the farm states of this

TABLE 1—Hip Fracture Rates per 1,000 Elderly, White Persons and Standard Errors ( ) by Age, Sex and Census Division

	Age (years)							
	Males				Females			
Census Division	65 & older	65–74	75–84	85 & older	65 & older	65–74	75–84	85 & older
New England	4.1(0.7)*	1.9(0.5)	5.6(1.4)	15.8(4.9)	9.7(1.0)*	3.6(0.8)	12.1(2.1)	36.0(6.2)
Middle Atlantic	4.4(0.5)*	1.7(0.4)	6.3(1.2)	18.3(4.0)	9.3(0.8)*	4.0(0.6)	12.4(1.7)	29.5(4.4)
East North Central	4.2(0.5)*	1.6(0.4)	5.6(1.1)	19.9(4.0)	8.9(0.8)*	3.5(0.6)	11.4(1.6)	30.1(4.4)
West North Central	6.0(0.8)*	2.9(0.7)	6.8(1.6)	27.0(5.7)	15.5(1.4)*	6.2(1.1)	20.7(3.0)	51.0(7.8)
South Atlantic	4.2(0.5)*	1.3(0.3)	5.4(1.1)	22.6(4.8)	10.0(0.9)*	3.5(0.6)	13.7(1.9)	34.5(5.3)
East South Central	4.7(0.8)*	2.0(0.6)	5.8(1.6)	22.2(6.4)	10.1(1.2)*	4.2(0.9)	14.7(2.6)	27.9(5.8)
West South Central	4.8(0.7)*	1.6(0.4)	7.1(1.7)	20.8(5.3)	9.5(1.0)*	2.5(0.6)	12.8(2.1)	38.3(6.5)
Mountain	5.6(0.9)*	2.4(0.7)	8.2(2.1)	21.1(6.5)	12.2(1.4)*	4.7(1.0)	15.9(3.0)	42.6(8.4)
Pacific	4.5(0.6)*	1.9(0.4)	8.2(1.6)	10.6(2.9)	11.5(1.0)*	4.3(0.7)	16.1(2.3)	36.6(5.5)
All (US)	4.8(0.5)	2.2(0.2)	6.4(0.5)	19.6(1.7)	10.4(0.9)	3.9(0.3)	14.0(0.9)	35.2(2.3)

*Age-adjusted.

region. Sunlight exposure is unlikely to be a major factor because rates are dramatically different among regions at similar latitudes (e.g., West North Central and East North Central Divisions). Further, a relationship of geographic variation in hip fracture rates to reported mortality from falls is not evident despite the fact that falls account for most hip fractures in the elderly.[20,21] All seven States in the West North Central Division have above-average death rates from falls in elderly Whites, and Iowa and Minnesota are among the 10 States with the highest rates.[22] However, New England has a high death rate for falls and a relatively low discharge rate for hip fractures. (If case-fatality rates for hip fracture are relatively constant, differences between discharge and mortality rates may reflect geographic variations in the reporting of falls as a cause of death on the death certificate.[23,24]) It is interesting that a relatively high proportion of residents in the West North Central region originate from Scandinavian countries[25] where the incidence of hip fractures is also high.[2,3,6] Further investigation of the factors responsible for the geographic variations observed may lead to new insights into the causes of hip fractures.

ACKNOWLEDGMENTS

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Lead Exposure and Radiator Repair Work

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Abstract: In 1986, the ambient air for lead in radiator repair shops in the Minneapolis-St. Paul metropolitan area exceeded the Occupational Safety and Health Administration (OSHA) action level in nine of 12 shops sampled by Minnesota OSHA. We therefore sought to determine the prevalence of lead exposure/toxicity in this industry. Thirty-five radiator shops were identified, 30 were visited, and 53 workers were studied. The mean blood lead level was 1.53 (range 0.24–2.80). Seventeen individuals had blood lead levels ≥1.93 μmol/L (40 μg/dl). The mean zinc protoporphyrin level (ZPP) was 0.55 μmol/L (range 0.16–1.43). No single worksite or personal characteristic was a strong determinant of either blood lead or ZPP level. (*Am J Public Health* 1989; 79:1558–1560.)

Introduction

Workers in the automotive radiator repair industry are known to be at risk for exposure to inorganic lead from lead-based solder used in the repair of radiators.[1–8] Lead fumes arise from molten lead and lead oxide dusts result from grinding and wire brushing radiator joints. These operations are done less frequently now than in the past, largely due to the recognition of the health hazards they cause.[9] It is claimed that more efficient ventilation, and self-contained breathing apparatus for radiator cleaning have reduced much of the lead exposure.

Results of recent studies in the upper Midwest area of the United States revealed that radiator repair shops may have elevated air lead levels, and that repair workers may have

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elevated blood lead levels, occasionally higher than 2.90 μmol/L (60 μg/dl).[2–5] An auto radiator trade journal performed a questionnaire survey in 1977,[6] in which 100 radiator workers voluntarily reported information including recent blood lead levels, work history and habits, shop characteristics, and personal health history. Blood lead levels ranged from 0.63 to 5.45 μmol/L. Departments of health in some states have attempted to study auto radiator repair shops in a systematic way, with varying degrees of success. Although no publications in the scientific literature resulted, blood lead levels in excess of 4.34 μmol/L were found in some workers (personal communication, Michigan and Wisconsin Departments of Health).

In the summer of 1986, Minnesota OSHA (Occupational Safety and Health Administration) sampled the ambient air for lead in nine radiator repair shops in the Minneapolis-St. Paul area. Air in two of these shops exceeded three times the OSHA standard for ambient lead, and three others exceeded the OSHA action level. The standard Permissible Exposure Limit for lead for a 40-hour work week is 0.05 mg/m^3. This information, together with the results of previous studies, led us to question the degree of lead exposure and toxicity in auto radiator repair workers in the Minneapolis-St. Paul area. We sought to determine the prevalence of lead exposure and toxicity in these workers and characteristics associated with elevated lead levels.

Methods

All automobile radiator repair shops within Minneapolis-St. Paul and their closest suburbs were identified using the Minneapolis-St. Paul yellow page directories. The owners were asked to permit a visit by a study investigator to administer a questionnaire to shop employees and obtain blood samples for lead and zinc protoprophyrin levels (ZPP).

During November 1986, a visit was made to each shop whose owner consented to participate in the study. The shop owner or manager was interviewed to obtain information on shop size, number of cars serviced, and workplace conditions. Permission was then obtained to interview all radiator repair workers present at that time. Each radiator repair worker was then asked demographic questions, recent work history, workplace and general health practices, directed health history, and whether the worker had children under the age of six living in the home. If such children were present, permission was requested to screen them for lead.

Blood lead determinations were done by the anodic stripping voltametry method.[7] ZPP determinations were done at the University of Minnesota using a hematofluorometer.[8] Individual workers were each informed of their blood lead levels by letter, with an accompanying explanation of the results. Shop owners were also furnished with this information if the employee had given written consent to do so.

Later, the shops were visited by an individual hygienist who took area samples for three to six hours using high flow air sampling pumps.[10] Personal breathing zone air samples were also done by attaching the filter cassette of the sampler to the shirt lapel of the participating worker. Analyses of samples were done by atomic absorption.[11] Each shop owner was informed of air measurements with an explanation of the level of lead in the air.

Statistical analyses were done using the Statistical Package for the Social Sciences (SPSS). Shop data for individuals from the same shops were identical, but were treated independently for purposes of analysis because the shops were small, with most having one or two workers, and analysis of covariance showed neither shop size nor number of radiators repaired per year in a specific shop to be significant covariates.

Regression analyses were done with blood lead and ZPP as dependent variables, and several of the items from the shop and individual questionnaires, and area and personal air lead samples, as independent variables. All specified variables were included in the regression model.[12,13] A chi square statistic was computed for categorical variables. In the latter analyses, blood lead (1.93 and ≥1.93 μmol/L) and ZPP (<0.89 and ≥0.89 μmol/L) were treated as categorical variables with two levels each.

Results

Shop Characteristics

Thirty-five radiator shops were identified in the metropolitan area. Of these, 30 were visited, making the shop participation rate 85.7 percent. One shop was not visited because, although the shop owner was cooperative, the radiator repair workers themselves refused to participate. The owners of the other four shops refused to participate.

Shops were small, usually 300 to 600 square feet, and there was generally no interior finish (Table 1). The amount of business varied greatly between shops. While use of specific work site ventilation was often reported by shop owners and the individual workers, only a few had local ventilation and most ventilation systems were not in operation. All but one shop supplied tools for their employees, all had salaried employees, and none had working showers. All shops used solder comprised of 60 percent lead and 40 percent tin.

Area and personal air sampling for lead levels were done in 24 of the 30 shops. Six shops refused to have air monitoring. Personal air lead samples were obtained from individuals in 16 of the 24 shops. Workers refused to wear a pump in eight shops. The mean area lead value was 0.040 mg/m^3 with a range of 0 to 0.281 mg/m^3 (SD = .060). The mean personal air lead value was 0.113 mg/m^3 with a range of 0 to 0.590 mg/m^3 (SD = .020).

Worker Characteristics

A total of 57 radiator repair workers were identified. Of these, four refused to participate in the study, but at least one radiator repair worker was evaluated in each participating shop. Of these workers, the mean age was 37.1 years (SD = 2.8 years), mean education was 11.9 years (SD = 2.8 years), and mean work experience was 14.3 years (SD = 11.2 years). Table 2 details other worker characteristics. Most of the workers identified job duties other than radiator repair that they normally performed, but none involved significant lead exposure. No worker reported ever having been treated for lead toxicity, although five out of 53 reported having had elevated blood lead levels in the past.

TABLE 1—Characteristics of Thirty Radiator Repair Shops

Variables	Mean	Standard Deviation	Range
Age of Shop (years)	17.0	15.5	<1–50
Number of Employees	3.6	2.7	1–10
Number of Radiator Repair Workers	1.9	1.5	1–8
Number of Cars/Trucks Repaired Per Week	43.2	40.8	0–200
Number of Radiators Repaired Per Week	35.5	38.2	1–200
Number of Radiators Repaired Per Year	1281.7	932.2	50–3500

TABLE 2—Self-reported Characteristics of Radiator Repair Workers

Characteristics	Category	Number (%)
Respiratory Protection	Never	41 (77.4)
	≤25% of time	8 (15.1)
	>25% of time	4 (7.5)
Gloves Used	Never	7 (13.2)
	≤50% of time	8 (15.1)
	>50% of time	38 (71.7)
Eat Lunch at Work Stations	Never	48 (90.6)
	≤25% of time	1 (1.9)
	>25% of time	4 (7.5)
Smoke Cigarettes	Yes	32 (60.4)
	No	21 (39.6)

Twelve individuals reported having children under the age of six living at home. Sixteen children were later screened for blood lead and, with one exception, levels were below 0.724 μmol/L, and a mean of 0.45 μmol/L (range = 0.24 to 1.06 μmol/L). This did not correlate well with workers' blood leads, and no further analyses were done.

As seen in Tables 3 and 4, there was a wide range of blood lead levels among the workers. Symptoms associated with lead toxicity included frequent headaches, abdominal cramps, constipation, and muscle aches. None of these symptoms was common in the workers but none was significantly associated with either blood lead or ZPP level.

In bivariate analyses, two variables were associated with increased blood lead levels: number of radiators repaired in the past month (0/8 vs 17/44; 95% CI = 0.09, 0.69)*; and shop age (4/24 vs 13/29; 95% CI = 0.21, 0.35). Years of work experience was the only variable found to be related to ZPP level (3/20 vs 18/33; 95% CI = 0.12, 0.67). Confidence intervals are for the difference in the proportion of workers with elevated blood lead in the high-risk (exposure) group compared to the proportion of workers with increased blood lead in the low-risk (exposure) group.

Multiple regression was used to examine the simultaneous effects of worksite and personal factors. Four variables were entered into the regression for blood lead level; years of work experience ($\hat{B}$ = 0.25, SE 0.15), worker time spent in radiator repair during the last month (2.00, SE 1.43) and last year (0.03, SE 0.18), and shop age (5.97, SE 744), the total r^2 was 0.25. Work experience was the only variable that contributed to the ZPP level (0.89, SE 0.18, r^2 = 0.34).

Discussion

About one-third of the workers had lead levels that were elevated by OSHA standards.[14] Minnesota radiator repair workers had lead levels that were similar to those found in recent studies in this country.[1–5,15] These levels are considerably less than those routinely seen in the high-risk occupations such as lead smelting, storage battery manufacturing, or metal foundries were levels occasionally exceed 4.83 μmol/L.[16] However, the blood lead level[3] found in radiator workers exceeds those in the general population, where mean lead levels in adult males is 0.63–0.72 μmol/L.[16] Auto radiator repair workers appear to be at a risk of mild to moderate elevations in blood lead levels.

No single worker characteristic or practice was a good predictor of elevated blood lead. Only two shop characteristics—volume of radiator repair done and shop age—were associated with elevated blood lead levels. Environmental data did not correlate well with blood lead levels. This may have occurred because of the variability of the volume of work in each shop from day to day as well as our having been invited into shops on "less busy days." The data presented here confirm earlier studies of this industry. There is need for worker education, safer worker practices, and better shop design and ventilation in the radiator repair industry.

*One worker had not done radiator repair during most of the month prior to the study.

TABLE 3—Number of Individuals with Blood Lead Levels by Approximate Quartiles*

Blood Lead Level (μmol/L)	Number of Workers
>1.93	15
>1.50–1.93	11**
>1.06–1.50	13
0.24–1.06	13

Mean = 1.53, Standard Deviation = .61
**Two workers in this group had a blood level of 1.93 μmol/L

TABLE 4—Number of Individuals with Free Erythrocyte Protoporphyrin Levels by Approximate Quartile*

FEP Level (μmol/L)	Number of Workers
>.067	15
>0.41–0.67	12
>0.30–0.41	12
0.16–0.30	13

Mean = 0.53, Standard Deviation = 0.31

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Acute Illnesses among Los Angeles County Lifeguards According to Worksite Exposures

CONSTANCE S. B. SULLIVAN, DRPH, AND MARY E. BARRON, MPH

Abstract: Due to public concern regarding the potential adverse health effects of exposure to Santa Monica Bay waters, a case-control study of acute illnesses among Los Angeles County lifeguards was conducted. Workers' compensation claimants (N = 112) were matched to healthy lifeguards working in the same year and having the same job classification. Conditional logistic regression showed that the southernmost worksites of the bay were associated with all acute illnesses (OR = 14.4, 95% CI = 4.7, 44.8) and with ear infections (OR = 12.5, 95% CI = 2.9, 53.4). (*Am J Public Health* 1989; 79:1561–1563.)

Introduction

The quality of water in Santa Monica Bay of Los Angeles County, California has been the subject of debate because of the dumping of industrial waste and sewage into the bay and untreated discharges into the surf zone from numerous storm drains along the shore.

Most studies of water quality and adverse health outcomes have established a direct correlation between pathogen levels in the water and disease incidence in swimmers,[1–5] while two found no relationship.[6,7] Cabelli and coworkers found a dose-response relation between swimming-associated gastrointestinal symptoms and water quality (as measured by enterococcus concentrations) at a group of New York City beaches.[1] In a study of five Ontario lakes conducted by Seyfried and Cook,[4] otitis externa was related to water levels of *Pseudomonas aeruginosa* and to the frequency of swimming.

To assess the potential adverse health effects of exposure to the waters of Santa Monica Bay, we studied work-related acute illnesses in an occupationally exposed population—Los Angeles County lifeguards. This is the first study of a defined population exposed to these waters.

Methods

We used a case-control design, with cases defined as all episodes in which lifeguards made a compensable claim for a work-related, acute illness in the years 1980 through 1986. Lifeguards with repeated acute illness claims in a single year were included only once. A healthy lifeguard was chosen as a control for each case. The control lifeguard worked the same year that the case was ill, had no acute illness claim in that year, and worked in the same job class (i.e., temporary assignment, permanent assignment, lieutenant, etc.).

For each case and control, the daily work assignment logs for the month including the case's date of illness were searched. Details of the work assignments on the case's date of illness and seven days prior to that were recorded, including section of beach, tower, and hours at each tower.

Descriptive analyses used SAS computer programs,[8] and conditional logistic regression estimates[9] used BMDPLR.[10]

Results

During the seven-year study period, 112 acute compensable illnesses occurred among the Los Angeles County lifeguards. The peak year was 1980 with 28 claims, while 1985 and 1986 had only eight claims each. Most claims were due to otitis externa and unspecified ear infections (N = 69). Other diagnostic categories were otitis media (N = 15), dermatitis (N = 6), rash (N = 2), skin infection (N = 6), and conjunctivitis (N = 5).

The Department of Beaches and Harbors of Los Angeles County has divided the county beaches into three sections for administrative purposes (Figure 1): Southern, Central, and Northern. The age-adjusted odds ratios (ORs) for working at each section of beach compared with not working at all (Table 1) showed a very strong association between working Southern section beaches and acute illness. Similar associations were observed with illness restricted to otitis externa or unspecified ear infections (OR for Southern section = 12.5, 95% CI = 2.9, 53.4). Ten cases and 41 controls did not work at all during the reference week. The odds ratio for working as a Los Angeles County lifeguard at any location was 8.3.

Since ear infections are related to the amount of time spent in the water,[7,11] we also analyzed the effect of beach location using only those pairs where both subjects worked at all during the reference week (N = 64) with working as a proxy for swimming. The southernmost beaches within the bay proper were most strongly associated with acute illness. To remove the effect of 13 repeated ear infections occurring in different years to some "disease-prone" individuals, we

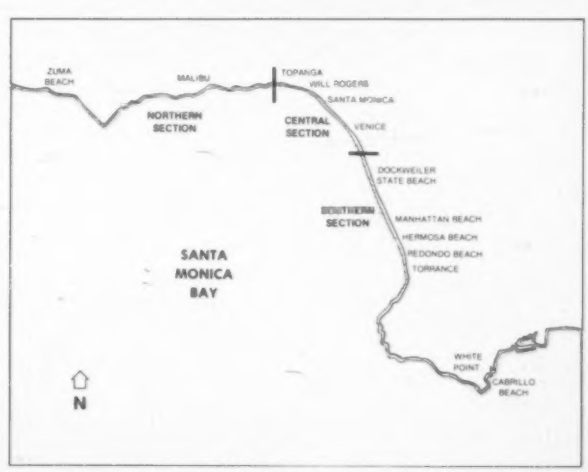

FIGURE 1—Los Angeles County Beaches

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TABLE 1—Age-Adjusted Conditional Disease Odds Ratios for Working at Various Beaches

Beach	Odds Ratio (95% CI)
All beaches combined	8.3 (2.9, 23.5)
Southern section beaches combined	14.4 (4.7, 44.8)
Cabrillo Beach-White Point	5.4 (0.8, 36.2)
Torrance-Redondo Beach	17.0 (2.9, 100.2)
Hermosa Beach	36.4 (6.7, 198.2)
Manhattan Beach	15.7 (2.8, 89.7)
Dockweiler State Beach	15.6 (2.2, 111.0)
Central section beaches combined	5.3 (1.8, 15.5)
Venice	10.7 (2.0, 57.2)
Santa Monica	7.0 (1.9, 24.6)
Will Rogers	6.4 (0.4, 103.8)
Northern section beaches combined	4.4 (1.4, 14.1)

repeated the analysis excluding these episodes. The odds ratios were diminished slightly but again the southernmost beaches within the bay were most strongly and significantly associated with illness.

Discussion

All illnesses studied for this report were compensated through Los Angeles County's workers' compensation system, and were therefore physician-diagnosed. Almost certainly less misclassification occurs with such data than with self-reports of symptoms.[2,6,12,13] On the other hand, workers' compensation reports probably yielded an underestimate of the total number of work-related illness events. It is possible that lifeguards who experienced minor symptoms did not seek medical attention or might not have associated symptoms with their jobs. This may explain the absence of gastrointestinal illnesses.

Although it is possible that lifeguards working at certain beaches were more likely to report acute illness, we know of no specific work pattern that would create such a bias. Also we noted a high odds ratio in Venice Beach in the Central section, which is administratively distinct from the Southern section.

We lacked data to control for other water exposures, e.g., swim workouts at the worksite or recreational exposure to the bay at the worksite or other locations. We had no data on swimming habits, such as head submersion, associated with ear problems.[11,13] Matching on job class partly controlled for differences in job tasks which could affect the subjects' amount of exposure to water while at work. For example, temporary lifeguards cover most of the tower assignments and therefore may have been required to enter the water more often, on average.

The reason for the decline in work-related acute illnesses over the study period is not known. Lifeguards may have begun mitigating their risk of illnesses by reducing their water exposures or by changing swimming habits.

A recent report on Santa Monica Bay indicated that storm drains and creeks are responsible for the levels of the indicator bacteria (enterococci and coliforms) measured at shoreline. Whether these bacteria are indicative of human pathogenic agents is unclear.[14] Using maps provided by the Los Angeles County Department of Public Works, we calculated the number of storm drains (500 feet or longer) or creeks per 10,000 linear feet of coastline (Table 2). These ratios were generally higher in Southern beaches, except for Dockweiler State Beach; but that beach includes the outlet for the largest drain into the bay which may contaminate a wider area than most drains. A recent raw sewage spill into this drain resulted in beach closures which included Venice and Dockweiler beaches.[15] We present this ecologic association as one possible explanation for our study results, recognizing that microbial contamination of Santa Monica Bay is a complex, multifactorial, and only partially understood process.

The findings of this study, the first of its kind, suggest that acute illnesses among lifeguards were associated with Southern Santa Monica Bay work locations.

ACKNOWLEDGMENTS

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TABLE 2—Storm Drain and Creek Outlets into Santa Monica Bay and other Los Angeles County Beaches

Beach Areas	Number of Storm Drains and Creeks per 10,000 Feet of Coastline	Characteristics of Catchment Area	Comments
Southern Section			
Cabrillo Beach-White Point	7.2	Residential and commercial	Los Angeles Harbor ship channel empties east of Cabrillo Beach
Torrance-Redondo Beach	7.9	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Hermosa Beach	9.0	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Manhattan Beach	5.5	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Dockweiler State Beach	2.2	Residential, commercial, and heavy industrial	Ballona Creek at Northern edge of beach drains extremely large area and occasionally drains raw sewage spills
Central Section			
Venice	3.1	Residential and commercial	Moderate catchment area
Santa Monica	4.4	Residential, commercial, and industrial	Moderate to large catchment area
Will Rogers	4.4	Residential and unimproved parkland	Moderate to large catchment area
Northern Section	2.9	Sparse residential and parkland	Large catchment area; some raw sewage contamination from septic tanks

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The Reliability of Health Risk Appraisals: A Field Trial of Four Instruments

KEVIN W. SMITH, MA, SONJA M. MCKINLAY, PHD, AND JOHN B. MCKINLAY, PHD

Abstract: A field trial was conducted to evaluate the reliability of four widely used health risk appraisals (HRAs). A total of 338 randomly selected adults aged 25 to 65 years residing in the Boston metropolitan area completed an HRA on two occasions seven to 12 weeks apart. Test-retest reliability was assessed by comparing self-reported baseline risk scores to follow-up scores for heart attack risk and selected coronary heart disease risk factors. Respondents generally gave consistent reports for family history, cigarette smoking, and relative weight (test-retest $r > .75$), but self-reported scores for physiologic status (blood pressure and cholesterol) and lifestyle (diet, physical activity, and stress) were much less stable. Coefficients for heart attack risk and appraised age ranged from .43 to .87 for the four HRAs. The reliabilities of two self-scored instruments were greatly reduced by mathematical errors made by respondents when computing their heart attack risk scores. These results were not affected by the length of the follow-up period (seven to 12 weeks). (*Am J Public Health* 1989; 79:1603–1607.)

Introduction

Reliability refers to the degree to which measurements are affected by random error. Random error effects may be assessed by the stability of the measures or scores produced by an instrument from one time period to another. The higher the correlation between scores measured at two points in time under identical conditions, the more reliable the instrument.[1]

For health risk appraisal (HRA) instruments designed to provide estimates of mortality or morbidity risk for various diseases, reliability is an important issue for several reasons. First, reliability is related to the validity of an HRA. Random errors in self-reported health status and physiologic measures will decrease the accuracy of an HRA's estimate of disease risk. Second, HRAs are frequently employed to evaluate the effectiveness of health promotion programs. If HRA scores are unstable during the pre- to post-program periods, changes attributable to the intervention may be indistinguishable from unreliability in the instrument itself. Moreover, respondents may be inclined to dismiss HRA estimates if they find that their risk scores fluctuate dramatically from one time period to another.

In recent years, several studies have raised questions about the reliability of HRA data. Best and Milsum found that participants in a smoking cessation program reported many improbable changes in their body frame size, medical history, and family history after six months.[2] Sacks and his associates found that only 15 percent of the subjects in a clinical trial gave consistent responses to all HRA items at both baseline and follow-up interviews.[3] On the other hand, some studies have reported test-retest reliability coefficients exceeding .7 for selected risk factors and overall risk estimates, especially for follow-up periods of short duration (three days to one month).[4–7]

Several methodological limitations associated with these studies make it difficult to generalize about HRA reliability.

- First, some investigators have reported only the percentage of respondents choosing the same response category for a particular item at both points in time. This is inadequate for assessing the degree of reliability because it does not take into account the differences between the risk scores assigned to the categories. In many instruments, selecting an adjacent category for a particular item produces little or no change in the risk score for that item; in other cases, the difference may be substantial.
- Second, much of the previous work has been based on limited samples of self-selected volunteers. In some instances, fewer than 30 subjects were involved in the analyses.[5] It is not clear whether the results from these studies are representative of the general population.
- A third problem common to all test-retest analyses is that reliability may be confounded with actual change. Scores for the same individual may differ from one administration to another because other behavioral changes have occurred in the intervening period.
- Finally, the effect of the length of the follow-up period on reliability coefficients for HRAs has yet to be established.

Recognizing these shortcomings, several recent reviews of the status of risk appraisal methods have called for more comprehensive investigations of HRA reliability.[8–10]

In response to these issues, a field trial was designed to assess the reliability and validity of four HRAs. For each HRA, this paper examines:

- the proportion of respondents choosing the same risk factor categories at both administrations,
- test-retest reliability coefficients for individual risk factors and total risk scores,
- the effect of behavioral changes occurring during the follow-up interval on reliability coefficients, and
- the influence of the length of the follow-up period on changes in risk scores.

Methods

The field trial was conducted from January to November 1987. Subjects were identified through random digit dialing of 15 telephone exchanges in the Boston, Massachusetts metropolitan area. Five exchanges were drawn from each of three strata comprising calling areas with below average, average, and higher than average household incomes. One adult was selected at random from each household and screened for eligibility. The study was restricted to persons between the ages of 25 and 65 at last birthday who did not

From the American Institutes for Research, Watertown, MA. Address reprint requests to Kevin W. Smith, MA, Research Scientist, American Institutes for Research, 9 Galen Street, Watertown, MA 02172. Dr. S. McKinlay is Principal Research Scientist, and Dr. J. McKinlay is Senior Research Fellow at AIR. This paper, submitted to the Journal January 23, 1989, was revised and accepted for publication June 23, 1989.

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have a history of coronary heart disease, diabetes, or hypertension, so that the sample would reflect the typical target population for HRAs that contain coronary heart disease components. An introductory letter explaining the purpose of the study was sent to each respondent who provided an address at the time of the screening.

Eligible cases were then contacted by a trained field technician to arrange a home visit. During the visit, the technician administered a standardized personal interview and one of the four HRAs evaluated by the project. Field technicians were not permitted to help respondents complete HRA items or compute risk scores. Subjects were reinterviewed seven to 12 weeks after the initial visit. Fifty-five percent of the eligible cases (the reliability group) were asked to complete the same HRA at follow-up. The follow-up interval was designed to be long enough to minimize memory effects, but short enough so that major changes in lifestyle and behavior would be unlikely to occur. The analyses presented in this paper are restricted to field trial participants completing the same HRA at both baseline and follow-up.

Four widely-used HRAs were chosen to represent the major types of instruments that are currently available:[11]

1) Health Risk Appraisal (Centers for Disease Control)
2) The Heart Test (Arizona Heart Institute)
3) RISKO (American Heart Association)
4) Determine Your Medical Age (Blue Cross/Blue Shield, NY)

Each instrument requires a respondent to select categories that best describe personal habits, physiologic status, and medical history. Point values or weights associated with these categories are then combined to produce an overall estimate of risk. In these four HRAs, overall risk is summarized by either a heart attack risk scale, where higher values represent greater risk, or by appraised age. Appraised age is the age of adults with the same all-causes mortality risk as a particular individual. The items and range of risk scores for each HRA are shown in the Appendix.

Reliability analyses were conducted by comparing the baseline scores for individual risk items, heart attack risk, and appraised age with the values chosen by the same respondent at follow-up. Consistent with psychometric practice, Pearson correlations between the baseline and follow-up risk scores served as reliability coefficients.[12] These correlations are approximately the same as weighted Kappas with weights equal to squared deviations between baseline and follow-up risk scores.[13,14] For four items, the influence of other changes occurring during the follow-up period was assessed by computing test-retest correlations only for those who said, in another part of the interview, that their behavior had not changed since baseline. For relative weight, diet, and physical activity, these correlations were restricted to those reporting that their behavior at follow-up was "about the same" as it had been at baseline. Analyses for smoking were based only on those who reported smoking the same number of cigarettes at both interviews. Finally, follow-up risk scores were regressed on baseline scores and the number of days that had elapsed between the dates of the baseline and follow-up were computed to evaluate the impact of the length of the follow-up interval on reliability.

In the absence of established minimum standards for test-retest coefficients, correlations of .8 or greater were adopted a priori as a criterion level in this study. This level is equivalent to the informal standard for internal consistency used in many social science applications.[12] Sample sizes for each of the four HRA groups in the field trial were designed to have sufficient power to detect a difference between the standard of .8 and a correlation of .65 or less, which was regarded as unacceptably small for reliable items.[15]

Results

Baseline interviews were completed by 732 of 1,276 eligible adults (57.4 percent) contacted for the field trial. Table 1 compares the demographic characteristics reported during the telephone screening by reliability group participants, other participants in the field trial, and eligible subjects who declined to participate. All three groups were similar with respect to these measures; a logistic regression analysis of response rates indicated that the probability of obtaining a completed baseline interview was not affected by these characteristics (likelihood ratio statistic = 5.70, df = 6). Of the 403 reliability group subjects interviewed at baseline, 338 (84 percent) consented to a follow-up interview and completed HRAs at both administrations (n = 83 to 87 persons for each instrument).

Response Category Comparisons for Risk Items

Table 2 compares the percentage of respondents who chose the same category for eight major risk items at both baseline and follow-up. The marginal distributions of these items were similar at each administration. Participants were generally very consistent in their reports of family history and smoking. Choices for relative weight were consistent at least 70 percent of the time. The lowest agreement rates occurred for lifestyle factors such as diet, stress, and physical activity, which may change even over short periods of time.

The consistency of blood pressure and cholesterol reports are more difficult to evaluate. These reports were the same at both administrations at least 87 percent of the time for two of the three self-scored instruments. Determine Your Medical Age, however, merely specifies a cut-off level so that only two choices were available to respondents. Similarly, the Arizona Heart Test specifies only three categories for blood pressure: normal, borderline, or high. The agreement rates for RISKO were below 70 percent, but this instrument offered more category options than the others. Like most of the Geller/Gesner mortality risk HRAs, the Centers for Disease Control (CDC) instrument requests actual blood pressure readings and cholesterol levels. The majority of participants left these items blank because they did not know

TABLE 1—Demographic Characteristics of Field Trial Participants and Nonparticipants[a]

Characteristics	Reliability Group Participants (N = 403)	Other Field Trial Participants (N = 329)	Non-Participants (n = 544)
Age (years)	38.8 (10.6)	40.0 (10.8)	40.2 (11.1)
Percent female	55.8 (49.7)	59.9 (49.1)	60.5 (48.9)
Percent Black/Hispanic	6.7 (25.1)	10.7 (30.9)	8.2 (27.4)
Income (thousands)[a]	41.1 (10.4)	41.2 (11.1)	40.5 (10.1)
Self-reported health status[b]	2.06 (.94)	1.92 (.86)	2.06 (.91)
Worry about health[c]	2.54 (.83)	2.54 (.85)	2.52 (.84)

[a]Mean and (standard deviation)
[a]Mean household income in subject's telephone exchange
[b]5-point scale; 1 = excellent; 5 = poor
[c]4-point scale; 1 = not at all, 4 = most of the time

TABLE 2—Percent of Respondents Choosing Same Risk Item Category at Baseline and Follow-up by HRA

Risk Items	Arizona Heart Institute	CDC	Determine Your Medical Age	RISKO
Family History	91.9	96.4	86.2	—
Smoking	82.6	91.6	83.9	85.7
Relative Weight	79.8	78.0	71.3	80.8
Physical Activity	64.3	73.5	65.5	—
Blood Pressure	94.6	NR	87.4	58.4
Cholesterol	—	NR	94.3	64.8
Stress	65.6	—	—	—
Diet	72.3	—	55.2	—

NR = value not reported by majority of respondents

the appropriate values. For this reason, no results for these two risk factors are presented for CDC in Table 2.

Test-Retest Correlations for Risk Items

The proportion of respondents selecting the same response categories at two points in time is, at best, a crude indicator of risk factor reliability because it fails to account for the degree of risk involved.[16] For the self-scored instruments, different response categories sometimes have the same risk values, so that selecting different categories may not result in any change in the risk score for that item. To provide a more sensitive measure of reliability, test-retest correlations were computed for each HRA on the basis of the scores associated with each risk factor category. The CDC instrument differs from the others in that risk weights rather than point values are assigned to item categories.

Table 3 shows the test-retest correlations for the risk scores associated with the eight HRA items. Correlations between baseline and follow-up risk scores for family history, smoking, and relative weight exceeded .75 for all applicable instruments. The remaining five items all had estimated reliability coefficients that were significantly lower than the criterion level of .8 for at least one HRA. In spite of differences in question wording and risk scores, the reliability estimates for a specific item were usually quite similar among the four HRAs.

Effects of Behavioral Change on Reliability

Discrepancies between pre- and posttest reports do not always mean that information has been reported unreliably because behavior may have changed since the baseline interview. Test-retest correlations were therefore computed for selected items only for those respondents who claimed their behavior had not changed during the follow-up interval. These items were weight (relative weight), exercise level (physical activity), sodium, fat, and caloric intake (diet), and smoking risk (only those who gave the same number of cigarettes per day at both interviews).

Results for these respondents are summarized in Table 4. Compared to the coefficients for all cases (Table 3), behavioral changes had the greatest impact on reports of dietary habits. The estimated reliabilities increased slightly (.04 on average) for smoking and relative weight, but were virtually identical for physical activity risk scores. Therefore, with the possible exception of dietary practices, it would appear that behavioral changes did not greatly influence the reliabilities of these risk items.

Test-Retest Correlations for Heart Attack Risk and Appraised Age

In the process of collecting data during the field trial, it quickly became apparent that many respondents who received self-scored HRAs had difficulty computing these risk scores. Heart attack risk scores and appraised age values were therefore checked for accuracy by project staff. Eighty-seven percent of those completing the Arizona Heart Institute HRA were able to calculate the correct score compared to 75 percent of the RISKO respondents and only 42 percent of those completing Determine Your Medical Age. These rates reflect the mathematical skills required by each instrument—the Arizona Heart Institute HRA involves adding point values, RISKO requires both addition and subtraction, while respondents must add, subtract and divide scores to determine their "medical" age. Error rates were also influenced by the educational attainment of respondents. Those without four-year college degrees were twice as likely as college graduates to make computational mistakes on both the RISKO (22.5 percent compared to 9.0 percent) and Determine Your Medical Age (61.4 percent compared to 30.0 percent) HRAs.

Test-retest reliabilities for heart attack risk scores and appraised age were calculated separately for the risk scores actually reported by respondents as well as score totals corrected for mathematical mistakes. The coefficients for reported risk scores were .87 for the Arizona Test HRA, .84 for the CDC HRA, and .74 for Determine Your Medical Age. The correlation between baseline and follow-up scores reported by RISKO respondents (r = .43), on the other hand, was significantly lower than the criterion level of .8. For this instrument, even minor addition or subtraction errors can produce misleading estimates of whether a person is above or below the average risk for heart disease. When these errors were corrected, RISKO's estimated reliability increased to .76. Correcting computational errors also produced a marked improvement in the reliability of Determine Your Medical

TABLE 3—Risk Item Test-retest Correlations and 95 Percent Confidence Intervals by HRA

	Arizona Heart Institute		CDC		Determine Your Medical Age		RISKO	
Risk Items	r	95% CI	r	95% CI	r	95% CI	r	95% CI
Family History	.90	(.84–.93)	.75	(.64–.83)	.89	(.83–.93)	—	—
Smoking	.89	(.84–.93)	.95	(.93–.97)	.82	(.73–.88)	.89	(.83–.93)
Relative Weight	.84	(.76–.89)	.96	(.94–.97)	.78	(.68–.85)	.78	(.67–.85)
Physical Activity	.60	(.45–.73)	.65	(.50–.76)	.70	(.58–.80)	—	—
Blood Pressure	.87	(.78–.92)	NR	—	.45	(.26–.60)	.55	(.36–.69)
Cholesterol	—	—	NR	—	.65	(.51–.76)	.58	(.40–.72)
Stress	.62	(.47–.74)	—	—	—	—	—	—
Diet	.52	(.34–.66)	—	—	.57	(.41–.70)	—	—

TABLE 4—Risk Item Test-retest Correlations and 95 Percent Confidence Intervals for Cases Reporting No Change in Behavior during Follow-up Interval

Risk Items	Arizona Heart Institute r	Arizona Heart Institute 95% CI	CDC r	CDC 95% CI	Determine Your Medical Age r	Determine Your Medical Age 95% CI	RISKO r	RISKO 95% CI	Mean Change in r*
Smoking	.95	(.92–.97)	.97	(.95–.98)	.86	(.78–.91)	.95	(.93–.97)	+.04
Relative Weight	.88	(.80–.93)	.96	(.94–.98)	.76	(.61–.85)	.91	(.84–.95)	+.04
Physical Activity	.67	(.49–.79)	.62	(.41–.77)	.66	(.46–.80)	—	—	.00
Diet	.78	(.63–.87)	—	—	.63	(.43–.77)	—	—	+.16

* = mean difference from correlation in total HRA sample

Age (from r = .74 to r = .99) but not the Arizona Heart Institute HRA, for which mathematical mistakes were much less common. While computational mistakes are not an issue with computerized Geller/Gesner method HRAs like the CDC's, these instruments typically insert risk weights of 1.0 for any unknown risk factors. This practice artificially inflates reliability coefficients.

Length of Follow-up Period

The reliability of most measurements decreases over time. In this study, the interval between baseline and follow-up interviews ranged from seven to 12 weeks (49 to 84 days). The final set of analyses was designed to determine whether the reliability of respondents' self-reports declined as the length of the follow-up period increased. To address this issue, follow-up risk scores were regressed on baseline scores and the number of days that had elapsed between the baseline and follow-up interviews. The regression results (Table 5) indicate that the length of the follow-up interval had little impact on the follow-up scores for any of the risk measures. However, this interval was comparatively short and had limited variability among respondents.

TABLE 5—Multiple Regression of Follow-up Risk Score on Length of Follow-up Interval* Adjusted for Baseline Risk Score: Unstandardized regression coefficient and (standard error)

Risk Score	Arizona Heart Institute	CDC	Determine Your Medical Age	RISKO
Heart Attack Risk	.044	28.2	—	.046
	(.047)	(21.7)	—	(.093)
Appraised Age	—	.040	−.192	—
	—	(.027)	(.166)	—
Family History	−.001	.001	−.014	—
	(.012)	(.001)	(.023)	—
Smoking	.028	.002	.045	−.009
	(.019)	(.002)	(.070)	(.007)
Relative Weight	.000	.001	.058	−.013
	(.010)	(.001)	(.054)	(.011)
Physical Activity	.027	.004	.149	—
	(.016)	(.007)	(.087)	—
Blood Pressure	−.021	NR	−.015	−.011
	(.012)	—	(.045)	(.012)
Cholesterol	—	NR	.005	−.016
	—	—	(.019)	(.010)
Stress	.019	—	—	—
	(.023)	—	—	—
Diet	.034	—	−.022	—
	(.020)	—	(.056)	—

*Length of follow-up interval = days elapsed between baseline and follow-up interviews
NR = value not reported by majority of respondents

Discussion

In this field trial, respondents generally gave consistent reports for three risk items: family history, cigarette smoking, and relative weight. Self-reports of physiological status (blood pressure and cholesterol) and lifestyle (diet, physical activity, and stress), on the other hand, were much less consistent between the baseline and follow-up interviews. The consistency of physical activity reports may have been diminished by seasonal variations in exercise patterns.

There were some important differences among the instruments with respect to the reliabilities of heart attack risk scores and appraised age. The test-retest coefficients for the Arizona Heart Institute and Centers for Disease Control HRAs both exceeded .84, but were only .74 and .43 for Determine Your Medical Age and RISKO, respectively. The correlations for the latter two instruments were reduced by mathematical errors made by respondents when computing their risk scores. Reliability coefficients for the CDC HRA, on the other hand, may be overstated due to the practice of substituting risk weights of 1.0 for blood pressure and cholesterol whenever the actual values are unknown.

Inconsistencies between baseline and follow-up values were more likely to be attributable to unreliable reporting on the part of respondents rather than to actual changes in risk factor status. Changes in risk scores were not affected by variations in the length of the follow-up period. However, testing reactivity could have lowered reliability levels. Completing an HRA for the first time can motivate respondents to obtain more accurate information about family history, blood pressure, or cholesterol, so that follow-up responses will then appear to be inconsistent.

The results of the field trial indicate that the reliability of HRA risk scores can vary greatly from one instrument to another. These findings have several implications for health educators using HRAs. First, computation errors may severely reduce the reliability of self-scored instruments. Computerized HRAs may therefore be the most desirable type unless manual calculations can be routinely checked for errors. Second, additional work needs to be done to improve the measurement of specific risk factors, particularly physical activity levels, dietary practices, and physiologic status. The reliability of blood pressure and cholesterol levels can be increased by measuring these values during physical examinations rather than relying on self-reports. Finally, as the reliability of an HRA declines, it becomes increasingly difficult to distinguish changes in risk status from random reporting errors. This is particularly important for investigators interested in assessing the effects of health promotion interventions because unreliability makes intervention effects more difficult to detect. Therefore, only HRAs for

which reliability can be demonstrated should be considered for evaluations of the effectiveness of these interventions.

APPENDIX

Risk Scores Ranges by HRA

	Arizona Heart Institute (self-scored)	Centers for Disease Control (computer-scored)	Determine Your Medical Age (self-scored)	RISKO (self-scored)
Heart Attack Risk	0–50	2–21,969	—	0–27
Appraised Age	—	18.4–65.1	12.0–70.6	—
Family History	0–5	.9–1.2	−30–15	—
Smoking	0–10	.8–2.1	−6–24	−2–4
Relative Weight	0–3	.8–1.6	−6–24	−2–3
Physical Activity	0–3	.6–2.5	−12–12	—
Blood Pressure	0–6	.4–1.1	0–12	−5–6
Cholesterol	—	.5–1.6	0–6	−3–3
Stress	0–6	—	—	—
Diet	0–6	—	−6–18	—

The Centers for Disease Control (CDC) HRA, which requires computer processing, provides probability estimates for the 12 leading causes of death as well as appraised age based on the Geller/Gesner method.[17]

The Arizona Heart Institute Test and RISKO are self-scored instruments focusing on heart attack risk.

In the Determine Your Medical Age HRA, appraised age is calculated by adjusting an individual's chronological age for medical history and lifestyle factors.

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Call for Papers:
Risk Management in Rural Health Care

The American Society for Healthcare Risk Management has issued a call for papers to be considered for its Fall 1990 issue of *Perspectives in Healthcare Risk Management*. Emphasis should be on the issues and problems inherent in rural health care risk management, as well as on suggestions for how to deal with them effectively. Topics and approaches include but are not limited to: credentialing issues, peer review, conflict of interest, patient transfer issues, swing-beds, risk pooling, and quality assurance/risk management integration.

Authors should submit a one-page abstract and detailed outline of an original article on any area of risk management in rural health care delivery settings. *Abstracts and outlines must be received no later than March 5, 1990.* Authors of those abstracts selected for further consideration will be asked to submit their completed papers (not to exceed 20 typewritten, double-spaced pages) by June 4, 1990. Manuscripts will be accepted with the understanding that their content is unpublished and not being submitted for publication elsewhere. Send abstracts to:

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Accuracy of Perceptions of Heart Attack Risk: What Influences Perceptions and Can They Be Changed?

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Abstract: Using perceived risk of a heart attack, we examined the relative importance of perceived risk factors and sociodemographic variables on subjects' perception of heart attack risk, the relationship between perceived and objectively measured heart attack risk, and the effect of health risk appraisal (HRA) feedback on risk perceptions. Data derive from a random sample of 732 Greater Boston, Massachusetts area men and women ages 25–65 years, who participated in a field trial of health risk appraisal instruments. At baseline and approximately two months later, all respondents completed a questionnaire assessing their own health-related behavior, risk factors, and perception of heart attack risk. At baseline, respondents also completed one of four HRA instruments. Physiologic measures of cholesterol, blood pressure, and weight were taken at either baseline or follow-up visit. Results showed that respondents used established risk factors in estimating overall risk; that compared to objective measures of risk, a high percentage of respondents displayed an optimistic bias; and that HRA feedback had some effect on perception of heart attack risk for those at high risk. Implications of these results for health promotion are discussed. (*Am J Public Health* 1989; 17:1608–1612.)

Introduction

A person's beliefs about his or her perceived susceptibility to a condition or disease figure prominently in models of health behavior such as the Health Belief Model,[1,2] the Theory of Reasoned Action,[3] Subjective Expected Utility Theory,[4,5] and Protection Motivation Theory.[6,7] These models assume that perceived susceptibility (also referred to as vulnerability or risk) is essential in motivating behavior.

Previous research, however, has shown that people tend to underestimate their own risk of developing certain conditions or diseases[8–17]—or they have what Weinstein[13] refers to as an "optimistic bias." This underestimation has important implications for health promotion and behavior change programs. If people do not perceive themselves as vulnerable to a disease or condition, they are less likely to adopt recommended behaviors.[1,18] It is therefore important to understand what determines a person's perception of risk and how these perceptions can be made more realistic.

Most of the research on perceived susceptibility uses comparative risk judgments to examine how people compare their risk for a particular condition or disease to that of their peers.[8–10,13–16] This method, however, does not address the accuracy of an individual's risk perception. In an attempt to examine this issue, Weinstein conducted two studies[15,16] in which an individual's comparative risk judgments were correlated with his or her relevant risk factors. Weinstein found that these associations were often weak or nonexistent. However, the measures of risk factors lacked precision and were based only on self-report. In addition, each risk factor was separately correlated with perceived risk, rather than combined to form an overall estimate of risk. One purpose of this study is to use epidemiologically based models to examine the relation between perceived risk and objectively measured risk for heart attacks.

Optimistic biases may have several sources. They may be due to cognitive errors (e.g., lack of information or experience with a condition) or motivational needs (e.g., self-esteem enhancement or defensive denial).[16] To the extent that they result from cognitive errors, individualized feedback about risk may overcome these biases. Health risk appraisal instruments (HRAs) are designed to estimate a person's mortality or morbidity risk for various diseases based on characteristics such as medical history, blood pressure, and smoking habits.[19] One of the goals of HRAs is to provide risk-related feedback that will enable a person to reduce risk in areas amenable to modification.[20] Another purpose of this study is to examine the effect of individualized risk feedback on risk perception.

Methods

Procedure and Sample

The data used in this study were collected during a field trial of health risk appraisal instruments conducted by the Cambridge Research Center of the American Institutes for Research in 1987. Detailed descriptions of the procedure and sample characteristics are reported in the article in this issue by Smith, McKinlay, and McKinlay.[21] Briefly, respondents were identified through random digit dialing of residential households in 15 neighborhoods and towns in the Boston metropolitan area. Eligibility was restricted to adults between the ages of 25 and 65 who did not have a history of coronary heart disease, diabetes, or hypertension. Following a telephone screening, baseline interviews were conducted by trained field technicians in respondents' homes. After completion of the baseline interview, respondents were randomly assigned to complete one of four risk appraisal (HRA) instruments (CDC, Arizona Heart Test, RISKO, and Determine Your Medical Age), or were assigned to a "no HRA" condition. (See Smith, *et al*,[21] for details of the HRAs). Field technicians were not permitted to help respondents complete HRA items or compute risk scores. Follow-up home visits were conducted seven to 12 weeks after the baseline to monitor changes in health status, health practices, and medical care utilization. During one of the two home visits (randomly determined), technicians measured blood pressure, height and weight, and drew a venous blood sample for cholesterol analysis.

Measures

Perceived risk of heart attack or stroke was assessed by the question: "Compared with persons of your own age and

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sex, how would you rate your risk of having a heart attack or stroke within the next 10 years?" Respondents answered on a 5-point scale ranging from "much lower than average" to "much higher than average."

Respondent characteristics that might be related to perceived risk were divided into sociodemographic characteristics, self-perceptions of health, and cardiovascular risk factors. Sociodemographic factors included education, race, gender, and age (although gender and age can also be considered risk factors). Self-perception of health status was measured on a 5-point scale ranging from excellent to poor.

Cardiovascular risk factors were measured as follows. Family history of heart disease was based on having a biological parent die of heart disease or stroke before age 65. Smoking was based on self-report of how many cigarettes per day the person currently smoked. Self-report of smoking is generally thought to be reliable, especially for respondents who are not in a smoking reduction program.[22] All respondents who smoked at least one cigarette per day were classified as smokers. The Harvard Alumni Activity Scale,[23] which measures recreational physical activity in terms of kilocalories (kcal) expenditure per week, provided the measure of exercise. Stress was determined by the number of stressful events (e.g., death of partner or spouse, divorce, loss of job, major change in health of family member), as derived from the Holmes and Rahe Scale[24] that were reported by the respondent as occurring within the past two months. Self-reports of weight, cholesterol, and blood pressure as compared to average were also obtained.

Results

Perceived Heart Attack Risk

Almost 56 percent of the respondents rated their risk as lower than average, 29 percent rated it about average, and only 13 percent rated their risk as higher than average. With over half the respondents rating their risk as lower than average, these results support previous findings that people tend to rate their own risk as lower than that of their peers—that people have an optimistic bias. Although respondents were healthier than their peers because those with hypertension, diabetes, and other chronic diseases were excluded from the study, this optimistic bias is confirmed when compared to objective risk (see below).

To examine the relative importance of perceived risk factor status, sociodemographic factors, and perceived health in people's perception of heart attack risk, a multiple regression analysis was performed in which perceived risk of a heart attack reported at baseline was the dependent variable. These results show associations between perceived heart attack risk and the following risk indicators: number of cigarettes smoked, death of a parent due to heart disease; and self-assessments of weight, blood pressure and cholesterol compared to average levels (see Table 1). Neither amount of exercise nor number of recent stressful events was related to perceptions. These results suggest that people do make use of cardiovascular risk indicators in estimating overall risk. These risk factors accounted for 24 percent of the variance in perceived risk. Self-reported health and education were also positively related to perceived risk and, combined with the risk factors already included, accounted for 30 percent of the variance. The correlation between self-reported health and risk is partly a function of the way in which self-reported health was scored (i.e., a 5-point scale). The Pearson correlations between these variables and perceived risk show a pattern similar to the multiple regression, suggesting that multicollinearity is not a problem in this analysis.

TABLE 1—Results of Multiple Regression on Variables Related to Perceived Heart Attack Risk at Baseline

Variable	Unstandardized Regression Coefficient	S.E.
Self-reported risk factors		
Cigarettes smoked per day	.02	.00
Cholesterol relative to average	.24	.05
Weight relative to average	.23	.05
Death of parent due to CHD	.38	.10
Blood pressure relative to average	.19	.06
Exercise	−.03	.03
Number of recent stressful events	−.02	.06
Perceived Health	.34	.05
Sociodemographic Factors		
Education	.08	.03
Age	−.01	.00
Minority	−.22	.13
Female	.12	.08

Education was positively related to perceived risk in the regression model, yet had a negative Pearson correlation with perceived risk. Because Pearson correlations revealed that education was related to self-perceived health, exercise, smoking, and blood pressure, and because we thought education might modify these effects, we also performed a multiple regression that included the interactions between education and these variables. The only significant result was an interaction between education and smoking, such that those with more education who smoked perceived their risk to be greater.

Perceived and Objective Heart Attack Risk

The risk equation from the new Carter Center Healthier People HRA was used to obtain an objective measure of coronary heart disease risk.[25] This logistic risk model builds on the results of the Risk Factor Update Project but takes into account more recent epidemiological data from the Framingham Heart Study. The variables included in the logistic equation to predict mortality from myocardial infarction are: sex, total cholesterol, age, number of cigarettes smoked per day, systolic blood pressure, relative weight, HDL cholesterol, and presence of diabetes. Family history and exercise are not included in the model because it is thought that their effects operate through other risk indicators in the model (cholesterol, blood pressure, and weight).[25]

Because respondents were instructed to answer the risk perception question in comparison to other people of the same age and sex, we adjusted the objective measure of risk for age and sex. Each respondent received a risk ratio which was based on the logistic estimate from the model divided by the average risk for that person's age, sex, and race. A ratio of 1.0 meant a person was at average risk for his or her age, sex, and race.

Respondents were then classified as to whether they were at above average, average, or below average risk. A ratio below .75 was considered below average risk, .75 to 1.25 average risk, and over 1.25 above average. In order to create a symmetric distribution, these ratios were transformed onto a log scale, in which the cut-offs became ln(.75) and ln(1.33). These ratios represent equal "risk distances" from the null value of 0.0 on the log scale [ln(1.33) = −ln(.75) = .29]. Using this distribution, 39 percent of the respondents were classi-

fied as below average risk, 24 percent as having average risk, and 36 percent as above average risk.

Perceived risk was then compared with the objectively measured risk categories. In Table 2, the rows indicate the percentage of respondents in each risk perception category, while the columns indicate the percentage of respondents in each objectively measured category. Summing over cells, a total of 42 percent of the respondents underestimated their risk (15% + 17% + 10%), while only 18 percent (11% + 3% + 4%) overestimated risk, and 40 percent (25% + 9% + 6%) were accurate. Based on these results, when using an objective measure of risk, this analysis also found evidence of an optimistic bias—a high percentage of respondents underestimated their risk.

We next performed two logistic regressions: one to predict the probability that risk was overestimated (pessimists), and the other to predict underestimation of risk (optimists). Variables included as predictors were the same variables listed in Table 1 that were used to predict perceived heart attack risk. The variables associated with pessimism were death of parent due to heart disease (O.R. = 2.72, 95% CI = 1.52, 4.87), self-assessed health status (O.R. = 1.80, 95% CI = 1.25, 2.58), and age in years (O.R. = 1.06, 95% CI = 1.02, 1.08). Thus, pessimists were more likely to be in worse health, have had a parent die of heart disease, and be older. The result for death of a parent may reflect the fact that people give weight to this risk factor while the Carter Center model does not include it. The variables that predicted optimism were age (O.R. = .90, 95% CI = .87, .92) and education (O.R. = .80, 95% CI = .66, .98). Those who were younger and had less education were more likely to be optimists.

Effect of Health Risk Feedback

Table 3 shows the effect of feedback on *change* in perceived risk. In this table, the columns indicate the type of feedback received, while the rows indicate whether perceived risk increased, did not change, or decreased from baseline to follow-up. The numbers in parentheses are the percentage of respondents who did not change their perceptions and whose perceptions accurately reflected their HRA feedback. In reading this table, it is important to keep in mind that those who received feedback that they were below average risk should have either been accurate or decreased their perceived risk, while those who received feedback that they were above average risk should have either been accurate or increased their perceived risk.

As seen in Table 3, the majority of respondents did not change their risk perceptions. Those who changed the most received feedback that they were above average risk. They were more likely to increase their perceived risk (21.8 percent of those receiving above average feedback). This group, however, was also the least accurate in their risk perceptions. Those who received feedback that they were at below average or average risk, or did not receive any HRA feedback were as likely to increase their perceived risk as to decrease it. It should be pointed out that because respondents tended to underestimate their risk at baseline, there were more respondents in the "above average" feedback category with the potential to change. While HRA feedback had its greatest effect on those at above average risk, 12 percent of those with above average risk changed their perceptions in the opposite direction of the feedback.

TABLE 2—Relationship between Perceived and Estimated Risk as Measured by the Carter Center Model

	Risk Ratio			
Perceived Risk[a]	Below %	Average %	Above %	Total
Lower than Average	25	15	17	57
Average	11	9	10	30
Higher than Average	3	4	6	13
Total	39	28	33	100
n	(226)	(138)	(205)	(569)

[a]As compared to persons of same age and sex.

TABLE 3—Relationship between HRA Feedback and Change in Perceived Risk

	HRA Feedback[a]			
Change in Perceived Risk	No Feedback	Below Average Risk	Average Risk	Above Average Risk
Increase	16.9	10.4	14.9	21.8
No Change	69.7	78.7	74.5	66.3
(accurate)		(60.8)	(26.6)	(16.8)
Decrease	13.5	11.0	10.6	11.9
	100%	100%	100%	100%
n	89	347	94	101

[a]Scored 1 = below average, 2 = average, 3 = above average.

To further explore whether receipt of HRA feedback altered risk perception, we looked at follow-up risk perception as a function of baseline risk perception, the same variables examined in the baseline risk perception analysis (this time using follow-up responses) plus HRA feedback. A multiple regression was performed in which follow-up risk perception was regressed on baseline risk, follow-up perceptions of risk factors, sociodemographic factors, perceived health, and HRA feedback. Feedback was measured by a scale relating HRA risk category to the respondent's initial perception. The respondent's initial perception was taken into account because feedback should only have an effect when it differs from the person's original perception. The feedback scale ranged from 1 to 5, where 1 meant the HRA risk category was much lower than the individual's perception, and 5 meant the HRA risk category was much higher than the respondent's perception.

These results, shown in Table 4, reveal only feedback from the Arizona Heart Institute (AHT) HRA had a significant impact on heart attack risk perception. Compared to the other three HRAs, the feedback from this HRA was presented in a concrete fashion directly related to heart disease risk. Respondents were clearly told that they were above average, average, or below average risk for developing heart disease. Thus, respondents may be more likely to understand and use the feedback from the AHT.

Risk Perception and Behavior

Although it was not a primary purpose of the study, because data on respondents' risk-related behavior were available, it was possible to examine the relationship between change in perceived risk from baseline to follow-up and corresponding behavior change. As previously mentioned, models of health behavior often assume a relationship between perceived susceptibility and behavior.[1–6] It follows from this assumption that increases in perceived risk should be related to risk-reducing behaviors. To address this issue,

TABLE 4—Results of Multiple Regressions on Variables Related to Perceived Heart Attack Risk at Follow-Up

Variables	Unstandardized Regression Coefficient	S.E.
Baseline Risk Perception	.56	.04
Risk Factors		
Cholesterol relative to average	−.12	.04
Cigarettes smoked per day	.01	.002
Weight relative to average	.11	.04
Death of parent due to CHD	.11	.08
Blood pressure relative to average	.09	.05
Exercise	−.02	.03
Number of recent stressful events	−.05	.04
Self-reported health	.12	.04
Sociodemographic Factors		
Education	.03	.03
Minority	−.04	.10
Female	−.03	.06
Age	.003	.003
Health Risk Appraisal Feedback[a]		
CDC	.01	.07
RISKO	.07	.07
Arizona Heart Institute	.26	.10
Medical Age	.09	.07

[a]Scored relative to baseline perception of risk.

we looked at behavior change from baseline to follow-up as a function of change in risk perception. The behaviors we analyzed were: smoking, exercise, weight; self-reports of reduction in salt, fat, and calories; and recent blood pressure or cholesterol screening. Results showed that those who increased their perceived risk of a heart attack were no more likely to change their behavior than those whose risk perception either decreased or remained the same.*

Discussion

Our results showed that people do use established epidemiologic risk factors in estimating their overall risk of heart attack, but that additional factors also influence a person's risk perception. One such variable is self-perceived health status: those people who perceive their overall health as worse are more likely to rate their risk as higher. This finding is consistent with other research in which people who were sick estimated their risk for other illnesses as higher.**

Using an objective measure of individual risk, a high percentage of respondents exhibited an optimistic bias, providing additional support for previous studies.[9,10,13–16] Those who were younger and less educated tended to be more optimistic, consistent with suggestions that younger people feel less vulnerable to conditions or diseases.[16] The finding that family history of heart disease is associated with pessimism suggests that people weigh this factor heavily and is consistent with Becker and Levine's[8] study of high risk siblings of patients with coronary heart disease that found less optimistic bias than seen in other studies.

The effect of health risk feedback on risk perceptions was greatest for those who were above average in risk: twice the percentage of respondents who were above average risk *increased* their perceived risk. These findings suggest that HRA feedback can effectively change the perceptions of those at high risk. It is important, however, to add a cautionary note: 12 percent of those who received high risk feedback actually *decreased* their perceived risk and this above average group was the *least* accurate to begin with. Furthermore, the results of the multiple regression showed that only feedback from the AHT was significantly associated with change in risk perception. Clearly, feedback did not always have consistent effects on risk perception and these effects differed by HRA.

With respect to various explanations of the optimistic bias, to the extent that feedback had the desired effect on accuracy, the cognitive explanation of this bias is supported. However, it is also clear that information did not have the desired effect for the majority of respondents.

In the present study we did not find a relation between increases in perceived risk and behavior change. Although some models of health behavior predict a relation between perceived susceptibility and behavior,[1–6] support for this association is strongest for disease prevention behaviors that do not require modification of health habits.[26] Our behavioral measures consisted of changes in personal health habits such as smoking, diet, exercise, and weight. Models of health behavior also suggest that factors other than cognitions (e.g., social support, social norms and self-efficacy) may be important in health habit modification.[3,26,27] It should be pointed out, however, that this analysis was based on limited statistical power as only 87 respondents increased their perceived risk.

Two important aspects of this study need to be pointed out. One is that the logistic model of actual risk is based on mortality from heart attack, while respondents were asked about their chances of *having* a heart attack or stroke. It is possible that they may have responded differently if asked about their chances of dying from a heart attack or stroke, although we feel it is unlikely the respondents made this distinction. The other important point has to do with HRA feedback. Respondents were only given the feedback provided by the HRA and did not receive any additional counseling or education, as is often done with HRAs. In some cases, respondents may not have understood the feedback.*** Thus, HRAs may not be effective for use in mass screenings, but may have a greater effect on risk perception when feedback is easier to understand, or when they are given in conjunction with a health education program, a conclusion also drawn by Schoenbach, *et al*,[28] in a review of the effectiveness of HRAs.

In conclusion, this study suggests that people do make use of established risk factors in estimating cardiovascular risk; that compared to objective measures of risk, a high percentage of people display an optimistic bias; and that risk feedback has some effect on risk perception for those at high risk, but such feedback is not optimally effective.

ACKNOWLEDGMENTS

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*Data available on request to author.

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NCHSR Grants $4 Million for Patient Outcome Research

The National Center for Health Services Research and Health Care Technology Assessment (NCHSR) has awarded new grants totaling nearly $4 million in first-year costs in a series that will measure the effectiveness of alternative medical procedures in treating specific health conditions. Total spending on the nationwide DHHS program will be $5.9 million this fiscal year, with $52 million proposed for FY 1990.

The new grants will support research for treatment of conditions that affect a large number of Americans every year—prostate gland enlargement, heart attack, cataracts, and lower back pain—for which millions of Medicare beneficiaries are treated every year. The research projects—expected to require about five years to complete—will assess comparative effects of alternative therapies on patient survival, quality of life, costs, and other factors. Research teams will consist of practicing clinicians and other experts.

Grants to support patient outcome research assessment teams were awarded to:

Richard Deyo, MD, University of Washington ($896,049), nonsurgical interventions for low back pain and different types of lumbar surgery;

Barbara J. McNeill, MD, Harvard Medical School ($900,000), for a national data base for outcomes from myocardial infarction treatment;

Earl P. Steinberg, MD, Johns Hopkins University ($899,986), assess cataract procedures;

John E. Wennberg, MD, Dartmouth Medical School ($933,535), transurethral resection, open prostate surgery, and nonsurgical interventions for enlargement of prostate gland.

Planning grants of $50,000 each awarded to seven research teams included:

Fred Featherstone, MD, American Academy of Orthopedic Surgery, hip replacement/disease; *Alvan Feinstein,* MD, Yale University, use of colonoscopy; *Emmett B. Keeler,* PhD, Rand Corporation, treating peripheral vascular diseases; *David B. Matchar,* MD, Duke University, prevention and management of stroke; *Lawrence W. McMahon, Jr.,* MD, University of Michigan, use rates and appropriateness of cholecystectomy, and screening/treatment for colon cancer; *David B. Pryor,* MD, coronary artery disease; and *Sanford Schwartz,* MD, University of Pennsylvania, gallstone disease.

For further information, consult *Research Activities* (special release), from Publications and Information Branch, NCHSR, 18–12 Parklawn Building, Rockville, MD 20857; Tel: 301/443-4100.

The Prevention of Silicosis and Prediction of Its Future Prevalence in China

JIEZHI LOU, MD, AND CHEN ZHOU, MD, MPH

Abstract: We studied the effects of preventive and therapeutic interventions on the 18-year cumulative incidence of silicosis of 26,603 dust-exposed workers in seven Chinese mines and industrial plants. Cumulative silicosis incidence decreased from 36.1 percent in workers employed before 1950 to 1.5 percent in workers employed after 1960. From the 1950s to 1970s, eight-year cumulative incidence of tuberculosis decreased from 54.7 percent to 16.7 percent and case fatality of silicosis patients dropped from 53.9 percent to 18.3 percent. From 1950s to 1980s, the average age at the detection of silicosis increased from 41.3 to 52.7 and the average survival time of silicosis patients prolonged from 2.0 to 12.2 years. Workers over age 40 who began exposure to dust before 1960 will be the main source of new silicosis patients in future. Most expected new cases of silicosis will occur within the next 15 years. The silicosis population will remain unchanged for the next 20 years and will decrease dramatically after 25 years. There will be few silicosis patients in 30 years and new cases of silicosis will be difficult to find in 45 years. These results show that the preventive and therapeutic actions against silicosis in China have been effective. (*Am J Public Health* 1989; 79:1613–1616.)

Introduction

Silicosis is a major occupational lung disease which remains a problem in industrialized and developing countries.[1,2] Since the 1950s, special attention has been paid to the prevention of silicosis in China. A regulation to control the silica dust hazard in industrial establishments and mines was published in 1956,[3] and a regulation concerning the prevention and treatment of silicosis was published in 1957.[4] Experiences with dust control were summarized and applied nationwide in 1958. During the past 30 years, many industrial companies have implemented these regulations which include monitoring dust concentrations in the work environment, applying wet mining and engineering control methods, screening for silicosis in dust-exposed workers, and improving the care of silicosis patients. The evaluation of the effects of these preventive and therapeutic efforts on the change in prevalence of silicosis and predicting its future prevalence are important for future preventive programs.[5–7] We, therefore, conducted an investigation on the prevalence and development of silicosis in seven industrial companies of different types. Based on these data, we predicted the future trend of silicosis in these establishments.

Methods

Subjects and Their Industrial Environments

The subjects of this investigation had been exposed to dust for at least three years and had physical examination, occupational history records, and chest X-rays available for the study. The 26,603 dust-exposed workers came from two metallic mines (one lead mine, one molybdenum mine), two coal mines (coal mine 1, and coal mine 2), a fireproof raw material workshop in a steel plant, a foundry in a large machinery plant, and a raw material workshop in a glass factory. These seven industrial companies were selected as representative of different industrial environments with silica dust hazards and also because their records were complete.

Preventive and Therapeutic Interventions

From 1955 to 1957, the two metallic mines and two raw material workshops initiated comprehensive dust control programs focusing on wet operation. The other three establishments took similar actions in 1962 or 1963. Although there was no systematic dust monitoring data, the dust concentration were very high according to a few random measurements. After applying wet mining and engineering control methods, the average dust concentration decreased sharply (Table 1). Dust was collected on membrane filter and measured by gravimetric methods. Free silica content was measured by chemical-gravimetric method.

Silicosis patients were systematically screened and identified from the raw material workshops in 1950 and 1951 respectively. Similar screening in the two metallic mines—coal mine 2, and the foundry—began in 1955–56 and 1958–59, respectively, while the first screening for silicosis in coal mine 1 began in 1963. As the numbers of silicosis patients increased, the companies set up special physician-directed organizations to examine dust-exposed workers and to treat the silicosis patients. Dust-exposed workers were systematically screened annually and approximately one-third of the dust-exposed workers were examined by chest X-rays each year. More than 90 percent of the silicosis patients were

TABLE 1—Average Dust Concentrations (total dust) in Seven Industrial Companies before and after Dust Control Regulations

Companies	Date (years)	No. of Samples	Mean (mg/M^3)	SiO_2 (%)
Lead	1956	18	340.0	31.5
	1957–81	2038	5.7	
Molybdenum	1956	24	313.1	5.0
	1957–81	43621	4.7	
Refratory	1956	33	287.7	49.2–98.6
	1957–81	4417	3.9	
Cast Steel	1959–62	111	200.5	85.0–97.0
	1963–81	144	29.3	
Glass	1953–56	28	263.5	92.0–98.5
	1957–81	185	4.3	
Coal 1	1960–62	24	211.0	31.0
	1963–81	5646	17.2	
Coal 2	1960–62	25	222.5	23.0
	1963–81	1242	17.8	

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transferred from jobs that exposed them to dust as well as given symptomatic treatment. Some companies also set up a special center to care for simple silicosis patients.

Diagnostic Criteria

The diagnosis of silicosis was performed by special silicosis diagnostic teams in each province or municipality. The criteria of radiographic classification of silicosis was based on the silicosis diagnostic criteria published by the Ministry of Health of People's Republic of China:[4]

> Stage I silicosis: definite evidence of nodular opacities. This demands at least 10 nodular opacities of 1 mm or larger in diameter in two square centimeters in any field of lung.
>
> Stage II silicosis: nodular opacities increase in number and enlarge in size and occupy more than four of the six lung fields.
>
> Stage III silicosis: nodular opacities conglomerate into large opacities of a minimum of 2 cm in diameter.
>
> Silicotuberculosis: tuberculosis developed in any stage of silicosis.

These criteria were set up for silicosis. They are crude and do not consider irregular opacities and profusion in comparison with ILO (International Labour Office) criteria, but they are accurate for silicosis diagnosis and easy to use.

If the subject was dead, the date of death, age at death, and cause of death were recorded. We interviewed the workers or their relatives and co-workers in a few cases when records were incomplete. We also recorded all workers who were no longer available because of non-silicosis related death or relocation.

Analysis

We divided the seven companies into three groups to represent three different industrial environments: metallic mines group (lead mine, molybdenum mine); coal mine group (coal mine 1 and coal mine 2); and plants group (a fireproof raw material workshop, a raw material workshop in a glass manufacturing factory, a foundry). We analyzed the prevalence of silicosis, case fatality rate, and causes of death among these three groups. To obtain the cumulative incidence rate, we divided observed workers into four cohorts according to the time of their first exposure to dust. The number of workers entered in these four cohort were 3,331, 7,108, 7,907 and 8,257, respectively, and the workers were followed up for 18 years, e.g., those with first exposure to dust before 1950 were followed up until 1966. A life table method was used to calculate the 18-year cumulative incidence of silicosis among workers who began exposure to dust before 1950, during 1950–54, 1955–59, and 1960–64.[8] The average annual incidence of silicosis was calculated as follows:

$$\text{average annual incidence (\%)} = \text{cumulative incidence (\%)/observed years.}$$

We also divided silicosis patients into three cohorts according to the time of diagnosis. Rates of silicosis progression, silicosis mortality and complication with tuberculosis in each eight-year diagnosis period.

To predict the future prevalence of silicosis, we used the average annual incidence rate derived from cumulative incidence of silicosis in dust-exposed workers, life expectancy of healthy dust-exposed workers of different age groups to predict the expected new cases of silicosis among healthy workers and the future trend of silicosis. The life expectancy of healthy dust-exposed workers of different age groups was calculated according to the life expectancy of male residents in Shenyang, China in 1976.[9] The details of this method were described in a previous report.[10]

Results

Silicosis

By the end of 1981, 3,803 silicosis patients were diagnosed (14.3 percent of the 26,603 dust-exposed workers). The rate in workers exposed to dust before 1950 was highest (36.1 percent), rates for workers employed later decreased gradually. For those who started employment in 1960 or later, the rates in the metallic mine group and the plant group decreased to 0.30 and 0.07 percent, respectively; the rates of the coal mine group remained somewhat high (3.0 percent) because of delay in dust suppression (Table 2).

The 18-year cumulative incidence of silicosis was highest in workers who started exposure to dust before 1950 and declined in those whose exposure started later (Table 3).

As the incidence of silicosis declined, the average age at disease occurrence and average duration from starting exposure to development of stage I silicosis (latent period) increased. The average age at which silicosis was diagnosed was 41.3 and average latent period 11.3 years in the 1950s, increasing to 45.7 and 14.5 in the 1960s, 48.7 and 17.0 in the 1970s, and 52.7 and 19.7 in the 1980s, respectively.

Disease Progression, Tuberculosis as a Complication

By the end of 1981, of the 3,803 diagnosed silicosis cases, 997 (26.2 percent) had progressed to a subsequent stage and 1,634 (43.0 percent) had been diagnosed as having tuberculosis. The average percent of progression (progressing from stage I to stage II and III or from stage II to stage III) was 26.2 percent and the average incidence of tuberculosis was 43.0 percent. Among the 1,634 cases of silicosis complicated with tuberculosis, 901 cases (55.1 percent) were identified at the time when silicosis was first diagnosed. The life table method was used to calculate the cumulative incidence of progression and of tuberculosis among patients diagnosed before 1955, in 1955–64 and in 1965–74 (Figures 1 and 2).

Death from Silicosis

By the end of 1981, 1,094 of 3,803 silicosis patients had died, a mortality rate of 28.8 percent. Figure 3 shows the case fatality within equal periods of eight years of the patients diagnosed in different time periods. The case fatality declined over time. Table 4 presents the average age at death and mean duration from silicosis diagnosis to death among three groups during different time periods. The average age at death and mean duration from diagnosis to death increased significantly over time.

TABLE 2—Cases of Silicosis among Workers Employed in Different Time Periods

Industry Group	Date (year)	No. of Workers	No. of Silicosis Cases	Percent
Metallic mine	1950	1106	442	39.96
	1950–	3281	397	12.10
	1955–	1980	84	4.24
	1960–78	3021	9	0.03
Coal mine	1950	1886	595	31.55
	1950–	2874	840	29.23
	1955–	5154	767	14.88
	1960–78	3814	115	3.02
Plant	1950	339	166	48.97
	1950–	953	309	32.42
	1955–	773	78	10.09
	1960–78	1422	1	0.07
Total	1950	3331	1203	36.12
	1950–	7108	1546	21.75
	1955–	7907	929	11.75
	1960–78	8257	125	1.51

TABLE 3—Comparison of Accumulated Silicosis Prevalence Rates in Worker Cohorts Beginning Exposure to Dust in Different Time Periods

Observed year	Time Entry into Cohorts			
	Before 1950	1955–54	1955–59	1960–64
0–	0.00	0.00	0.00	0.00
1–	0.03	0.00	0.01	0.00
2–	0.12	0.00	0.01	0.00
3–	0.18	0.04	0.01	0.00
4–	0.63	0.17	0.02	0.03
5–	0.93	0.41	0.13	0.06
6–	1.12	0.74	0.21	0.13
7–	3.23	1.16	0.47	0.33
8–	5.09	1.57	0.66	0.84
9–	6.79	2.19	0.81	1.42
10–	8.43	2.84	1.41	1.70
11–	9.04	3.69	1.92	1.80
12–	9.42	4.80	2.77	1.87
13–	10.29	6.05	3.46	1.97
14–	12.58	7.19	4.06	2.36
15–	17.28	8.58	4.61	2.64
16–	18.47	9.96	5.51	2.98
17–	20.49	10.95	6.38	3.28
18–	—	—	—	—

TABLE 4—Average Age at Death and Mean Duration from Diagnosis to Death (survival time) among Silicosis Patients in Different Time Periods

Industry Group	Time (year)	No. of Deaths	Age at Death (years)	Survival Time (years)
Metallic mine group	1950–	23	42.6	1.9
	1960–	112	47.4	5.8
	1970–	128	56.2	11.0
	1980–	48	61.2	14.3
Coal mine group	1950–	—	—	—
	1960–	74	51.2	3.0
	1970–	358	53.4	6.7
	1980–	101	56.5	10.1
Plant group	1950–	30	39.5	2.0
	1960–	90	44.7	5.0
	1970–	94	54.2	10.1
	1980–	36	59.3	16.8
Total	1950–	53	40.8	2.0
	1960–	276	47.5	4.8
	1970–	580	54.2	8.2
	1980–	185	58.0	12.2

The distribution of specific causes of death of silicosis patients is shown in Table 5. Tuberculosis was a major cause of death (62.1 percent). After improvement in health care, the proportion of death caused by tuberculosis dropped from 98.1 percent in the 1950s to 48.7 percent in the 1980s. With the increase in the mean age at death, the proportion of other pulmonary diseases, cardiovascular diseases, and cancer as causes of death increased from 1.9 percent in 1950s to 44.7 percent in 1980s.

Predicting New Cases of Silicosis

Using annual incidence rates of silicosis in dust-exposed workers, age distribution, and life expectancy of healthy dust-exposed workers, we calculated the expected number of cases of silicosis in the future (Tables 6 and 7). We estimate 464 new cases of silicosis are predicted to develop in the remaining 8,146 healthy workers in the metallic mine group, 1,412 cases are predicted to occur in 10,284 healthy coal miners and 248 cases in 2,520 healthy workers in the plant group. Workers over age 40 who started their exposure to dust before 1960 will be the main source of new cases of silicosis. Most expected cases of silicosis will occur within the next 15 years. The development of new cases of silicosis will be less after 35 years and will not be found in 45 years.

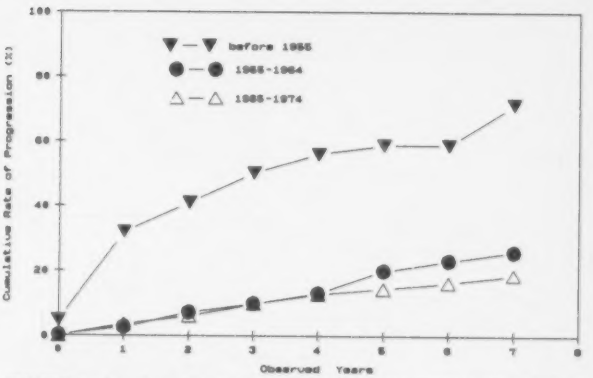

FIGURE 1—Cumulative Rate of Progression of Silicosis among Silicosis Patients Diagnosed at Different Time Periods.

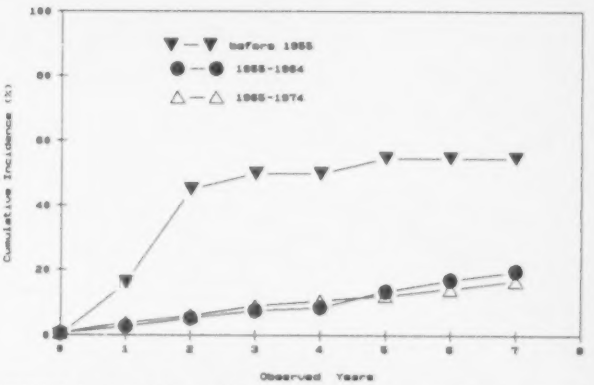

FIGURE 2—Cumulative Incidence of Tuberculosis among Silicosis Patients Diagnosed at Different Time Periods.

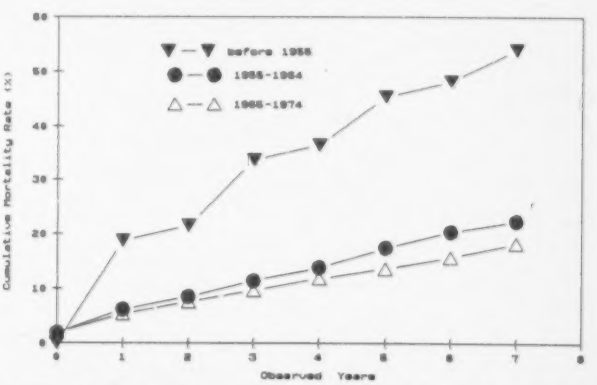

FIGURE 3—Cumulative Mortality Rate among Silicosis Patients Diagnosed at Different Time Periods.

Discussion

During the rapid industrialization of China in the 1950s,

TABLE 5—The Distribution of Specific Causes of Death of Silicosis Patients in Different Time Periods

Time (year)	No. of Deaths	Tuberculosis		Pulmonary Disease		Cancer		Cardiovascular Disease		Other Disease	
		No.	%	No.	%	No.	%	No.	%	No.	%
1950–	53	52	98.1	1	1.9	0	0.0	0	0.0	0	0.0
1960–	276	202	73.2	30	10.9	12	4.3	2	0.7	30	10.9
1970–	580	335	57.8	93	16.0	60	10.3	38	6.6	54	9.3
1980–	185	90	48.7	38	20.5	27	14.6	17	9.2	13	7.0
Total	1094	679	62.1	162	14.9	99	9.0	57	5.2	97	8.8

TABLE 6—Expected Numbers of Silicosis among Healthy Dust-exposed Workers at Different Age Groups

Industry Group	No. of Workers	Expected Numbers of Silicosis at Different Ages						
		25–	30–	40–	50–	60–	70–	Total
Metallic mine	8146	0.7	22.8	63.5	283.7	80.3	13.1	464.1
Coal mine	10284	1.8	208.4	267.8	719.4	214.8	11.8	1424.0
Plant	2520	0.4	25.8	13.3	129.8	37.9	3.9	211.1
Total	20950	2.9	257.0	344.6	1132.9	333.0	28.8	2099.2

TABLE 7—Expected New Cases of Silicosis among Healthy Dust-exposed Workers with First Exposure to Dust at Different Time Periods

Entry Time of Cohort	No. of Workers	Projected New Cases of Silicosis in Future Years						
		<5	5–	15–	25–	35–	45–	Total
<1950	1889	108.8	203.2	75.2	0.7	0.0	0.0	387.9
1950–	5087	177.4	337.1	167.0	2.9	0.0	0.0	684.4
1955–	6376	139.7	276.6	187.9	24.2	0.0	0.0	628.4
1960–	2688	24.1	48.2	43.5	30.9	3.4	0.0	150.1
1965–	4910	32.9	65.9	64.2	57.9	27.4	0.1	248.4
Total	10950	482.9	930.0	538.8	116.6	30.8	0.1	2099.2

dust concentrations in the industrial environment were very high. In the seven establishments we studied, total dust concentrations were between 200 and 300 mg/M^3. The prevalence of silicosis under these high concentrations was about 36 percent, the latent period was 11.3 years, and patients died within two years after diagnosis. But since the Chinese government set up the regulations concerning the control of silica dust and prevention and treatment of silicosis in 1956 and 1957, all of these companies have made special efforts to prevent silicosis. Compared with the 1950s, the cumulative incidence of silicosis, incidence of tuberculosis in silicosis patients, and case fatality rate all declined dramatically. The average age at silicosis occurrence and survival time of silicosis patients increased significantly. These changes strongly demonstrated the effectiveness of preventive and therapeutic efforts in these companies. These silicosis prevention experiences could provide a valuable reference for rapidly industrializing countries.

Our results demonstrated that the prevalence of silicosis in dust-exposed workers decreased with improved working conditions and abatement of the dust concentration. In the coal mines, where dust level had not been completely controlled under the maximum allowable concentration, the prevalence of silicosis in workers employed after 1960 was still relatively higher than in other companies which had lower dust concentrations. Further action to suppress dust in these mines is necessary.

Recently, many researchers paid much attention to predicting epidemiological trends of silicosis incidence.[6,7,10] Since many factors could affect silicosis incidence, it is difficult to estimate the trends accurately. The life table method we used here is a simple but effective approach. With the average annual incidence rate derived from the cumulative incidence of silicosis, the age distribution and life expectancy data of healthy dust-exposed workers, new cases of silicosis could be estimated accurately.

In this study, we found that the workers age 40 and older who were exposed to dust before 1960 will be the major source of new silicosis patients. This result is consistent with those of two investigations reported by Lou, *et al.*[10,11] The future preventive work in these companies should concentrate on early detection of silicosis among these older workers and delay the development of the disease.

Since this investigation was done in 1985 and most new cases of silicosis will develop in the next 15 years, the silicosis population will not decrease within this century. Also, because the survival time of silicosis is between 10 to 15 years, the silicosis patient population will decline sharply after 25 to 30 years. In the years 2020 or 2030 (35 or 45 years later), new cases of silicosis will be very difficult to find. These predictions show that silicosis will remain a problem for the next 40 years, therefore, it is also necessary to improve health care delivery for silicosis patients and develop new anti-silicosis drugs or therapies to prolong their life expectancy.

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Risk of Carotid Endarterectomy in the Elderly

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Abstract: We used the Medicare claims files to describe operative mortality for 2,089 New England residents over the age of 65 who underwent carotid endarterectomy in 1984 and 1985. For patients ages 65 to 69, the risk of death within 30 days of surgery was 1.1 percent, (95% confidence interval = 0.5, 2.1), for those ages 70 to 74, 2.8 percent (1.7, 4.4), for those ages 75 to 79, 3.2 percent (1.8, 5.2), and for those over age 80, 4.7 percent (2.3, 8.5). Nearly 80 percent of patients underwent surgery at hospitals performing 40 or fewer carotid endarterectomies per year on the Medicare population. The adjusted odds ratio for 30 day mortality for patients undergoing surgery in these low-volume hospitals was 2.8 (95% CI = 1.1, 7.2) compared to higher volume hospitals. Although the Medicare claims data provided only limited data about post-operative strokes, analysis of post-operative stroke risk supported these findings. (*Am J Public Health* 1989; 79:1617–1620.)

Introduction

Carotid endarterectomy is commonly performed, costly and controversial. In 1985, more than 100,000 operations were performed in the United States at a cost of $1.2 billion.[1,2] While the operation may reduce the risk of stroke compared to medical management, the currently available scientific evidence is insufficient either to reject or endorse this hypothesis.[2–5] Only two randomized trials on this question have been completed, each with inconclusive results.[6,7] Clinical uncertainly about the appropriate indications for the procedure is reflected in the high degree of regional variation in the utilization rates of carotid endarterectomy.[8,9] For example, residents of Boston, Massachusetts are twice as likely to undergo carotid endarterectomy as residents of New Haven, Connecticut.[10]

The potential benefit of the operation depends primarily on the operative mortality and the risk of stroke with medical or surgical management and high post-operative complication rates virtually eliminate the potential benefit of the operation for all patient subgroups.[3,11,12] Many argue that carotid endarterectomy should be restricted to specialized centers[13,14] or to those with documented operative stroke plus death rates under 5 or 6 percent.[15]

Published case series demonstrate wide variation in complication rates, with stroke plus death rates ranging from 3 percent to over 15 percent.[9,16–19] Three small studies found no significant increased risk in patients over the ages of 75 or 80 and advocated the use of the procedure in elderly patients.[20–22] Low surgical volume has been found to be associated with increased risk for many surgical procedures. Carotid endarterectomy has been specifically examined in two studies, neither of which found an effect of surgical volume on outcome. However, the statistical power of these two studies was limited, as was the spectrum of institutions included in their analyses.[23,24]

We used the Medicare claims files for the six New England states to address three issues: the effect of age on the operative risk of carotid endarterectomy, the association of low surgical volume with increased risk, and the evaluation of the performance of carotid endarterectomy at individual hospitals.

Methods

Data Source

Data for the current study were obtained from three computer files maintained by the Health Care Financing Administration:[25]

- the Medicare Provider Analysis and Review (MEDPAR) file, which contains computerized discharge abstracts for each hospitalization for Medicare enrollees and includes patient and hospital identifiers and data on diagnoses, procedures, and resource utilization;
- the Health Insurance Skeletonized Eligibility Writeoff (HISKEW) file which records whether a patient died and the date of death, regardless of whether that death occurred within a hospital; and
- the Provider of Services file, which describes the relevant characteristics of all Medicare institutional providers.

Selection of the Study Population

We reviewed all 2,171 hospital discharge abstracts submitted for New England resident Medicare beneficiaries 65 years of age and older discharged between April 1, 1984 and June 30, 1985 who underwent carotid endarterectomy (ICD-9-CM procedure code 38.12). After eliminating the 81 patients who received surgery outside New England and one patient with inconsistent coding, 2,089 patients were selected for inclusion in the study. If the endarterectomy was not listed as the primary procedure (n = 83, 3.8 percent of cases), we assigned the date of surgery for the index endarterectomy to the midpoint of the hospital stay. (We repeated all reported analyses using the date of admission as the date of surgery for these 83 cases; none of the analyses were affected.)

Outcomes

Deaths were determined by searching the write-off file. Stroke was ascertained by inference from the diagnostic codes recorded on the discharge abstract for the index admission and readmissions within 30 days of surgery. If patients' diagnosis codes could be consistent only with a post-operative stroke (n = 35), they were coded as having definite post operative strokes. If the coding of the stroke was ambiguous and thus might have represented a pre-operative stroke, the patient was coded as having a possible stroke (n = 161).* Based on these definitions, we calculated the

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*Details of coding available on request to authors.

proportion of patients who experienced the following outcomes within 30 days of surgery: death; death or definite post-operative stroke (the minimum estimate of the stroke plus death risk); death or any possible post-operative stroke, which represents the maximum risk of death plus stroke that can be ascertained from the discharge abstract (some additional strokes might be detected through chart review). Confidence intervals for these proportions were calculated according to the exact method.[26]

Independent Variables

Patients' age, sex ,and race were ascertained from the discharge record. To identify important medical conditions of patients, we used diagnostic codes from both the surgical (index) admission and from any admissions whose discharge date fell within the six months preceding the date of the index surgical procedure, as previously described.[27] If an index admission diagnosis could reasonably be interpreted as a potential complication of the endarterectomy, a patient was coded as having that condition only if it was also present on admissions that preceded the one in which the endarterectomy occurred. Variables evaluated as risk factors for adverse outcomes were: coronary artery disease, pulmonary disease, diabetes, renal disease, congestive heart failure, valvular heart disease, stroke, and the presence of any hospital admission during the previous six months.

Hospital surgical volume was defined based on the number of index carotid endarterectomies performed by each hospital on the Medicare population during the 15-month study period. Institutional size was defined as a continuous variable based on the number of hospital beds. Hospital teaching status was defined as a continuous variable based on the number of housestaff in accredited residency programs per hospital bed.

Analysis

We used multiple logistic regression to test the association between each of the three outcomes and the clinical and institutional predictors described above.[28] Of the clinical variables considered, only coronary artery disease was significantly associated with 30-day mortality after controlling for age, sex, and surgical volume. Both sex and coronary artery disease were included in the reported logistic regression models. Alternative specifications of the model, in which teaching status and hospital size were treated as both dichotomous and ordinal variables, were examined. Regardless of the specification of the model, the results were similar to those presented below. Two-way interactions between surgical volume and age, and age and sex were examined and were not significant.

We used the binomial distribution to test the hypothesis that the observed number of deaths plus definite strokes at an individual hospital exceeded the number that would have been expected had the hospital's outcomes equaled those of the highest volume hospitals.

Results

Operative Mortality

Operative mortality increased markedly with age (Table 1). Those over age 70 faced a progressively increased risk, with those over age 80 facing a more than four-fold increase in the adjusted odds of death compared to those ages 65 to 69. Operative risk was also higher in hospitals with low surgical volume on the Medicare population (Table 1). Patients undergoing carotid endarterectomy in hospitals in each of the three lower volume groups faced an increased risk of death compared to those receiving surgery in hospitals performing more than 40 endarterectomies per year. The increased risk associated with low-volume surgery was consistent across all specifications of the model examined. When a dichotomous variable was substituted for the three levels of lower hospital volume in an otherwise identical model, the adjusted odds ratio for 30-day mortality at hospitals performing 40 or fewer endarterectomies per year compared to higher volume hospitals was 2.83 (95% CI = 1.11, 7.19). When volume is treated as a continuous variable in the same model it is also significant ($p = .03$), and remains so after controlling for teaching status and hospital size.

TABLE 1—Death within 30 Days of Carotid Endarterectomy, Adjusted Odds Ratio and Risk by Patient Subgroup

Patient Age (years)	(N)	Adjusted Odds Ratio* (95% CI)	% Risk (95% CI)
65–69	(733)	1.00	1.1 (0.5, 2.1)
70–74	(671)	2.58 (1.12, 5.95)	2.8 (1.7, 4.4)
75–79	(473)	2.91 (1.22, 6.95)	3.2 (1.8, 5.2)
80+	(212)	4.46 (1.73, 11.50)	4.7 (2.3, 8.5)
Hospital Volume**	**(N)**	**Adjusted Odds Ratio* (95% CI)**	**% Risk (95% CI)**
Over 40	(457)	1.00	1.1 (0.3, 2.5)
21–40	(561)	2.63 (0.94, 7.32)	2.7 (1.5, 4.4)
6–20	(913)	2.90 (1.10, 7.62)	3.0 (2.0, 4.3)
5 or less	(158)	3.14 (0.89, 11.06)	3.2 (1.0, 7.2)
Total	(2089)	—— Average	2.5 (1.9, 3.3)

*Derived from multiple logistic regression with age, sex, surgical volume, and presence or absence of coronary artery disease as covariables. The first level of each category is the reference.

**Volume defined as the number of carotid endarterectomies performed per year on the Medicare population at each hospital.

Risk of Death or Stroke

Because we were unable to characterize the timing in relation to surgery of the majority of strokes recorded on the discharge abstract, we present both a minimum and maximum estimate of post-operative death plus stoke risk in Table 2. Regardless of the definition used, the risk of death or stroke increases significantly with age and at lower volume hospitals.

Individual Hospital Outcomes

We identified all hospitals where the number of observed deaths or definite strokes was greater than what would have been expected ($p < .10$), using as the standard of comparison the outcome rate in those hospitals performing more than 40 carotid endarterectomies per year (Table 3). Twenty-two of the 139 New England hospitals performing endarterectomy were found to have high complication rates compared to high volume hospitals. Only seven New England hospitals performed more than 40 carotid endarterectomies per year on the Medicare population, while 50 hospitals performed five or fewer.

Discussion

Several authors have found no increase in operative mortality in the elderly and have argued that age should not be a consideration when choosing endarterectomy.[21–23] In

TABLE 2—Death or Stroke within 30 Days of Carotid Endarterectomy Adjusted Odds Ratios (AOR)* and Risk by Patient Subgroup

		Death or Definite Stroke		Death or Possible Stroke	
Patient Age (years)	(N)	AOR (95% CI)	% Risk (95% CI)	AOR (95% CI)	% Risk (95% CI)
65–69	(773)	1.00	2.7 (1.7, 4.2)	1.00	8.7 (6.8, 11.0)
70–74	(671)	1.59 (0.89, 2.84)	4.3 (2.9, 6.2)	1.61 (1.15, 2.27)	13.1 (10.7, 15.9)
75–79	(473)	1.87 (1.02, 3.44)	5.1 (3.3, 7.5)	1.54 (1.06, 2.25)	12.7 (9.8, 16.0)
80+	(212)	2.54 (1.25, 5.14)	6.6 (3.7, 10.8)	2.21 (1.42, 3.45)	17.0 (12.2, 22.7)
Hospital Volume**	(N)	AOR (95% CI)	% Risk (95% CI)	AOR (95% CI)	% Risk (95% CI)
over 40	(457)	1.00	2.0 (0.9, 3.7)	1.00	9.2 (6.7, 12.2)
21–40	(561)	2.52 (1.16, 5.44)	4.6 (3.0, 6.7)	1.19 (0.78, 1.80)	10.9 (8.4, 13.7)
6–20	(913)	2.55 (1.23, 5.28)	4.7 (3.4, 6.3)	1.46 (1.00, 2.12)	13.0 (10.9, 15.4)
5 or less	(158)	3.13 (1.22, 8.07)	5.7 (2.6, 10.5)	1.96 (1.15, 3.32)	16.5 (11.0, 23.2)
Total	(2089)	—	4.2 (3.3, 5.1)	—	11.9 (10.5, 13.3)

*Derived from multiple logistic regression with age, sex, surgical volume, and presence or absence of coronary artery disease as covariables. The first level of each category is the reference.

**Volume defined as the number of carotid endarterectomies performed per year on the Medicare population at each hospital.

TABLE 3—Number of Hospitals Identified as Exceeding Outcome Thresholds by Surgical Volume

Volume of Operations per Year	Number of Hospitals in Group	Hospitals Identified with Definite Stroke plus Death Rates Significantly above Expected ($p \leq .10$)*
1 to 5	50	6
6 to 20	65	10
21 to 40	17	6
Over 40	7	0
Total	139	22

*For each hospital, the expected proportion of definite strokes plus deaths is assumed to be .0197, the proportion that occurred in the high-volume hospitals. Given this proportion, the probability that a hospital would have at least as many strokes plus deaths as actually reported is computed. A hospital where this probability is .10 or less is considered to have a significantly high rate. The number of definite strokes plus deaths is assumed to have a binomial distribution.

contrast, our study demonstrates a markedly increased mortality risk with increasing age. Two-thirds of the New England Medicare patients undergoing endarterectomy were over the age of 70 and faced an average three-fold increased risk of operative death compared to younger patients. Patients undergoing surgery at low-volume hospitals also faced a three-fold increased risk of death compared to those receiving surgery at high-volume hospitals.

Our analyses of post-operative stroke risk are consistent with the findings of our mortality analyses, but leave uncertainty about the actual risk of non-fatal stroke following endarterectomy. Several lines of evidence suggest that the actual risk is considerably higher than our minimum estimate of death plus stroke risk (4.2 percent). First, a population-based sample of Medicare patients undergoing carotid endarterectomy in 1981 were found to have death plus stroke risks of 9.8 percent overall, based on review of hospital records.[29] Second, each of the five recent series that described the outcomes of carotid endarterectomy across multiple hospitals report a ratio of non-fatal strokes to deaths of approximately two-to-one.[23,29–32] Applying this ratio to our data would suggest a stroke plus death risk of 7.5 percent overall, high enough to raise serious questions about the relative risks and benefits of endarterectomy in the elderly.[29]

A potential criticism of our findings is that the data on disease coded in discharge abstracts may not provide sufficient information to adequately control for severity of illness and comorbidity.[33] Although we share this concern,[27] particularly with respect to the comparison of aggregate hospital mortality rates, the limitations of the diagnostic coding should not undermine our current analysis. It is clear from both the Rand consensus panel's conclusions[12] and from decision analysis[11] that high post-operative complication rates virtually eliminate the potential benefit of the operation for all patient subgroups, no matter what assumptions are made about the operation's efficacy or the stroke rate among medically managed patients. Thus patients who undergo surgery at hospitals which experience high rates of post-operative morbidity and mortality are unlikely to benefit from the surgery, regardless of whether the high rate is due to patient selection or to poor technical performance of the procedure itself. In fact, severity adjustment could obscure important differences in outcomes across institutions due to what may be inappropriate patient selection.

Given the current uncertainty about the value of carotid endarterectomy, the importance of operative risk to the expected benefit of the procedure and the wide variation in post-operative complication rates at individual hospitals, patients and their physicians should seek reliable data on the operative risk of death and stroke at the hospital where the operation is to be performed. These data are currently unavailable. One solution would be for individual hospitals to review their records and report their results. Such a moni-

toring program would be limited by the degree to which hospitals could ascertain post-discharge events and by concern about the potential conflict of interest for hospitals collecting and reporting the data.

Our study suggests a practical alternative for the 70 percent of patients undergoing endarterectomy who are over the age of 65[1] and thus eligible for Medicare. Data in the claims files could be used to establish an ongoing monitoring program that would identify all patients undergoing endarterectomy and ascertain post-operative mortality. Chart reviews could be carried out to ascertain post-operative stroke rates and accurately classify patients' severity of illness and comorbidity. Accurate risk stratification, whether by age or clinical indications or both, would improve hospital comparisons and enhance individual clinical decision-making. The effort required to establish such a monitoring program would be far less than that required to establish an independent prospective data collection system to provide patients and physicians with accurate information on institution-specific operative risks.

One limitation of any monitoring program is that hospitals where few operations are performed cannot be reliably evaluated; small numbers make it impossible to distinguish the few with excellent outcomes from those with worse than average performance.[34] Given the importance of a low complication rate to the potential benefit of carotid endarterectomy, the difficulty of evaluating low volume hospitals' outcomes and the demonstrated volume-outcome association, it may be reasonable to restrict carotid artery surgery to high volume hospitals that demonstrate excellent outcomes.

The controversy surrounding carotid endarterectomy has been heightened by recent reports confirming that a significant proportion of the carotid endarterectomies performed in the United States are carried out in clinical settings where experts believe the risk outweighs the potential benefit.[11,29] Our findings of increased risk in the elderly and at low-volume hospitals are consistent with this conclusion. An ethical solution to the controversy would be to provide patients with the best available information on the potential benefits and risks of alternative treatments so that the decision may reflect their values.[34] The Medicare claims files, supplemented as necessary with chart review, provide a readily available and feasible method to improve the quality of information available to patients and their physicians.

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Geographic Variation in the Onset of Decline of Male Ischemic Heart Disease Mortality in the Netherlands

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Abstract: We studied variation in the year of onset of ischemic heart disease mortality decline among regions (n = 39) in the Netherlands. Using loglinear regression methods, a quadratic regression model was fitted to the observed numbers of male deaths in each region in the period 1950–84, controlling for changes in age-structure of populations. The quadratic regression model proved inadequate to describe the mortality experience of females.

For the country as a whole, the estimated year of onset of male ischemic heart disease mortality decline is 1973.9. The difference between the earliest and the latest region is almost nine years (1970.0 and 1978.9, respectively). An early onset of decline (≤1972) is only found in the urbanized, western part of the country. A later (≥1975) onset of decline is characteristically found in more peripheral regions in the South-West and South-East, as well as in the North.

Exploratory correlation and regression analyses show that both average income and percent of population living in larger cities have independent, negative associations with the year of onset of male ischemic heart disease mortality decline.

We argue that regional variation in the timing of lifestyle changes is a more plausible explanation of these observations than regional variation in the timing of medical care improvements. (*Am J Public Health* 1989; 79:1621–1627.)

Introduction

After more than 30 years of rising death rates from ischemic heart disease, a dramatic decline has occurred in many industrialized countries, with large differences in the year of onset of decline.[1]

In the United States, the mortality rates plateaued in the early 1960s, and had definitely started to decline in 1968.[2–4] In Australia, death rates from ischemic heart disease reached their highest levels in 1965–67,[5] and in New Zealand in 1968.[6]

In Europe, the decline started in the first half of the 1970s in Denmark, Norway, Finland, the Federal Republic of Germany, Belgium, the Netherlands, and England and Wales. On the other hand, in Ireland, France, Switzerland, Austria, Italy, and Sweden, ischemic heart disease mortality started to decline in the second half of the 1970s. In most Eastern European countries, including Hungary, Poland and Romania, mortality rates were still increasing at that time.[1]

These international differences suggest a process of gradual and worldwide diffusion of the factors responsible for ischemic heart disease mortality decline: from a focus in the New World (including Australia and New Zealand) to Europe, and within Europe from West to East.

Such a process of gradual diffusion has also been found within the United States. Two studies of geographic variation in the year of onset of ischemic heart disease mortality decline within the United States have been reported, showing a considerable range of variation between State Economic Areas[7] and States,[8] respectively. Early declines were reported for California[9,10] and New York.[11] In general, metropolitan areas had earlier years of onset of decline than non-metropolitan areas, suggesting that the factors responsible for the mortality trend reversal have spread across the population according to a general pattern of cultural diffusion.[7]

In the Netherlands, age-standardized mortality from ischemic heart disease reached its highest level in 1972, for both males and females (Figure 1).

Among males, ischemic heart disease death rates more than doubled from 105 per 100,000 person-years in 1950, to 236 per 100,000 person-years in 1972. Thereafter, a gradual

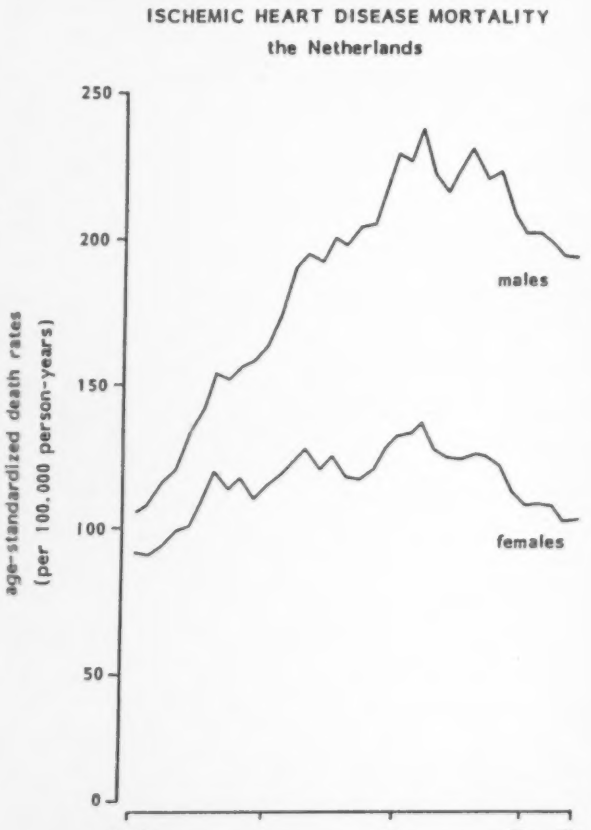

FIGURE 1—Age-Standardized Trends in Mortality from Ischemic Heart Disease, by Sex, the Netherlands, 1950–84

ICD-code numbers	ICD-6/7 1950–68	ICD-8 1969–78	ICD-9 1979–
Ischemic heart disease	420,422.1	410–414	410–414, 429.2

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decline set in, with a secondary peak in 1976, to a rate of 191 per 100,000 person-years in 1984. Among females, the increase between 1950 and 1972 was much less steep than in males, but the decrease after 1972 was almost as rapid.

The causes of this trend reversal have not been extensively investigated in the Netherlands. There is some evidence for a reduction of serum cholesterol between 1960 and 1979.[12,13] The prevalence of self-reported smoking (but not the number of cigarettes sold per capita) has been declining since 1958 in males and since 1970 in females.[14,15] There is no information on trends in the prevalence or the detection and treatment of hypertension in the Netherlands. Changes in the medical management of patients with ischemic heart disease have probably mirrored those in other industrialized countries, but quantitative information on trends at the national level is not available.

The objective of our study of regional variation in the year of onset of mortality decline is to provide some clues to the causes of the mortality trend reversal for ischemic heart disease: if a pattern of geographical variation has been identified, it will be easier to find the factors responsible for the trend reversal, because these must have followed the same pattern. Because of estimation problems in females, we will report only on variation in the year of onset of decline in males.

Methods

Numbers of deaths by age (<1, 1–4, 5–14, ... 85–94, 95+ years), sex, calendar-year (1950–84), region, and four-digit-number of the International Classification of Diseases (ICD) were available from a large computer file supplied by the Netherlands Central Bureau of Statistics.

For statistical purposes, the Netherlands have been divided into 40 regions, each representing a geographical area serviced by one or more larger cities (Figure 2). Total population of the Netherlands was 14.4 million in 1984; the median population size of these 40 regions was approximately 300,000 in 1984.

The economic and cultural center of the country is located in the largely urbanized quadrangle bordered by the regions containing the four largest cities of Amsterdam, The Hague, Rotterdam, and Utrecht. In the rest of the country, a number of secondary centers is found (indicated in Figure 2, which shows the location of other cities with more than 100,000 inhabitants), with other regions occupying a more "peripheral" position, from both a geographic and a cultural point of view. Region no. 40, land which was recently claimed from the waters of the former Zuiderzee, was excluded from the analysis because its population was very small before the end of the 1970s.

For ischemic heart disease, ICD-revisions 6 and 7, we included 420 (arteriosclerotic heart disease) and 422.1 (other myocardial degeneration with arteriosclerosis). The latter number was included because bridge-coding between revisions 7 and 8 revealed that this category was transferred to the ischemic heart disease-section in ICD-revision 8, 410-414.[16] For ICD-revision 9 we included 410-414 (ischemic heart disease) and 429.2 (cardiovascular disease, unspecified), the latter because bridge-coding between revisions 8 and 9 showed that its separation from ischemic heart disease in revision 9 was the main factor behind the lower-than-1 comparability ratio.[17] As Figure 1 shows, the mortality trend shows no signs of sudden changes at the moments of introduction of new ICD-revisions (1969 for ICD-8, and 1979 for ICD-9), suggesting that comparability over time of our cause-of-death category "ischemic heart disease" is satisfactory.

FIGURE 2—Map of the Netherlands, Showing Division into 40 Statistical Regions and Location of Cities with More than 100,000 Inhabitants

Higher age-groups were not excluded from the analysis, because declines in ischemic heart disease mortality have occurred at all ages, and have even been remarkably uniform in the Netherlands.[18]

Population numbers by age, sex, calendar-year, and region were available from several sources based upon the Dutch population register which is kept up to date continuously. Approximate person-years at risk for the deaths occurring in a given calendar-year were calculated by averaging the population numbers for December 31 of both the current and previous years.

Loglinear regression analysis was used to obtain estimates for the years of onset of decline of ischemic heart disease mortality. We chose the loglinear model because our dependent variable is a person-time rate. The model was fit by assuming a Poisson distribution for the number of cases observed. For each region a quadratic regression equation was fit to the observed number of deaths with the following structure:

$$E(y_i) = N_i e^{\alpha_i + \alpha + \beta_1 X + \beta_2 X^2}$$

in which:

- $E(y)$ = expected number of deaths
- N = person-years at risk
- e = base of natural logarithm
- α = national mortality level, 1950
- α' = regional departure from national mortality level, 1950
- β_1, β_2 = regional mortality trend parameters

X = calendar-year
i = subscript denoting age-group.

We used national mortality levels by age (α_i) to adjust the regional trend parameters for changes in age-composition of the population. From β_1 and β_2 the year of maximum mortality rates, marking the onset of decline, can easily be calculated. The variance and covariance estimates of β_1 and β_2 from the regression calculations and a Taylor series expansion[19] were used to obtain variance estimates for the years of maximum mortality. An approximate asymmetric 95 percent confidence interval was derived with the help of Fieller's theorem.[20] The calculations were performed with the GLIM-package.[21]

In order to test the goodness-of-fit of our quadratic regression model, we inspected plots of observed and expected (under the model) numbers of deaths and performed a Wald-Wolfowitz "runs test" on the residuals.[22] Both methods suggested a considerably better fit in males than in females.

In males, the estimated years of onset of mortality decline closely correspond to what one would guess on the basis of visual inspection. At the national level, for example, the estimate is 1973.90, slightly later than 1972 due to the secondary peak in 1976, but still a satisfactory measure. In females, however, the estimated years of onset are considerably earlier than 1972, due to the flat rise and steep decline of the curve (Figure 1). At the national level the estimate is 1969.03.

The "runs test" identified only two regions with statistically significant ($p < .05$) deviations from a quadratic model in males (to be expected from chance alone), but four in females. In these cases, fitting higher order polynomials produced more satisfactory fits, but did not change the estimated year of onset of decline in males. In females, however, it produced very different estimates, suggesting that the quadratic regression model is really inadequate. For these reasons we decided to restrict this presentation to the results obtained for males.

The national number of deaths from ischemic heart disease among males over the entire period 1950–84 was 412,613. Regional numbers varied from 1,744 to 41,785, with 8,121 deaths in the region with the median population size.

Results

The estimated years of maximum male mortality from ischemic heart disease, marking the year of onset of mortality decline, are presented in the appendix, together with their 95% confidence intervals. Even in a small country like the Netherlands, the difference between the earliest and the latest region is almost nine years: the range of variation is from 1970.0 to 1978.9. Although confidence intervals are sometimes wide, especially for smaller regions, there is no overlap between the confidence intervals for regions with an early onset of decline (≤1972) and those with a late onset of decline (≥1977).

There appears to be a bimodal frequency distribution of the year of onset of decline of ischemic heart disease mortality among males in Dutch regions (Figure 3). The first peak in the distribution represents 17 regions (containing 50 percent of the Dutch male population) with an estimated year of onset of decline in 1973 or 1974, i.e., corresponding to the national average. Only six regions (containing 26 percent of the Dutch male population) had years of onset of decline before 1973. After 1974, a smaller second peak in the

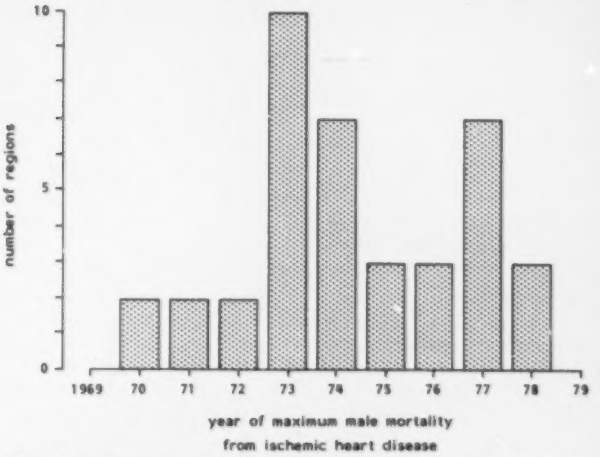

FIGURE 3—Frequency Distribution of the Year of Maximum Male Mortality from Ischemic Heart Disease, 39 Regions, the Netherlands

frequency distribution appears in 1977. This second peak may partly explain the second peak in the national mortality trend in 1976 (Figure 1), although the years do not exactly correspond.

Figure 4 illustrates the spatial distribution of the year of

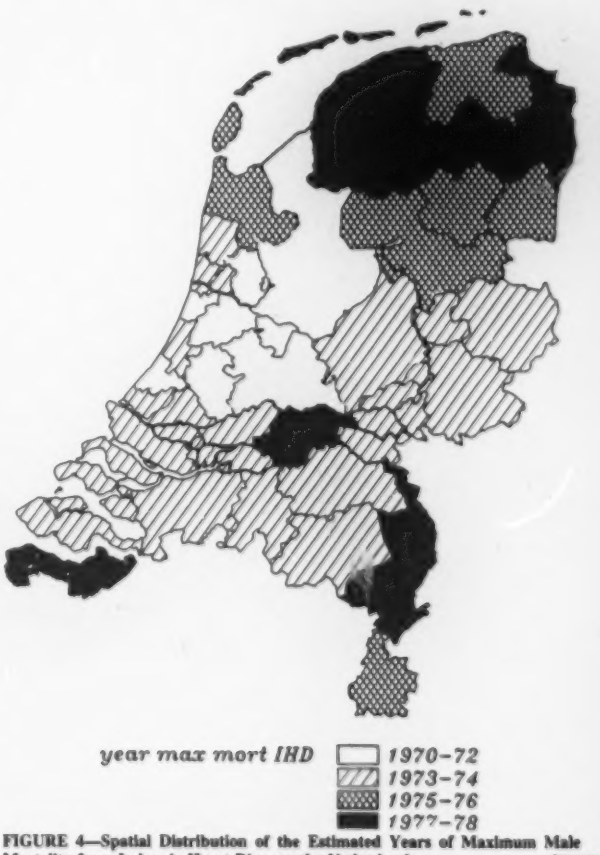

FIGURE 4—Spatial Distribution of the Estimated Years of Maximum Male Mortality from Ischemic Heart Disease, the Netherlands

onset of decline of ischemic heart disease mortality among males in the Netherlands.

An early onset of decline (≤1972) is only found in a number of regions located in the urbanized, western part of the country, including the regions containing Amsterdam and the Hague, but not the region containing Rotterdam. A later (≥1975) or late (≥1977) onset of decline are characteristically found in more peripheral regions in the South-West and South-East, as well as in the North. There is only one exception: region no. 16, very centrally located from a purely geographical point of view but rural in character, had its year of onset of mortality decline in 1977.

Regional differences in the year of onset of mortality decline represent important differences in the timing of the ischemic heart disease mortality "epidemic" as a whole. Figure 5 shows two "cross-sectional" views of ischemic heart disease mortality among males, for the early 1950s and early 1980s, respectively. In 1980–84, the regions in the highest quintile of the distribution were mainly located in the North and South-East, parts of the country which were also characterized by a late onset of mortality decline.

This pattern gradually emerged from a completely different pattern of variation in the early 1950s, when areas with higher than average mortality were mainly found in the West. (The product-moment correlations between year of onset of decline and mortality levels in 1980–84 and 1950–54 were 0.69 and −0.48, respectively.) This suggests that the epidemic of ischemic heart disease mortality started and ended earlier in the western parts of the Netherlands.

In an exploratory search for covariates of regional differences in the year of onset of male ischemic heart disease mortality decline we calculated product-moment correlation coefficients with a number of sociodemographic variables, all measured in a year close to (mostly just before) 1973 (Table 1).

The correlations confirm the impression that lower values of year of onset of decline are found in more urbanized areas: the correlation coefficient with percentage population living in larger cities is 0.74. On the other hand, the year of onset of decline also appears to be strongly associated with socioeconomic characteristics: lower average income goes together with a later onset of mortality decline.

Two variables measuring another important cultural division, percentage population of protestant c.q. roman-catholic religious affiliation, usually powerful predictors of mortality patterns in the Netherlands, are not associated with the year of onset of ischemic heart disease mortality decline.

The results of a multiple regression analysis (Table 2) show that both average income and percentage living in larger cities have an independent, negative association with year of onset of decline. After controlling for these two powerful predictors two additional socioeconomic variables appear to be associated with

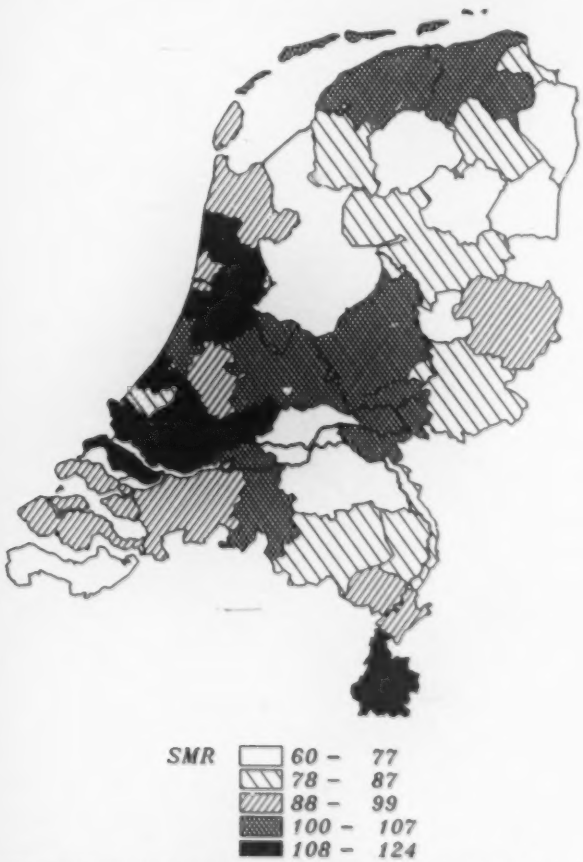

FIGURE 5a—Spatial Distribution of Age-Standardized Ratios of Male Ischemic Heart Disease Mortality, the Netherlands, 1950–54

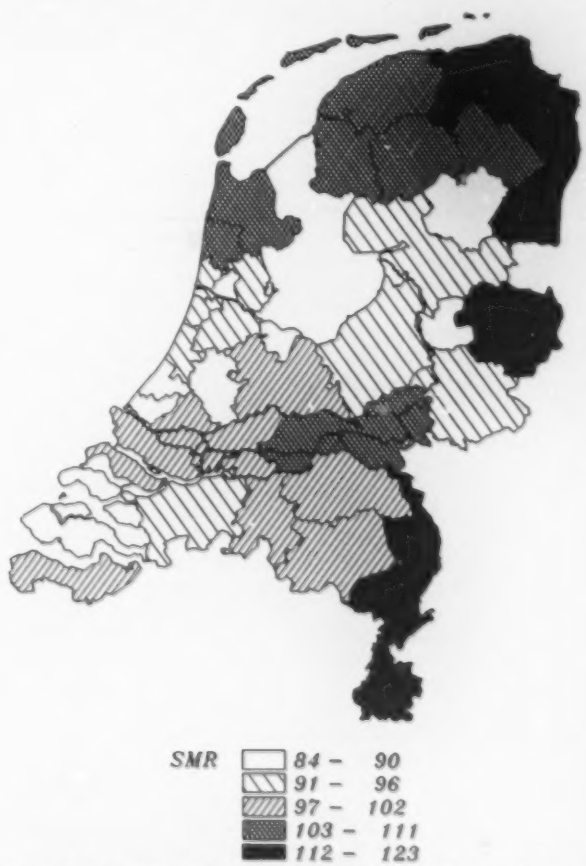

FIGURE 5b—Spatial Distribution of Age-Standardized Ratios of Male Ischemic Heart Disease Mortality, the Netherlands, 1980–84

TABLE 1—Correlations between the Year of Onset of Male Ischemic Heart Disease Mortality Decline and Sociodemographic Variables, the Netherlands

Variables[a]	Mean	Coefficient of Variation	Simple Correlation Coefficient[b]
Average income per inhabitant (guilders, in 000s)	4.95	0.13	−0.79
% Working population unemployed	1.14	0.40	0.32
% Economically active population working in agriculture	2.09	0.80	0.34
% Economically active population working in industry	38.4	0.26	0.23
% Population living in larger cities	23.5	1.00	−0.74
% Population of protestant denomination	37.5	0.55	0.25
% Population of roman-catholic denomination	37.0	0.77	−0.08
% Population without Dutch nationality	1.73	0.55	−0.26

[a]measured 1969–72
[b]product-moment correlation coefficient r

TABLE 2—Regression of the Year of Onset of Male Ischemic Heart Disease Mortality Decline on Sociodemographic Variables, the Netherlands

Variable[a]	Multiple Regression Coefficient[b]	95% Confidence Interval
Average income	−1.693	−2.695, −0.691
% Living in larger cities	−0.046	−0.070, −0.022
% Working in industry	−0.053	−0.096, −0.010
% Unemployed	1.122	0.168, 2.076

$R^2 = 0.773$
[a]Variables were selected from those included in Table 1 in a forward stepping procedure on the basis of F-tests, and are mentioned in the order of inclusion into the regression equation.
[b]Ordinary least squares regression coefficient β.

the year of onset of decline: percentage working in industry (negative association) and percentage unemployed (positive association). The percentage of variance explained by these four variables is considerable, 77 percent.

Discussion

In their analysis of the onset of decline of ischemic heart disease mortality in the United States, Ragland and associates found a range of variation between States of almost 15 years for White males aged 55–64 years, from 1952.82 in California to 1967.29 in West Virginia.[8] If their units of analysis had been smaller, they would probably have found an even wider range of variation. This is evident from the study of State Economic Areas by Wing and associates, who found that 1.6 percent of these areas had a year of onset of decline as late as in or after 1973.[7] We feel that a range of variation of almost nine years between regions in such a small country as the Netherlands is no less dramatic.

Although differences in cause-of-death certification are a potential threat to the validity of all studies of geographical variation in cause-specific mortality, it is implausible that such differences could account for the variation in year of onset of decline of ischemic heart disease mortality observed in this study. As changes in certification have probably not contributed much to the trend reversal of ischemic heart disease mortality at the national level,[4] it is difficult to see how any regional differences in such changes could have produced such dramatic variation in year of onset of decline. Moreover, the validity of Dutch cause-of-death certification compares favorably with that of many other European countries.[23,24] Coding of death certificates is done centrally in the Netherlands.

Another potential threat to the validity of our study might arise from violations of the assumptions underlying the quadratic regression model (i.e., a unimodal distribution of mortality over time, and equal rates of increase before and decrease after the top-year). These assumptions were reasonably satisfied for males, however. Although the national mortality trend for males is bimodal (Figure 1), this appears to be the result, at least in part, of the superposition of two unimodal trends with a different timing (Figure 2). For females, the rate of increase before the early 1970s is much smaller than the rate of decrease thereafter, so that a quadratic regression model cannot be used to estimate years of maximum mortality. This is unfortunate, because a comparison of regional patterns for females with those for males could give important additional information. Further study of female ischemic heart disease mortality decline in the Netherlands, using an appropriate model form, is therefore to be recommended.

Despite a vast amount of literature, there is still no consensus on the relative contributions of changes in risk factor levels and improvements in medical care to the reduction of ischemic heart disease mortality.

A decline in incidence of myocardial infarction has been found in a number of population-based studies, both from the United States[25–27] and from a few other countries.[28–30] Lifestyle changes, leading to reduced tobacco consumption and lower serum cholesterol levels, have almost certainly contributed to this decline.[4,6,31,32]

Examinations of trends in myocardial infarction survival have produced a more diffuse picture than those of trends in incidence. Some studies have reported a decrease in in-hospital case fatality rates[26,27] but others have not done so.[33,34] Studies which have examined long-term survival reported no improvements.[26,35,36] (In most of these studies, the time-periods examined preceded widespread use of beta-blocking agents for long-term patient management.) Nevertheless, a recent comparison of two cohorts suggested that early detection and treatment did contribute to the observed decline in ischemic heart disease mortality.[37] Estimates of the contribution of specific medical interventions to the decline in the United States between 1968 and 1976 range as high as 40 percent.[32]

Although the pattern of international variation in onset of decline mentioned in the introduction could be consistent with a diffusion process of either lifestyle or medical care innovations, a detailed comparison of available data suggests that lifestyle factors are the better candidates.[38] Sweden is a particularly enlightening case because it combines a very advanced health care system with a late year of onset of decline of ischemic heart disease mortality.[39]

It is important to note that the spatial distribution of the year of onset of ischemic heart disease mortality decline within the Netherlands is analogous to that observed in the

United States.[7,8] In both countries the decline started in the highly urbanized areas which are regarded as the cultural center(s) of the nation, and gradually diffused to more "peripheral" regions. In the Netherlands, as in the United States, the factors responsible for the mortality trend reversal thus seem to have spread according to a general pattern of cultural diffusion. In the Netherlands, as in the United States,[40] socioeconomic factors seem to play an important role in this diffusion process. Theoretically, both changes in medical care (improved control of hypertension and/or better management of patients with cardiovascular disease) and changes in lifestyle could have spread according to such a pattern. For a number of reasons, however, we believe that differences in timing of lifestyle changes are a more plausible explanation for the Netherlands:

- If differences in timing of changes in medical care were to explain the regional variation in onset of decline of ischemic heart disease mortality, one would have to assume that the providers of medical care in the most "backward" regions adopted these improvements almost nine years later than those in the most "advanced" regions. In view of the short lines of communication within the Dutch medical care system, and of the geographically homogeneous insurance schemes, this seems a very unrealistic assumption. Diffusion of changes in lifestyle in the population at large, on the other hand, is probably a much slower process.
- If the degree of urbanization were an indicator of early diffusion of innovations in the medical care of ischemic heart disease, one would also expect an association between the presence of a university hospital, as a typically metropolitan and diffusion-prone institution, and the year of onset of mortality decline. In 1970, there were seven university hospitals in the Netherlands, all located in cities with 100,000+ population. The average year of onset of male ischemic heart disease mortality decline in regions with a university hospital was 1973.6, almost exactly the same as the national average. This is due to the fact that one university hospital is located in the North (region no. 3, with its year of onset of decline in 1976), and only three are located in regions with an early decline (one in no. 17 and two in no. 23, with their years of onset of decline in 1972 and 1971, respectively). Also after controlling for average income, there is no association between the percentage of hospital beds located in university hospitals and the year of onset of decline.
- The association between year of onset of decline and average income suggests that an early adoption of factors promoting ischemic heart disease mortality decline is not only determined by circumstances directly linked to living in large cities, but also to the more favorable socioeconomic circumstances prevailing in the western parts of the country. This suggestion is supported by the results of analyses at the individual level from England and Wales, which have shown that ischemic heart disease mortality decline started earlier in higher socioeconomic groups.[41] In England and Wales, this variation corresponds to differing trends in lifestyle related risk factors such as smoking and high cholesterol intake.[42] In the Netherlands, too, the decline in smoking among males started in the higher socioeconomic groups.[14]

APPENDIX

Estimated Years of Maximum Male Mortality from Ischemic Heart Disease, by Region, the Netherlands

Region[a]	Top-year	95% confidence intervals
1	1977.2	1975.8, 1979.0
2	1977.5	1974.4, 1984.5
3	1976.9	1975.6, 1978.7
4	1977.1	1974.7, 1978.0
5	1978.0	1975.5, 1982.4
6	1977.8	1976.1, 1980.5
7	1977.7	1975.8, 1980.6
8	1976.2	1974.9, 1977.9
9	1976.5	1974.7, 1979.4
10	1975.1	1974.0, 1976.6
11	1974.2	1973.0, 1976.0
12	1973.7	1973.2, 1974.4
13	1974.9	1973.8, 1976.4
14	1974.8	1973.7, 1976.3
15	1973.6	1972.9, 1974.3
16	1977.0	1975.3, 1979.6
17	1972.9	1972.3, 1973.7
18	1975.7	1974.3, 1977.6
19	1974.8	1972.8, 1987.2
20	1973.0	1971.7, 1974.8
21	1970.2	1969.4, 1971.2
22	1973.2	1971.2, 1976.5
23	1971.5	1971.1, 1972.0
24	1971.4	1970.5, 1972.5
25	1973.6	1972.3, 1975.3
26	1970.0	1969.6, 1970.5
27	1973.6	1972.4, 1975.3
28	1972.7	1971.7, 1973.9
29	1973.2	1972.7, 1973.9
30	1974.0	1973.0, 1975.4
31	1977.2	1974.8, 1981.3
32	1974.3	1972.8, 1976.5
33	1973.7	1973.0, 1974.6
34	1973.1	1972.3, 1974.0
35	1974.2	1973.5, 1975.0
36	1973.4	1972.8, 1974.0
37	1978.3	1976.5, 1981.1
38	1978.9	1976.7, 1982.6
39	1975.0	1974.3, 1975.9
Netherlands	1973.9	1973.7, 1974.1

[a]see Figure 2

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WHEN: June 11–25, 1990

WHERE: Stockholm, Sweden

CO-SPONSORS: People to People International, University of Missouri-Kansas City

COURSE: 3 Credit Hours; CEUs available

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The program includes weekend homestay with Swedish families. For more information, contact: Dr. Alan M. Warne, Vice President for Programs, People to People International, 501 East Armour Blvd., Kansas City, MO 64109; tel: 816/531-4701.

Effect of a Gatekeeper Plan on Health Services Use and Charges: A Randomized Trial

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Abstract: A randomized trial was conducted to determine the effectiveness of a health care plan which uses physicians as gatekeepers to control health services use and charges. New enrollees in United Healthcare (UHC), an independent practice association, were randomly assigned to the standard UHC plan requiring a gatekeeper, or to an alternate plan with equal benefits but without a gatekeeper. Individuals in both plans were similar in demographic characteristics, perceived health status, and other health insurance coverage. The gatekeeper plan had 6 percent lower total charges per enrollee than the plan without a gatekeeper. There were minor differences in hospital use and charges. Ambulatory charges were $21 lower per person per year in the plan with a gatekeeper (95% CI = −39.9, −2.1) and these were due to .3 fewer visits to specialists (95% CI = −0.50, −0.10). We conclude that a health plan which incorporates incentives and penalties for physicians to act as gatekeepers can reduce the cost of ambulatory services by limiting specialist visits. (*Am J Public Health* 1989; 79:1628–1632.)

Introduction

Many health plans use a variety of mechanisms to encourage primary care physicians to act as "gatekeepers" in managing the medical care of their patients.[1,2] Incentives and penalties presumably will control cost by changing the gatekeeper's own practices and by limiting inappropriate hospital and referral care. The effectiveness of gatekeeper plans depends on the ability of participating physicians to coordinate the medical care system efficiently.

Gatekeepers are used by many health maintenance organizations (HMOs), independent practice associations, preferred provider organizations, and some Medicaid plans,[3,4] but evidence that they save money is lacking.[5] Descriptive data exist showing that gatekeepers can decrease the use of services if the physicians share financial risk of providing care,[6–8] but there are no data from controlled studies.

We therefore conducted a randomized trial of a plan using primary care physicians as gatekeepers to control health services use and charges. We randomly assigned study participants to United Healthcare, an independent practice association using gatekeepers, or to Northwest Healthcare, a plan with the same benefits but no gatekeeper. Because the economic incentives in the gatekeeper plan encouraged use of the gatekeeper and discouraged hospital and referral care, we expected that United Healthcare would have fewer hospitalizations, fewer visits to specialists, and lower use of procedures.

Methods

Population and Random Assignment

United Healthcare (UHC) was one of three health care plans available to Washington State employees in the Seattle metropolitan area during the 1979 open enrollment. All subscribers (n = 1,239) and their family members who enrolled in UHC for the first time were asked to participate in the experiment and complete an initial telephone interview. Retirees were excluded from the study.

The 90 percent of subscribers (families) who agreed to participate were assigned to a health plan according to a randomization scheme stratified by previous health care plan and type of employer, because State health insurance information showed that use of services differed significantly for these subgroups.[9] Previous health care plans were: Blue Cross of Washington and Alaska, which provided comprehensive inpatient and outpatient coverage; Group Health Cooperative of Puget Sound, a staff model HMO; or no previous State health plan, for new employees. The two types of employer were: an institution of higher education, or other State agency. We assigned 555 subscribers and their dependents (1,419 enrollees) to the standard United Healthcare plan with a gatekeeper and 558 subscribers (1,408 enrollees) to the plan without a gatekeeper, Northwest Healthcare.

The Two Plans

Both plans were offered by the SAFECO Insurance Company and had the same insurance premiums and benefits. Premiums for the subscriber and all dependents were paid in full by the State. Both plans provided inpatient, outpatient, and preventive care coverage with copayments and limits on maternity and mental health services. There was no deductible or coinsurance, but there were copayments of $2 for each prescription and $25 for each general physical examination.

The plan with a gatekeeper, United Healthcare, attempted to contain costs by enlisting primary care physicians in private practice as gatekeepers. The details of this plan have been published earlier.[10] When the present study began, most gatekeepers had been in the plan for two years. Hospitalization and other major procedures entailed no utilization review, prior authorization, or mandated second opinions, and there was little emphasis on quality assurance or provider education. Because gatekeepers had only 25 plan patients on average, capitation reimbursement was not practical, and almost all were reimbursed at 95 percent of their charges. Thus, primary care physicians had no financial incentive to limit costs of the care they provided.

There were stronger incentives for gatekeepers to monitor referral care. In 1979, referral care consumed 74 percent of the amount paid for health services by United Healthcare.[9] A risk-sharing account was established for each gatekeeper based on the age and sex of his or her panel of enrollees. These funds were then used to cover hospitalization, drugs, specialty, ancillary, and other referral costs. Catastrophic

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costs greater than $5,000 per patient were paid by a reinsurance program. At the end of the year, the gatekeeper shared with United Healthcare any deficit or surplus remaining in the account. If there was a surplus in the account at the end of the year, it was split equally between the gatekeeper and UHC. If there was a deficit, the gatekeeper had to pay back what was over-spent, up to an amount equal to 10 percent of his fee-for-service charges. The financial risk was not great to the physician. If the account was overdrawn, the minimum reimbursement rate was 85 percent (95 percent initial payment minus 10 percent), which was considered to be a reasonable return on charges. Furthermore, the risk applied to only a small proportion of the physician's total practice.

UHC gatekeepers could refer patients to the specialists or hospitals of their choice. Referrals could be made only to a few types of non-physician health professionals such as optometrists, psychologists, chiropractors, and occupational and physical therapists. There were no financial incentives for specialists, non-physicians, or hospitals to practice efficient care, because they were paid 100 percent of their fee-for-service charges.

United Healthcare required each enrollee to select a primary care physician from a list (N = 391) of family physicians (46 percent), internists (22 percent), pediatricians (19 percent), obstetrician/gynecologists (11 percent), and the few general surgeons (2 percent) who had joined the plan as gatekeepers. The gatekeeper was responsible for providing primary care and coordinating referrals for specialty care, ancillary services, and hospitalization. If the enrollee received care from a specialist or visited an emergency room for non-urgent care without approval from the gatekeeper, UHC would not pay the claim and the enrollee was responsible for payment.

Enrollees in the plan without a gatekeeper, Northwest Healthcare, were not required to choose a gatekeeper or to obtain approval for hospitalization, emergency, or specialist visits. These enrollees could receive care from any physician or non-physician health care provider in the Seattle area and the plan would reimburse the provider 100 percent of charges. Many of the same physicians who joined United Healthcare as risk-sharing gatekeepers also saw patients in Northwest Healthcare and were reimbursed in full for charges by that plan. Whereas physicians' offices submitted all claims to United Healthcare, Northwest Healthcare enrollees were required to submit claims to the plan for payment. Northwest Healthcare enrollees were provided with addressed, stamped envelopes to facilitate claims submission.

Data Collection

The adult who made most of the health care decisions for the family was asked to complete a baseline telephone interview. Information obtained from the interview included family composition, demographic information, perceived health status, health insurance coverage, and usual source of health care. This information was used to stratify the random assignment to plans and assess the similarity of the two groups. Enrollees in both plans were followed for 12 months. Data on the use of health services and charges were collected from SAFECO computerized membership and claims records. At the end of the study, a questionnaire was mailed to participants to assess experience with the plans and other health insurance during the previous year and to ascertain out-of-pocket expenditures and satisfaction with the plans. The response rate for the mail questionnaire was 95 percent for each plan.

Charges and Use of Services

We examined charges, percentage of enrollees using any services, and volume of services overall and separated according to primary care physicians versus specialists; ambulatory versus hospital care; type of procedure; and diagnostic category. We partitioned the enrollees into nonusers and users of services as described by Duan, *et al*,[11] to separate differences in access to care from the effect of gatekeeper control once someone had entered the system.

Total billed charges for 12 months were calculated for each enrollee, including all charges by primary care providers, specialists, hospitals, and ancillary services. Billed charges, rather than the amount paid by SAFECO, were used to ensure comparability between plans. Payments for coordination of benefits were not taken into account.

We measured the percentage of enrollees using any services or a specific service during the study. We also measured the volume of services used per enrollee or per user, e.g., number of ambulatory care visits, hospital admissions and days.

Definition of Primary Care Physician

A physician was designated as a primary care physician (PCP) if he or she was a general or family practitioner, osteopath, pediatrician, general internist, or obstetrician/gynecologist. All other physicians were defined as specialists. Although we tested two other PCP definitions with similar results, we used this definition to make primary care physicians as similar as possible in the two plans. This was the most conservative definition because it tended to overestimate primary care physicians in the plan without a gatekeeper and therefore any differences in primary care physician use between the two plans would be underestimated.

Characteristics of Enrollees

Data obtained during the baseline telephone interview showed enrollees in the two plans to be similar in demographic characteristics, social structural variables, other insurance coverage, perceived health status, and usual source of health care (Table 1). Enrollees in the gatekeeper plan were more likely to have had internists, obstetrician/gynecologists, or other specialists as their usual source of care. Assuming that care by internists and specialists is more costly than care by family physicians,[12] this difference between groups might reduce the difference in cost between the two plans. On the other hand, internists or obstetrician/gynecologists might be able to handle problems that a family physician would refer to a specialist.

Analysis

The differences in the means or proportions of outcome measures and the 95% confidence intervals around those differences were calculated. Analysis of covariance was used to adjust for the control variables. In general, the plan differences remained significant after controlling for main and independent variables for all enrollees and for users.[9] Although there were few differences between the plans on key independent variables, many independent variables were significantly related to charges in both plans. Including these independent variables in the analysis did not change the findings of the univariate analysis in any substantial way, confirming the success of the randomized design. Because the adjusted and unadjusted results were essentially the same, the unadjusted rates are presented in this paper.*

*Data available on request to author.

TABLE 1—Selected Characteristics of Enrollees in Gatekeeper or No Gatekeeper Plans*

Characteristics	Gatekeeper (n = 1419)	No Gatekeeper (n = 1408)
Age (years)		
0–5	9.2%	8.3%
6–17	24.3	24.6
18–44	52.6	50.7
45+	13.7	16.4
	26.9	27.7
Mean (years)		
S.D.	15.8	16.0
Percent Female	53.8%	53.7%
Relationship to Subscriber		
Subscriber	39.1%	39.6%
Spouse	22.6	21.3
Dependent	38.3	39.1
Family Size		
Mean number	2.6	2.5
S.D.	1.5	1.5
Subscriber Years of Education		
Mean years	15.6	15.7
S.D.	3.2	3.1
Spouse Years of Education		
Mean years	15.1	14.8
S.D.	2.9	2.9
Family Income		
Under $10,000	15.1%	16.2%
$10,000–20,000	43.1	41.4
$20,000–30,000	25.8	26.1
Over $30,000	16.0	16.4
Other Health Insurance Coverage	27.9%	27.5%
Perceived Health Status		
Excellent	73.6%	74.0%
Good	23.0	23.4
Fair	3.3	2.2
Poor	.1	.4
Had a Usual Source of Care before Study Period	82.9%	84.2%
Usual Source of Care Was a UHC gatekeeper	45.4%	49.5%
Specialty of Usual Source of Care before Study Period		
Family/General Practitioner	49.6%	57.1%
Pediatrician	22.7	21.7
Internist	13.9	9.5
Obstetrician/Gynecologist	8.0	6.2
Other	5.8	5.5
Months Membership in Plan During Study		
Mean months	10.5	10.3
S.D.	2.2	2.3

*Other characteristics which were measured but are not presented here due to small differences between plans include: marital status, family composition, family health risk scale, employment status, months residence in community, and type of health insurance coverage.

Results

Use and Charges for All Enrollees

Access to care, as reflected by the percentage of enrollees using any services or having at least one ambulatory visit, was high in both plans (Table 2). People in the gatekeeper plan had more visits to a primary care physician (difference = 6%, 95% CI = 2.43, 9.57) and fewer visits to a specialist (difference = −9%, 95% CI = −12.28, −5.72). Ambulatory charges per enrollee were $21 lower in the gatekeeper plan (95% CI = −39.9, −2.1, Table 3), due to significantly fewer visits to specialists.

There were minor differences between plans in hospital use and charges per enrollee (Tables 2 and 3). The gatekeeper plan had lower hospital use than the no gatekeeper plan (Table 4). The plans had similar inpatient charges for specific services, such as medical, surgical, laboratory, and radiological procedures (data not shown).*

Charges and Visits for Ambulatory Care

We examined the effect of the gatekeeper plan among those people who used ambulatory services. PCP ambulatory visits and charges for users were similar, but users in the gatekeeper plan had significantly fewer visits to specialists and lower specialist charges (Table 5).

There were no differences between plans in primary care physicians' use of surgical, laboratory, or radiological procedures, as defined by categories of Current Procedural Terminology codes (data not shown). PCPs in the gatekeeper plan ordered or performed fewer medical procedures on their patients than did PCPs in the no gatekeeper plan (difference = −4%, 95% CI = −5.13, −2.87). Specialists in both plans had similar use and charges for various types of procedures.

We also compared ambulatory use between plans for 20 common diagnostic categories and found that people in the gatekeeper plan had fewer visits for mental health problems, back problems, and acute and chronic respiratory problems, and more of their visits for these problems were with a PCP rather than a specialist (data not presented).*

Experience Using the Plans

The end-of-study survey showed no difference between plans in enrollees' use of other health insurance plans, in the proportion of claims submitted, or in out-of-pocket expenditures during the study period. Enrollees in the gatekeeper plan more often identified internists, obstetrician/gynecologists, and specialists as their usual providers of care, while enrollees in the no gatekeeper plan had more often named family physicians (the distribution is similar to source of care before the study, Table 1). Enrollees were more satisfied with the no gatekeeper plan than the gatekeeper plan (very satisfied: 75% − 64% = 11%, 95% CI = 7.63, 14.37).

TABLE 2—Percentage of Enrollees Using Services in the Gatekeeper and No Gatekeeper Plans

Percentage of Enrollees With:	Gatekeeper (n = 1419)	No Gatekeeper (n = 1408)	Difference (95% CI)
	%	%	
Any Services	74	77	−3 (−6.17, +0.17)
≥1 Ambulatory Visit	68	70	−2 (−5.41, +1.41)
≥1 Primary Care Physician Visit	65	59	+6 (+2.43, +9.57)
≥1 Specialist Visit	23	32	−9 (−12.28, −5.72)
≥1 Hospitalization	4.5	5.0	−0.5 (−2.07, +1.07)

TABLE 3—Annual Charges and Visits for Enrollees in the Gatekeeper and No Gatekeeper Plans

	Gatekeeper (n = 1419) Mean	No Gatekeeper (n = 1408) Mean	Difference (95% CI)
Total Charges	$239	$254	−15 (−73.0, +43.0)
Primary Care Physician	85	96	−11 (−24.4, +2.4)
Specialist	155	158	−3 (−52.8, +46.8)
Hospital Charges	$ 93	$ 87	+6 (−44.9, +56.9)
Primary Care Physician	9	16	−7 (−15.4, +1.4)
Specialist	84	71	+13 (−32.2, +58.2)
Ambulatory Charges	$146	$167	−21 (−39.9, −2.1)
Primary Care Physician	76	80	−4 (−13.2, +5.2)
Specialist	70	87	−17 (−30.1, −3.9)
Ambulatory Visits	2.7	3.0	−0.3 (−0.60, +0.02)
Primary Care Physician	2.1	2.0	+0.1 (−0.12, +0.32)
Specialist	.63	.93	−0.3 (−0.50, −0.10)

TABLE 4—Hospital Use for Enrollees in the Gatekeeper and No Gatekeeper Plans*

	Gatekeeper Mean	No Gatekeeper Mean	Difference (95% CI)
Hospital Admissions per 1000 Person Years	49	56	−7 (−25.1, +11.1)
Hospital Days per 1000 Person Years	205	277	−72 (−207.8, +63.8)
Average Length of Hospital Stay	4.2	4.9	−0.7 (−2.7, +1.3)
Hospital Charges			
Primary Care Physician	$ 196	$ 320	−124 (−280.4, +32.4)
Specialist	$1867	$1432	+435 (−378.9, +1248.9)

*The gatekeeper plan had 64 persons with a total of 70 hospitalizations, and the no gatekeeper plan had 70 persons with a total of 79 hospitalizations.

TABLE 5—Charges and Visits for Ambulatory Users in the Gatekeeper and No Gatekeeper Plans

Per User Per Year	Gatekeeper (n = 1052) Mean	No Gatekeeper (n = 1087) Mean	Difference (95% CI)
Ambulatory Charges	$197	$216	−19 (−42.4, +4.4)
Primary Care Physician	102	104	−2 (−13.3, +9.3)
Specialist	95	113	−18 (−34.8, −1.2)
Ambulatory Visits	4.0	4.3	−0.3 (−0.72, +0.12)
Primary Care Physician	3.1	2.9	+0.2 (−0.08, +0.48)
Specialist	.9	1.3	−0.4 (−0.67, −0.09)

Although satisfaction differed, similar percentages of families switched to other plans during the next open enrollment.

Discussion

In this randomized trial, the gatekeeper plan had lower ambulatory charges per enrollee, primarily due to lower use of specialists. The gatekeeper plan had little impact on hospital use or charges perhaps because of small numbers. The total charges per enrollee were 6 percent lower in the plan with a gatekeeper, a modest difference that was not statistically significant.

The gatekeeper plan required patients to see a primary care physician first, so it was not surprising to find that more enrollees in the gatekeeper plan had a visit to a primary care physician. The gatekeeper plan provided no incentives for PCPs to reduce visits to themselves, and indeed PCP visits and charges were similar in the two plans. There were incentives for gatekeepers to limit referrals and this apparently resulted in lower expenditures for specialist services in the gatekeeper plan. Specialists were offered no incentives by the gatekeeper plan to reduce costs, and once patients were referred to specialists, their volume and charges for various types of procedures were similar in both plans.

A gatekeeper effect was seen for certain diagnoses for which treatment is at the discretion of the patient and physician, such as mental health problems and back problems. We do not know if these differences result from: self-selection, decreased initial visits by the enrollee, differences in diagnostic coding, or the practice patterns or specialty of the regular doctor. Future studies of gatekeeper plans should explore utilization by diagnostic category in more detail.

True gatekeeper effects are probably at least as large as those reported here. First, the population consisted of subscribers and their family members who chose United Healthcare rather than one of the other state health insurance options available to them. Because the pre-enrollment information described the standard UHC plan, all study participants may

have been oriented to use PCPs more than the general population; therefore, the differences between the plans may be underestimated. Second, differences between plans may have been underestimated because people in the no gatekeeper plan were more likely to have a regular doctor who was already participating in United Healthcare as a gatekeeper.

Two other issues should be considered when interpreting these results. First, we could not examine the gatekeeper plan effect in more detail because we were using claims data. We have no indication whether a specific visit was initiated by the patient or provider, or whether the patient was seeing a particular provider as a primary care physician or as a specialist. Furthermore, claims data did not allow us to describe how or to what extent a primary care physician was functioning as a gatekeeper. Second, this 12-month study period may not be representative of the plans' or enrollees' experience over time. One possibility is that individuals in the no gatekeeper plan may have used services differently because they knew that the study period was limited to one year. Another possibility is that the "learning period" for new enrollees may have affected the results. We would expect this learning period to be more important for the gatekeeper plan because enrollees had to adjust to more restrictions on their use of services than in the no gatekeeper plan.

Other studies have reported mixed results of the gatekeeper effect on the use of services. Catlin, *et al*, surveyed use of primary care physicians in staff and group model HMOs and excluded independent practice associations (IPAs).[6] They found no significant differences in hospital or ambulatory use among HMOs using more primary care physicians, but the financial incentives offered to primary care physicians were not described. Burkett's descriptive study of an IPA-HMO with gatekeepers and financial incentives similar to UHC found the rate of referral visits to be low.[8] In comparing his results to ours, primary care visits per person per year appeared to be slightly higher and use of referral visits even lower than those in this study.

The findings of another study of UHC conducted at about the same time provide additional evidence for assessing the gatekeeper concept. In that study, we compared the standard UHC gatekeeper plan with two other health plans: Group Health Cooperative, a staff model HMO with primary care case management; and Blue Cross, which provided fee-for-service hospital and ambulatory care. Use and estimated expenditures were compared across the three plans for a different cohort of Washington State employees which was followed for two years.[9] Initial access to care was high at both UHC and Group Health. UHC had lower expenditures for specialists than the other plans, but the savings were not sufficient to decrease overall expenditures. Total annual expenditures per person were similar for UHC and Blue Cross and were significantly higher than expenditures for Group Health.

These two studies of United Healthcare, using different study designs, both reported savings on specialist costs in the UHC gatekeeper plan compared to other plans. However, incentives for gatekeepers alone may not be sufficient to contain overall plan costs. In a subsequent case study of United Healthcare, we examined charges for all enrollees from numerous employers in three states. Although United Healthcare may have contained specialist charges, from 1979 to 1982 specialist charges as a percentage of total charges rose, whereas the percentage of charges by gatekeepers fell.[13]

After operating for nine years in Washington, California, and Utah, United Healthcare was discontinued by SAFECO because of large financial losses, due to management problems and inadequate incentives for specialists and consumers to control costs.[14] The evidence presented in this study suggests that the gatekeeper concept actually may have reduced UHC losses.

Hillman describes the conflict of interest that may exist between financial incentives and providing high quality care.[2] Whenever utilization decreases, especially for referral care, the question of underutilization of services arises. There is no evidence from our studies or those of others which shows plans with gatekeepers providing higher or lower quality care than other plans. Our study was too small to observe any differences in the overall quality of care.

The results of this study are particularly relevant to independent practice associations with incentives similar to United Healthcare. (Since our study population represents employees and their families, these results may not apply to the poor or to retirees.) As the number of gatekeeper plans in the United States continues to grow, the findings of this study have important implications for the design of those plans. First, the United Healthcare incentives for channeling enrollees to primary care physicians increased enrollees' initial use of PCPs and decreased the use of specialists. Second, relatively small financial incentives were sufficient to decrease primary care physician referrals to specialists. It should be emphasized that these incentives applied only to a small proportion of a primarily fee-for-service based practice. Third, the finding of no difference between plans in specialists' use of procedures suggests that in the future plans should incorporate incentives for cost control by specialists.

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Increased Suppressor T Cells in Probable Transmitters of Human Immunodeficiency Virus Infection

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Abstract: To evaluate behavioral and immunologic factors related to transmission of human immunodeficiency virus (HIV) by homosexual intercourse, we studied a population of 329 homosexual/bisexual men (155 partner-pairs) seen in a community health center and medical outpatient clinic. Logistic regression analysis showed that behavioral risk factors for infection in the 130 HIV-infected men included: receptive anal intercourse (OR 4.6, 95% CI-1.8, 12.1); receptive fisting (OR 2.5, CI-1.1, 7.0); nitrite use (OR 2.3, CI-1.2, 4.6); history of gonorrhea or syphilis (OR 2.3, CI-1.4, 3.9); and history of sexual contact with men from areas with many AIDS cases (OR 1.9, CI-1.0, 3.5). Comparing seven men who were probable transmitters of HIV and 11 men who had not transmitted HIV to their uninfected partners despite unprotected insertive anal intercourse, we found no differences in HIV isolation from peripheral blood mononuclear cells, circulating HIV antigen detection, or presence of neutralizing antibody to HIV. Helper T-cell numbers were not significantly different between the two groups, but transmitters had more suppressor T-cells than did nontransmitters. (*Am J Public Health* 1989; 79:1638–1642.)

Introduction

The major behavioral risk factors in the transmission of human immunodeficiency virus (HIV) in homosexual/bisexual men include receptive anogenital sexual contact and a large number of sex partners.[1–3] However, some men with many receptive anal sexual contacts remain uninfected while others with infrequent contact become infected.[4,5] Thus, while particular sexual behaviors may increase the likelihood of HIV transmission, other factors presumably contribute to the efficiency of sexual transmission of the virus.

Transmission of HIV requires that the infected partner transmit the virus and that the exposed partner be susceptible to the infection. Studies of susceptibility to HIV infection of exposed persons have failed to reveal either immunologic or genetic susceptibility factors.[6,7] However, several studies have shown a correlation between worsening clinical status or declining numbers of helper T cells in the HIV-infected partner and increased transmission of HIV.[8–12]

In this study, we evaluated the role of specific sex practices in HIV transmission among homosexual men. We also examined the relation of immunologic laboratory parameters to transmission by comparing seropositive men who appeared to have infected their partners with seropositive men who did not infect their partners despite unprotected insertive anal intercourse.

Methods

Selection of Study Participants

Men were eligible for enrollment in our study if they were homosexual or bisexual, were 18 years of age or older, and resided within the Boston, Massachusetts Standard Metropolitan Statistical Area. Men were seen at the Fenway Community Health Center (FCHC) and/or the New England Deaconess Hospital. AIDS was diagnosed according to the Centers for Disease Control (CDC) classification system (Group IV subgroups C-1 and D-2); AIDS-related complex (ARC) was defined as CDC classification group III (persistent generalized lymphadenopathy), IV-A (fever, weight loss or diarrhea), or C-2.[13] Asymptomatic men were those without AIDS or ARC. Sexual contact was defined as orogenital, anogenital, or oroanal contact involving an exchange of body fluids and/or penetration.

We recruited asymptomatic participants from: men who had at least one visit to the FCHC from May 1984 through March 1987; men referred by friends or physicians outside of the FCHC; advertisements soliciting participation in the study placed in two newspapers; and men referred by persons already enrolled in the study. The protocol for this study was approved by the Human Studies Committees of all participating institutions.

Data Collection

At enrollment, all participants were interviewed to obtain data on demographic characteristics, occupational and travel histories, sexual behaviors, and drug and alcohol use. A complete medical history was obtained for the five years before study enrollment, and a standard clinical examination was performed. All participants were counseled regarding high- and low-risk sex practices and ways to reduce their risk for HIV infection.

Laboratory Studies

All participants were evaluated with a complete blood count (CBC) and lymphocyte subset determination, performed using specific monoclonal antibodies (Ortho Diagnostics, Raritan, NJ). Antibodies to HIV were determined by an enzyme immunoassay (EIA) (Abbott Laboratories, Chicago, IL). All serum samples initially positive by EIA and selected other serum samples (as specified later) were examined by Western blot using previously described methods.[14] Culture of HIV was performed in H9 cells, and reverse transcriptase activity was measured by standard techniques.[15] Antigen-capture assay for HIV employed a sandwich EIA using polystyrene beads coated with human anti-HIV (Abbott Laboratories, Chicago, IL).[16] Neutralizing antibody assays were performed in H9 cells with the HTLV-IIIB isolate of HIV using a modification of the method of

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Robert-Guroff.[17] The assay was performed by mixing 100 50 percent tissue culture infectious doses of HIV with 200 ml of heat-inactivated human serum at various dilutions, and incubating at 4°C for 1.5 hours. Virus was then absorbed onto 1×10^6 H9 cells for one hour and incubated at 37°C. Neutralization titer was defined as positive when the assay blocked 90 percent or more of the control virus infection at a 1:20 dilution.

Statistical Analysis

Confidence intervals for infection rates were calculated based on the chi-square function method of Miettinen.[18] Exposure variables were categorized depending on the distribution of the variable without regard to serologic status. Unadjusted prevalence odds (exposure odds) ratios were estimated and exact 95% confidence intervals (CI) calculated by the method of Rothman.[19] Adjusted estimates were maximum likelihood estimates from logistic regression and 95% CI were calculated using the standard errors of the beta estimates.[20] Continuous laboratory variables were compared using the Wilcoxon 2-sample test.[20] Dichotomous laboratory variables were compared by Fisher's exact test.[20]

Results

All interview information and laboratory samples were obtained at the initial enrollment visit. Three hundred twenty-nine men were enrolled in the study: 23 men had AIDS, 31 had ARC, and 275 were asymptomatic. Six participants were linked to three partners, 12 to two partners, 113 to one partner, and 43 to no partner, for a total of 155 partner-pairs.

The mean age of the study participants was 32.8 years; mean level of education 16 years; median income $10,000–$19,999; and mean length of time residing in metropolitan Boston 144 months. Ten percent of the men had been married; 316 (96%) were White, 9 (3%) were Black, and four (1%) were Hispanic.

One hundred thirty (40%) men were HIV-seropositive, while 199 (60%) were seronegative. Of the 155 partner-pairs, 62 (40%) were concordant seronegatives, 35 (23%) were concordant seropositives, and 58 (37%) were discordant (one seropositive and one seronegative partner). Thirteen of 24 partners (54%) of men with AIDS were seropositive (95% CI 34, 74); 12 of 31 partners (39%) of men with ARC were seropositive (CI 22, 57); 10 of 24 partners (42%) of asymptomatic seropositive men were seropositive (CI 23, 61).

Sexual Practices

To determine which potential risk factors were associated with seropositivity, we analyzed all 329 men in the cohort. In bivariate analysis, six variables were associated with antibody to HIV: number of sex partners in the previous five years; receptive anal sexual activity; receptive oral sexual activity; receptive fisting; sexual contact with persons from areas of high HIV prevalence; history of gonorrhea or syphilis; use of nitrites; and use of other illicit drugs (Table 1).

TABLE 1—Distribution (%), Crude and Adjusted Relative Odds for HIV Infection by Behavioral Risk Factors

History over Past Five Years	HIV-Antibody Status: Percent Seropositive (N = 130)	HIV-Antibody Status: Percent Seronegative (N = 199)	Unadjusted OR	Adjusted[a] OR	Adjusted[a] 95% CI
No. of male partners					
≤30	36.1	60.4	1.00	—	—
>30	63.9	39.6	2.70	1.36	(.58, 3.22)
No. of men with whom the subject had a specific sex practice					
Receptive penile oral					
1–10	24.2	44.8	1.00	—	—
>10	75.8	55.2	2.53	.83	(.56, 1.23)
Receptive penile anal					
0	6.1	18.8	1.00	—	—
1–10	34.1	57.4	1.84	1.50	(.62, 3.67)
>10	59.9	23.8	7.82	4.63	(1.78, 12.06)
Receptive fisting					
never	87.1	96.5	1.00	—	—
ever	12.9	3.5	4.12	2.54	(1.08, 7.03)
Sex with partners from areas of higher HIV prevalence[b]					
never	20.3	44.6	1.00	—	—
ever	79.7	55.5	3.16	1.88	(1.,01, 3.50)
History of gonorrhea or syphilis					
never	42.1	72.3	1.00	—	—
ever	57.9	27.8	3.59	2.30	(1.36, 3.88)
Nitrite use					
never	12.0	34.7	1.00	—	—
ever	88.0	65.3	3.88	2.31	(1.17, 4.60)
Illicit drug use[c]					
never	8.3	18.3	1.00	—	—
ever	91.7	81.7	2.49	1.37	(.59, 3.22)

a) Adjusted using a logistic regression model that included age and the variables listed in this table.
b) New York City, San Francisco, Los Angeles, and/or Miami.
c) Marijuana, cocaine, PCP, LSD, and/or quaaludes.

A logistic regression model was developed to assess potential confounding by any of the variables significantly associated with HIV antibody in the bivariate analysis. Factors that correlated with seropositivity in the multiple regression model were: receptive anal sexual activity; receptive fisting; nitrite use; history of gonorrhea or syphilis; and sexual contact with persons from areas of high HIV prevalence (Table 1).

Characteristics of Transmitters

We examined partner-pairs in which both partners were seropositive to identify possible transmitters. Ten partner-pairs were identified in which one partner had had over 80 percent of his receptive anal contacts within the relationship (in no case did both partners have over 80 percent of their receptive anal contacts with each other). The partner whose contacts were in large part within the relationship was therefore presumed to have been infected by the other (more sexually active) partner, who was designated the likely transmitter. Peripheral blood cells were available from seven of the transmitters; of these, four had a partner who had no receptive anal intercourse with any other partner, while the other three had partners with 4, 13, and 14 episodes outside the relationship.

Partner-pairs in which one partner was seropositive and one was seronegative were examined to identify men who had exposed their partner to HIV without transmitting infection. All seronegative men were confirmed to be uninfected by negative Western blot and HIV antigen assay. Since we could not be certain that the seropositive partner was positive more than a short time before entry into the study, we excluded partner-pairs who had not had at least six episodes in which the seronegative partner had receptive anal intercourse with an insertive seropositive partner during the six-months before entry into the study. The HIV seropositive partners in these partner-pairs were designated the likely nontransmitters. Sixteen nontransmitters were identified. Peripheral blood cells were available from 11 of these men.

One of the transmitters had AIDS and three had ARC; no nontransmitters had AIDS but three had ARC. Transmitters had longer relationships with their partner (mean ± SD: 58 ± 24 months) than did nontransmitters (36 ± 36 months), but transmitters had fewer total episodes of insertive anal intercourse with their partner (151 ± 185 episodes) than nontransmitters (192 ± 192 episodes). Neither difference was statistically significant. No differences were noted between transmitters and nontransmitters in history of syphilis, gonorrhea, hepatitis B, herpes simplex, herpes zoster, or cytomegalovirus infection, or infectious mononucleosis. Furthermore, transmitters and nontransmitters had similar histories of fever, skin rash, and enlarged lymph nodes.

Transmitters did not differ from nontransmitters with respect to circulating HIV antigen, presence of neutralizing antibody to HIV, or ability to recover virus from peripheral blood mononuclear cells (Table 2). Phenotyping of peripheral blood mononuclear cells of transmitters revealed a median of 1150 total T cells, 430 helper T cells, 960 suppressor T cells, and a median helper/suppressor ratio of 0.40; nontransmitters had a median of 1170 total T cells, 390 helper T cells, 650 suppressor T cells, and a median helper/suppressor ratio of 0.80 (Figure 1). While total and helper T-cell numbers were similar in both groups, transmitters had higher suppressor T-cell numbers than did nontransmitters (Wilcoxon, $p = < .02$). Helper/suppressor ratios were lower in transmitters than in nontransmitters (Wilcoxon, $p = .12$). Five of the six transmitters with elevated suppressor cells were re-evaluated after 12 months; only one had persistently elevated suppressor cells.

TABLE 2—Distribution (%) of Laboratory Parameters in Transmitters and Nontransmitters

Parameter	Transmitters (%)	Nontransmitters (%)
HIV culture	1/5 (20)	3/9 (33)
Circulating HIV antigen	1/7 (20)	4/13 (31)
Neutralizing antibody to HIV	3/3 (100)	3/7 (43)

Discussion

Several studies have shown that the major risk factors for acquisition of infection among homosexual men are exposure to a large number of sex partners and exposure to infected persons by receptive anal intercourse.[1–3] Demographic and behavioral analysis of our cohort affirms that it is similar to previously reported cohorts. We assessed risk by estimating exposure odds ratios for HIV infection. The interpretation of such ratios can be difficult when both the temporal sequence and the duration of exposure are unknown. In some cases, the relevant period of risk may have been overestimated, resulting in exposure odds ratios that underestimate the true values.

The risk of acquiring HIV infection was positively correlated with increased number of sex partners with whom the person was the receptive anal sex partner. In our analysis, both fisting and use of nitrites were also associated with seropositivity after controlling for confounding. Fisting might facilitate HIV transmission by causing rectal trauma, thereby allowing virus to bypass mucosal defenses. The biologic mechanism for the increased risk in nitrite users is unclear; the role of nitrites as potential effect modifiers needs to be assessed further.

While these practices are associated with an increased risk of becoming infected with HIV, they do not assure it. Many persons appear to be sexually exposed to HIV on multiple occasions, yet do not become infected.[4,5] Possible explanations for this phenomenon are that some persons are more efficient transmitters than others; that some persons, when exposed, are more susceptible than others; or both.

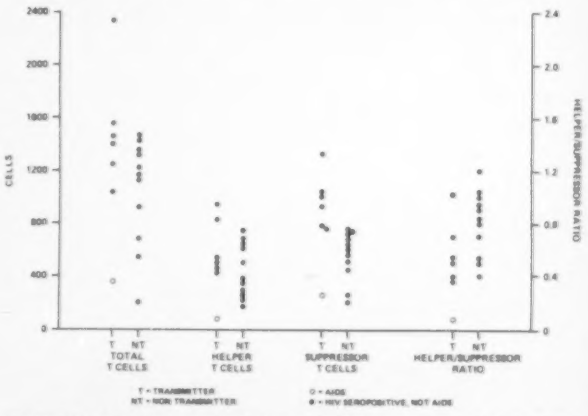

FIGURE 1—T-cell Phenotypic Markers in HIV Transmitters and Nontransmitters; Transmitters had Higher Numbers of Suppressor T-cells

Since previous studies have failed to identify genetic or immunologic susceptibility factors for HIV infection,[6,7] we focused our investigation on factors relating to transmission.

Partner-pairs in which both partners were seropositive were analyzed to select a group of men who were most likely to have transmitted infection to their partners. Such a determination would ideally require that a seropositive man have only been exposed to a single seropositive partner during the time the virus could be assumed to have been circulating in the community. However, monogamous relationships were rare in our cohort. We therefore computed a scale of relative fidelity for each partner. Seven partner-pairs included a man who had had more than 80 percent of his receptive anal contacts since the end of 1981 with one seropositive person (four had 100 percent of their contacts with the presumed transmitter). Therefore, although the date of HIV transmission could not be definitively established and contact with other seropositive partners could not always be completely excluded, we feel the anal insertive partners in these relationships were likely HIV transmitters.

Because of the retrospective nature of the study, the dates of infection of the possible transmitters and nontransmitters were unknown and the exact duration of exposure could not be established. However, the duration of relationships was not significantly different between transmitters and nontransmitters. Moreover, similar numbers of potentially infective episodes were reported for both transmitters and nontransmitters.

We found increased suppressor cells in HIV transmitters. This suggests a role for host immunity in modulating infectiousness by controlling the amount of virus present in host secretions and thereby regulating the dose of virus to which a sex partner is exposed. We were not able to measure the amount of virus in the semen of our transmitters and nontransmitters. However, no relationship was seen between the ability to culture virus from the blood, the presence of neutralizing antibody, or the presence of p24 antigen and the ability to transmit HIV. This may have been due to the small number of samples available for study; alternatively, the presence of detectable HIV in blood may not be related to the presence of infectious virus in semen.

Increased suppressor T-cell numbers have been reported in primary HIV infection.[21] The transmitters in our study therefore may have recently experienced acute HIV infection. Alternatively, since many infectious processes (notably herpes virus infections) can temporarily increase suppressor cells,[22,23] increased suppressor cells in our transmitters may be a marker of an intercurrent infectious process other than HIV infection. Such a process might increase HIV replication by activating HIV amplification genes[24,25] or by interfering with the as yet unknown mechanisms that permit the host to at least temporarily control HIV replication.

Our results suggest that some men with higher numbers of suppressor cells may be more infectious to their sex partners than other infected men. Francis, *et al,* have also reported increased suppressor cells in mothers who appeared to have transmitted HIV to their neonates.[26] On the other hand, several studies have found that HIV-infected men with low helper T-cell numbers or advanced HIV infection are more likely to transmit HIV to their female partners.[8–12] Our HIV-infected subjects were largely asymptomatic with relatively normal numbers of helper T cells; thus we would not have expected to detect transmission by many men with AIDS or low numbers of T-helper cells. Our observations thus support the hypothesis that HIV infectivity is not limited to the period of immunodeficiency seen with advanced HIV infection.[27]

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1987 National Medical Expenditure Survey Data Available on Magnetic Tape

The 1987 National Medical Expenditure Survey (NMES), a nationwide survey sponsored by the National Center for Health Services Research, is available from the National Technical Information Service (NTIS) on magnetic tape. The Institutional Population Component was designed to yield national estimates of the use of, and expenditures for, care during 1987 by persons who reside in nursing homes or facilities for the mentally retarded.

The files contain data from two Phase I instruments: the Facility Questionnaire and the Baseline Questionnaire.

- The Facility Questionnaire was collected from approximately 1400 participating facilities, and the data records contain bed size, type of ownership, facility certification, services routinely provided, average cost, and other information.
- The Baseline Questionnaire file contains data collected from approximately 6,900 persons who were residing in the sampled facilities, and include sociodemographic information such as age, race, sex, information on residences prior to admission, measures of functional limitations and chronic condition, and the work history of persons in facilities for the mentally retarded.

Documentation is provided with the tape and includes a description of NMES, questionnaires, and technical information on sample design, weights, and variance estimation. The NMES is available in EBCDIC or SAS format, and is contained on 9 track, half-inch tape only. Specify density requirement (1600 or 6250 bpi) when ordering.

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4T

A Survival Analysis of Hospitalization among Patients with Acquired Immunodeficiency Syndrome

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Abstract: Survival analysis techniques were used in estimating lifetime inpatient utilization among patients diagnosed with acquired immunodeficiency syndrome (AIDS) using data on 863 members of the Kaiser Permanente Medical Care Program in the Northern California Region diagnosed with AIDS between January 1, 1981 and June 30, 1987. Using information on both deceased and living patients, we estimated means of 40.3 lifetime inpatient days and 3.3 hospitalizations among all AIDS patients. Those presenting with Kaposi's sarcoma experienced a mean of 7.6 fewer lifetime inpatient days than those presenting with *Pneumocystis carinii* pneumonia (95% confidence interval = .61, 14.6) and a mean of 11.0 (3.9, 18.6) fewer inpatient days than all other AIDS patients. Older patients had fewer hospital admissions than younger ones. Year of diagnosis does not appear to be related to lifetime utilization, and there is an indication that increased survival has been accompanied by decreased inpatient utilization intensity as measured on a per person-year basis. We recommend the use of survival analysis methods in the study of utilization among groups of patients with incomplete follow-up. (*Am J Public Health* 1989; 79:1643–1647.)

Introduction

The cost of providing health care for patients with acquired immunodeficiency syndrome (AIDS) continues to be of national concern. Recent research on the economic impact of the epidemic has provided different estimates of the mean lifetime cost per AIDS patient, ranging from $23,000 to $168,000.[1–6] Much of the variability can be attributed to differences in estimates of the mean lifetime inpatient days accrued by AIDS patients.[1] The disparity in these estimates of inpatient utilization is due in part to true differences across the populations from which the samples of patients were drawn but also in part to the different sampling and estimation techniques. Some investigators have restricted the sample to patients who have died during a given time period, which generally results in a sample biased toward shorter lived individuals. Using survival analysis techniques, we provide estimates of the distribution of lifetime hospitalizations and inpatient days in a large well-defined sample of both deceased and living patients with AIDS.

Methods

Data Collection

We identified 863 cases of AIDS diagnosed between January 1, 1981 and June 30, 1987 who were members of the Kaiser Permanente Medical Care Program in the Northern California Region (KPNCR), a private nonprofit prepaid group practice program with over two million members receiving care in 11 counties; these include the San Francisco Bay Area, a region which has experienced a relatively high incidence of AIDS. According to the California State Health Department, as of June 30, 1987, 4,251 cases were reported in the geographic area served by the Health Plan; we estimate that over 20 percent of these cases were members of the Kaiser Permanente Medical Care Program.

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AIDS cases were identified retrospectively by obtaining reports from infection control nurses, and by examining three computerized datasets:

- the Inpatient Utilization System file which contains a record for each hospitalization at any Kaiser Foundation hospital including up to 13 diagnostic codes (*International Classification of Diseases, 9th Revision, Clinical Modification* (ICD-9)) per admission;
- pathology files containing biopsy reports from five major KPNCR hospital pathology departments; and
- a file maintained by the Health Plan's regional pharmacy administration containing all patients who had received zidovudine (AZT).

Each hospital admission from January 1, 1981 through June 30, 1987 was scanned for ICD-9 codes used specifically for AIDS: 279.10 and 279.19, which were used prior to October 1986, and 042.0, 042.1, 042.2, and 042.9 which were used subsequently. In addition, possible cases were identified by searching the hospitalization file diagnosis codes for AIDS-related conditions included in the Centers for Disease Control (CDC) case definition of AIDS.[7] Possible cases were also identified by searching the pathology files for diagnoses of Kaposi's sarcoma, *Pneumocystis carinii* pneumonia, candidiasis, and certain non-Hodgkin's lymphomas indicative of AIDS. For cases identified through the pathology file only or the AZT file only, the medical records were reviewed to verify the presence of AIDS. The medical records of those on the hospitalization file with a possible AIDS diagnosis were also reviewed. Patients were removed from the sample if the CDC criteria were not met.

Analyses are based on the 863 AIDS cases confirmed by chart review (N = 166), infection control nurse reports (N = 171), hospitalization records with diagnosis codes for AIDS (N = 506), and those on the AZT file with an additional record of an AIDS-related infection or neoplasm on the hospitalization or pathology files (N = 5). Males less than 60 years old with a biopsy report of Kaposi's sarcoma and no other source of confirmation were also included (N = 15).

For each case, date of diagnosis and diseases present on that date were obtained from the source of diagnosis confirmation. For those not confirmed either by chart review or nurse report, the earliest biopsy report or hospitalization record with an AIDS-related ICD-9 code was used. Sex and date of birth were obtained from computerized Health Plan

membership files. It is unlikely that there are patients in this case file who do not have AIDS, but it is possible that some Health Plan members diagnosed with AIDS are not included. Given the variety of sources used to identify AIDS patients, however, the number of unidentified cases is probably small.

The hospitalization file was used to obtain for each of the 863 cases the number of hospitalizations and total inpatient days from date of diagnosis to either date of death, date of leaving the Health Plan, or June 30, 1987, the end of this study. Date of death was obtained by a linkage to death records using the California Automated Mortality Linkage System.[8] Health Plan membership files were used to identify either date of leaving the plan or continued membership in the plan until death or June 30, 1987.

Data Analysis

While survival analysis techniques are often used to study time to an event such as death or onset of disease, these statistical methods are also appropriate for analyzing other random variables.[9,10] One of the main features of these methods is that the random variable of interest can be censored, which occurs when follow-up is incomplete. Information is provided by a censored observation because the final outcome will be at least as great as the measurement taken when follow-up is halted. In this study, a patient's lifetime number of hospitalizations and inpatient days were censored by leaving the Health Plan (N = 13) or by surviving through June 1987 and remaining in the plan (N = 348). We considered a patient's complete lifetime utilization to be measured only if death in the health plan occurred prior to June 30, 1987. The application of these statistical methods to the analysis of inpatient utilization is analogous to the analysis of survival time; we are treating inpatient utilization just as one would treat survival time or any other random variable subject to censoring.

The distributions of survival time, lifetime hospitalizations, and inpatient days were estimated by using the product-limit (Kaplan-Meier) method[11]; associated 95% confidence intervals were formed using Greenwood's estimation method.[12] This method provided estimates of the mean, median, and other percentiles of the distributions of inpatient utilization and survival time. Because lifetime hospitalizations as opposed to lifetime hospital days takes one of a very limited number of values, the sample median and other percentiles are less informative than the proportion of patients attaining selected values, and we present product-limit estimates of the latter. The log rank test[13] was used to assess differences in inpatient utilization distributions across subgroups defined by age, diagnosis at presentation and diagnosis date.

As an alternative to measuring hospital utilization from diagnosis to death of an AIDS patient, utilization rates on a per person-year basis and average length of stay estimates are also presented. Hospitalization and inpatient day rates are calculated for each time period as the observed number of events among AIDS patients divided by the total follow-up time for the cohort in that period. When study subjects enter and exit at any point during the year, this rate is a more appropriate measure of annual utilization intensity than statistics based on annualized data (e.g., multiplying by 12 the utilization by a patient observed for one month). Average length of stay is calculated as the mean number of inpatient days among all admissions during the given time period.

SAS was used for all data analyses[14]; the LIFETEST procedure was used to obtain product-limit estimates of distribution functions, means and percentiles, and to perform log rank tests.

Results

Patient characteristics are listed in Table 1. The mean age at diagnosis was 39.3 years; 98.7 percent were male. Fifty percent of the cases presented with *P. carinii* pneumonia only, and 16.1 percent presented with Kaposi's sarcoma only. Other opportunistic infections accounted for 17.7 percent of the cases. The sex and diagnosis at distributions are similar to those for cases in the same geographic area reported to the California State Health Department except there appears to be a somewhat smaller proportion of KPNCR patients presenting with Kaposi's sarcoma.[6] This may be an artifact due to our reliance on inpatient discharge data for diagnosis information for the majority of cases. In addition, our cases were slightly older than the AIDS patients reported to the State, whose mean age was 38.1 years.[6]

Among all 863 cases of AIDS, the median survival time was 309 days; the 25th and 75th percentiles were 103 and 552 days, respectively. The estimated probabilities of surviving one and two years (and associated standard errors) were .44 (.020) and .20 (.020). The product-limit estimates of the survival distributions for those with an initial diagnosis of Kaposi's sarcoma only, *P. carinii* only, and all others are presented in Figure 1. Diagnosis at presentation was strongly related to survival time (P = .0001). The median time to death for patients with Kaposi's sarcoma was 531 days as compared to 331 days for those with *P. carinii*. The group of AIDS patients with other diagnoses generally had the shortest survival times with a median of 187 days.

The product-limit estimate of the distributions of lifetime hospitalizations and inpatient days are presented in Figures 2 and 3. Only .5 percent of these patients had no hospitalizations. Although the majority of patients (66 percent) were hospitalized for a total of 45 days or less, there was a subset

TABLE 1—Characteristics of 863 AIDS Patients Identified at KPNCR from January 1, 1981 through June 30, 1987

	N	%
Age at Diagnosis (years)		
0–19	4	.5
20–29	110	12.7
30–39	361	41.8
40–49	262	30.4
50–59	97	11.2
60+	29	3.4
Sex		
Male	852	98.7
Female	11	1.3
Date of Diagnosis		
1/81–12/81	3	.3
1/82–12/82	16	1.9
1/83–12/83	39	4.5
1/84–12/84	119	13.8
1/85–12/85	190	22.0
1/86–12/86	282	32.5
1/87–6/87	214	24.9
Diagnosis at Presentation		
Kaposi's sarcoma (KS)	139	16.1
Pneumocystis carinii pneumonia (PCP)	432	50.1
KS and PCP	36	4.2
Other neoplasms	11	1.3
Other infections	153	17.7
Unspecified	92	10.7
Deaths through June 1987	504	58.4

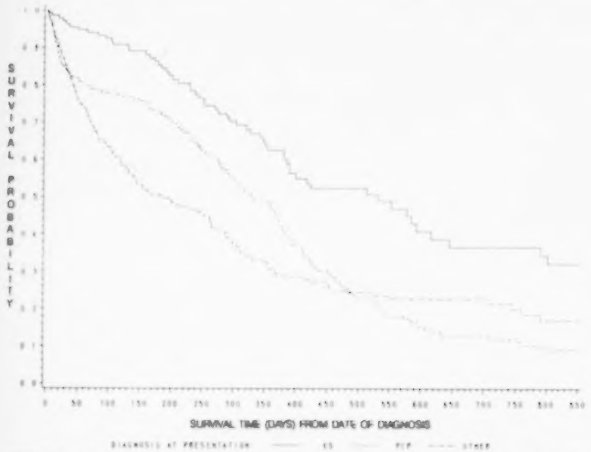

FIGURE 1—Product-Limit Estimate of the Distribution of Survival Time from Date of Diagnosis Among 863 KPNCR AIDS Patients, by Diagnosis (to provide greater detail where most of the sample falls, the distributions are truncated at 850 days).

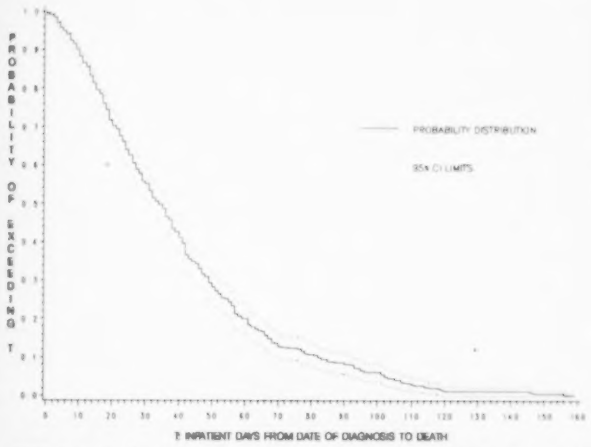

FIGURE 2—Product-Limit Estimate of the Distribution of Lifetime Inpatient Days among 863 KPNCR AIDS Patients Diagnosed between January 1981 and June 1987.

of patients that were relatively high utilizers; an estimated 8 percent were hospitalized more than 90 days. Similarly, 64 percent of the patients required three or fewer hospitalizations, but 8 percent required seven or more.

The distributions of lifetime hospital utilization for all AIDS patients by diagnosis, age, and date of diagnosis are summarized in Tables 2 and 3. The mean number of hospitalizations was 3.3, corresponding to mean and median inpatient days of 40.3 and 34. The distribution of lifetime inpatient days differed among diagnosis groups; those presenting with Kaposi's sarcoma had a lower median number of days compared to those with *P. carinii* and all others. Diagnosis at presentation, however, did not appear to be related to number of hospitalizations. Older AIDS patients experienced fewer hospitalizations but did not differ from younger patients in their inpatient days. Year of diagnosis was not related to either lifetime number of hospitalizations or days hospitalized.

Average length of stay and utilization rates are presented

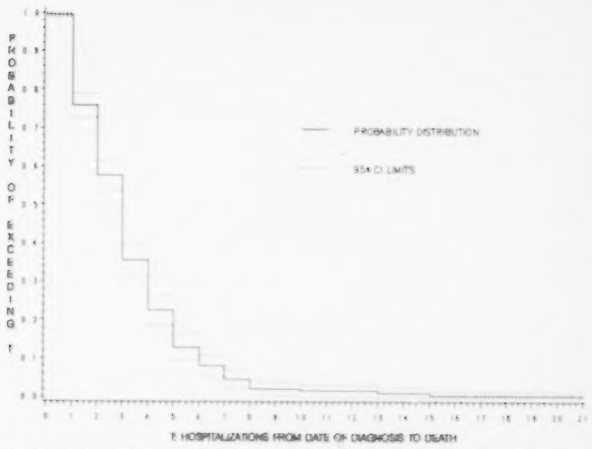

FIGURE 3—Product-Limit Estimate of the Distribution of Lifetime Hospitalizations among 863 KPNCR AIDS Patients Diagnosed between January 1981 and June 1987.

in Table 4. During 1981 through 1983, we observed 105 hospitalizations and 1,767 inpatient days in 32.9 person-years of follow-up on 58 cases, corresponding to utilization rates of 3.2 hospitalizations and 53.7 inpatient days per person-year. Based on 150.1 person-years of follow-up in the first six months of 1987, the hospitalization and inpatient day rates had declined during 1984 through 1986 to 2.6 and 31.1 per person-year, respectively. Similarly, the average length of stay per AIDS-related admission declined from 17.23 days for admissions in 1981, 1982, and 1983 to 10.69 days during the first six months of 1987.

Discussion

Our estimate of mean lifetime inpatient days is substantially lower than the 168 days reported by Hardy, *et al*,[4] and in fact is among the lowest of current estimates as summarized by Bloom and Carliner.[1] Our estimates, however, confirm the finding of other researchers that AIDS patients are hospitalized less often in the San Francisco Bay Area than in other regions of the United States. Scitovsky, *et al*,[3] suggested that strong community-based social support services in San Francisco tend to prevent or shorten hospital stays. They also suggested that in other regions there may be a larger proportion of intravenous drug users, who lack the social support network that other patients with AIDS have developed.

Diagnostic information was obtained from inpatient discharge data for 59 percent of the cases, and thus, there is some misclassification with respect to both date of diagnosis and diseases at presentation. Our estimate of the distribution of survival time from date of diagnosis to death and our utilization analysis by diagnosis subgroup are therefore less reliable than estimates obtained using complete diagnostic information from medical records on all patients. Nevertheless, our survival estimates are in general agreement with other reports from San Francisco.[15] Since it is likely that AIDS patients would generally not be hospitalized without an AIDS-related diagnosis, our primary analyses of inpatient utilization are probably minimally biased by this diagnosis date misclassification.

We examined lifetime hospital utilization among AIDS patients with well-known statistical methods usually used to

TABLE 2—Distribution of Lifetime Inpatient Days among 863 KPNCR AIDS Patients, by Diagnosis at Presentation, Age, and Year of Diagnosis

	N	Median (25th, 75th Percentile)	Mean (S.E.)
Diagnosis			
PCP only	432	35 (17, 56)	39.93 (1.78)
KS only	139	26 (15, 43)	32.33 (3.09)
Other	292	38 (19, 57)	43.38 (2.34)
χ^2 = 8.36, d.f. = 2, P = .015**			
Age at Diagnosis (years)			
0–29	114	38 (16, 68)	47.37 (4.75)
30–39	361	33 (18, 53)	38.83 (1.96)
40–49	262	36 (19, 56)	42.60 (2.59)
50+	126	31 (17, 51)	35.82 (2.54)
χ^2 = 6.18, d.f. = 3, P = .10**			
Date of Diagnosis			
1/81–12/83	58	36 (24, 61)	46.08 (4.76)
1/84–12/84	119	36 (19, 57)	42.71 (2.88)
1/85–12/85	190	36 (19, 53)	39.38 (2.30)
1/86–12/86	282	31 (17, 49)	38.12 (2.48)
1/87–6/87	214	35 (18, 53)	37.73 (4.21)*
χ^2 = 3.85, d.f. = 4, P = .43**			
All cases	863	34 (18, 55)	40.32 (1.32)

*Because the patient with the largest observed number of inpatient days was alive, the product-limit estimate of the mean could not be calculated. The estimate presented is calculated after "uncensoring" this observation and is therefore slightly biased.
**Log rank test for association; d.f. = degrees of freedom.

TABLE 3—Percent Distribution of Lifetime Hospitalizations among 863 KPNCR AIDS Patients by Diagnosis at Presentation, Age, and Year of Diagnosis

		Number of Hospitalizations					
	N	0	1	2	3–4	5+	Mean (S.E.)
Diagnosis							
PCP only	432	0.0	22.8	18.7	32.7	25.8	3.37 (.143)
KS only	139	1.4	21.8	18.8	41.9	16.1	3.11 (.278)
Other	292	0.7	25.2	17.4	36.4	20.3	3.32 (.264)
χ^2 = 1.68, d.f. = 2, P = .43**							
Age at Diagnosis (years)							
0–29	114	0.9	21.6	14.4	35.2	28.0	4.34 (.564)
30–39	361	0.3	22.8	19.2	30.2	27.4	3.26 (.131)
40–49	262	0.0	22.9	18.2	39.9	19.0	3.12 (.152)
50+	126	1.6	28.5	18.6	37.5	13.8	2.93 (.339)
χ^2 = 9.46, d.f. = 3, P = .024**							
Date of Diagnosis							
1/81–12/83	58	1.7	21.8	22.7	37.1	16.6	2.97 (.246)
1/84–12/84	119	0.0	16.5	26.9	34.4	22.2	3.27 (.218)
1/85–12/85	190	1.1	25.9	19.0	32.7	21.3	3.06 (.153)*
1/86–12/86	282	0.4	28.3	11.1	35.3	25.0	3.85 (.455)
1/87–6/87	214	0.0	19.0	17.6	35.2	28.2	3.15 (.221)
χ^2 = 3.40, d.f. = 4, P = .41**							
All Cases	863	0.5	23.6	18.2	35.1	22.6	3.32 (.127)

*Because the patient with the largest observed number of hospitalizations was alive, the product-limit estimate of the mean could not be calculated. The estimate presented is calculated after "uncensoring" this observation and is therefore slightly biased.
**Log rank test for association; d.f. = degrees of freedom.

analyze time to an event. These estimation procedures properly account for the problem of not observing all patients to their deaths. Excluding patients who have not died results in a sample that is biased toward the utilization levels of shorter-lived individuals. Among the patients who died as KPNCR members, the mean number of hospitalizations and inpatient days are 2.6 and 34.6, compared to the product-limit estimates of 3.3 and 40.3; restriction of our sample to the deceased results in a 21 percent reduction in the estimate of mean lifetime number of hospitalizations and a 14 percent reduction in estimated mean inpatient days. Our estimate of the mean lifetime hospital days based on the deceased is extremely close to Scitovsky's[3] estimate of 34.7, which is obtained from a sample of deceased AIDS patients receiving care at San Francisco General Hospital, indicating that the inpatient utilization of the KPNCR patients is similar to that of other AIDS patients in the region.

Our estimates are based on utilization patterns observed only through June 30, 1987. As new therapies are introduced or as patient population characteristics change, our estimates of lifetime utilization may need to be revised. For example, improving survival rates may lead to a change in the inpatient utilization distribution. In this study, there was no evidence of a change in lifetime utilization by year of diagnosis (Tables 2 and 3), although there was a fairly strong downward trend across calendar time in utilization intensity as measured on a person-year basis (Table 4). There was a general increase in survival rates through calendar time. For those with Kaposi's sarcoma, the one year survival rate increased from .60 for those diagnosed in 1981 through 1983 to .65 for those

TABLE 4—Average Length of Stay and Inpatient Utilization Rates among 863 KPNCR AIDS Patients

Admission Date	Average Length of Stay	Observed Person-Years	Number of Hospitalizations* (per person-year)	Inpatient Days** (per person-year)
1/81–12/83	17.23	32.91	3.19	53.69
1/84–12/84	13.78	60.96	3.58	47.68
1/85–12/85	13.56	133.96	3.13	42.67
1/86–12/86	11.72	216.88	2.82	31.43
1/87–6/87	10.69	150.15	2.62	31.10

*Classified according to the date of admission.
**Classified according to the year in which the day occurred.

diagnosed in 1986. For those without Kaposi's sarcoma, the one year survival rate increased from .37 for those diagnosed in 1981 through 1983 to .43 for those diagnosed in 1986. This suggests that the constancy in lifetime utilization while utilization intensity decreased can be explained in part by an increase in survival rates in recent years, i.e., as a group, the same amount of utilization was spread over a longer time period. Also, there appears to be a shift in inpatient utilization from the time near date of diagnosis toward the time of death. Given no change in lifetime utilization, this would result in declining measures of annual utilization intensity during years with less follow-up time close to death than to diagnosis.

Although not presented here, the dependence of utilization on treatment modalities, diagnostic category, and other factors could be examined using methods for regression analysis of survival data.[9,10] In summary, survival analysis methods are appropriate for examining health services utilization in groups of patients with incomplete follow-up. These methods are also useful in estimating the monetary cost of illness with censored data,[6] and an ongoing study of a random sample of KPNCR AIDS patients will examine costs associated with both inpatient and outpatient care.

ACKNOWLEDGMENTS

We gratefully acknowledge the technical assistance of Bill Frank, Merril Jackson, Betty Jue, May Kuwatani, Joanne Owings, Susan Tam, and Joan Thomas.

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Family Access to Health Care: Report Available

Family access to health care is discussed in the report of a conference held in May 1988 at the University of South Carolina and sponsored by the Health Resources and Services Administration.

Single copies of the 136-page report, *Empowering Families for Better Health,* are available from the National Maternal and Child Health Clearinghouse, 38th and R Streets, NW, Washington, DC 20057; tel: 202/625-8410.

Commentary

A Ten-Year Experience in Control of Poliomyelitis through a Combination of Live and Killed Vaccines in Two Developing Areas

THEODORE TULCHINSKY, MD, MPH, YEHIA ABED, MD, MPH, SAID SHAHEEN, MD, NADIM TOUBASSI, MD, YITZCHAK SEVER, MD, MICHAEL SCHOENBAUM, BA, AND RACHEL HANDSHER, MSc

Abstract: We describe a successful program of poliomyelitis control using a combination of killed and live polio vaccines over a 10-year period in two developing areas, the West Bank and Gaza, adjacent to a relatively developed country, Israel. During the 1970s, immunization using live trivalent oral polio vaccine (OPV) in these areas covered more than 90 percent of the infant population. Nevertheless, the incidence of paralytic polio continued to be high, with many cases occurring in fully or partially immunized persons. It was thought that this could be due to interference with OPV take by other enteroviruses present in the environment due to poor sanitary conditions in these areas. A new policy combining five doses of OPV with two doses of inactivated polio vaccine (IPV) was adopted and implemented in 1978. In the 10 years since then, immunization coverage of infants increased to an estimated 95 percent and paralytic poliomyelitis has been controlled, despite exposure to wild poliovirus from neighboring countries including an outbreak in Israel in 1988. This experience suggests that wide coverage using the combination of IPV and OPV is an effective vaccination policy that may make eradication of polio possible even in developing areas. (*Am J Public Health* 1989; 79:1648–1652.)

Introduction

The West Bank is located between Israel and Jordan, and Gaza lies between Israel and Egypt (see map). Israel has governed these areas since June 1967. Prior to that, the West Bank was administered by Jordan, and Gaza was under Egyptian rule. In 1988, the Palestinian population of the West Bank was estimated at 887,000, and that of Gaza was 600,000.[1–4] Population density in the West Bank is about 155 residents per square kilometer, and in Gaza about 1,570.[1–4] Approximately 12 percent of the West Bank population, and 53 percent of the Gaza population, live in refugee camps.[2]

Both the West Bank and Gaza are developing areas. In 1987, per capita gross national product was $2,090 in the West Bank and $1,486 in Gaza (up from $836 and $605, respectively, in 1975). In 1987, the crude birth rate in the West Bank was 41 per 1,000 population, while the Gaza birth rate was 48/1,000. In both areas, about half the population is under age 16.[4] Sanitary conditions have improved steadily over the years but still vary widely, with open sewage drainage remaining a problem in Gaza refugee camps.[4,5]

Persistence of Poliomyelitis

In the early 1970s, the Government Health Services in both areas established an expanded childhood immunization program as a major priority. This included four feedings of oral polio vaccine (OPV) in the first year of life.[4–9] By 1975, 85 percent of infants were receiving polio vaccinations, and by 1977 coverage had exceeded 90 percent.[5,7,8] Despite this, however, there continued to be many cases of paralytic polio throughout the 1970s, with many of these cases occurring in children who had received at least one, and up to four, doses of OPV. In Gaza in 1976, half of the 77 polio cases had been fully immunized with OPV, as had eight of 13 cases in 1977.[5]

In response to this problem, a review of polio policy was undertaken in 1978 by the Government Health Services. Vaccine failure was first thought to be due to a deficient cold chain, but testing indicated that the vaccine used in the field was adequately potent.[5,8–10] Diarrheal diseases were widespread, and it was then felt that these might be interfering with OPV take.[5,7,8] It was decided to modify the immunization program to include a combination of inactivated polio vaccine (IPV) and OPV for both the West bank and Gaza, in addition to emphasizing increased immunization coverage and cold chain integrity.[4–6]

Combined OPV/IPV Policy

Since 1978, IPV has been given in the West Bank in combination with diphtheria-pertussis-tetanus vaccine (DPT) in two doses, at 3-1/2 months and 5 months, in addition to five feedings of OPV, given at 2, 3-1/2, 5, 6-1/2, and 12 months. In Gaza, OPV (type I only) is given at age 1 month, followed by IPV and OPV given together at 2-1/2 and 4 months, and OPV alone at 5-1/2 and 12 months. During 1987–88, adequate supplies of IPV were unavailable, and OPV only was used.

Immunization has reached 95 percent of the infant population in both areas, as indicated by immunization data and by serosurveys.[4,8–11] Vaccinations are given through clinics, village health rooms, and mobile services by the Government Health Services, and through United Nations Relief and Works Agency (UNRWA) clinics, which provide primary health care to residents of refugee camps in both areas. UNRWA has followed the combined immunization policy in Gaza; in the West Bank, where UNRWA provides care for only 12 percent of the total population, UNRWA gives OPV alone.

Cases and suspected cases of paralytic polio are hospitalized and reported to public health officials. There is a high

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level of awareness of polio in the medical and general community, and cases are likely to be reported.[4–6] All suspected cases are investigated clinically and with serial laboratory tests carried out at the Central Virus Laboratory of the Israel Ministry of Health. This includes serum antibody levels and stool cultures. Confirmation of cases is based on clinical data, stool cultures, and demonstration of antibody levels increasing over time.[5]

Polio Control

Figure 1 shows annual incidence rates over the 1968–88 period. Both the West Bank and Gaza experienced a decline in polio cases and incidence rates since 1980. During the period 1968–77, Gaza averaged 41.5 cases per year (incidence rate of 10.6 cases/100,000 population). A dramatic decline in polio incidence began in 1978: average cases per year dropped to 7.4 and 0.6 in the subsequent five-year periods (incidence rates of 1.6 and 0.1/100,000, respectively), with one case in 1988. In the West Bank, polio cases averaged some 20 cases per year (3.2/100,000) in the period 1968–77, and began to decline in 1980. There were an average of eight cases per year between 1978–82 (1.1/100,000) and then two cases per year in the 1978–82 period (0.3/100,000), with two cases in 1988.

During 1968–82, Israel averaged 13 cases of polio per year (average annual incidence rate of 0.4 cases/100,000 population). This fell to an average of 1.6 cases per year between 1983–87 (0.04/100,000), but there were 15 cases in 1988 (Table 1).

Table 2 shows a summary of various serosurveys for polio antibody levels carried out in the West Bank and Gaza.[6,8–9,12–14] While the serosurveys did not group children by vaccination status, high levels of immunity in the latter years are due primarily to increased vaccination coverage and improved seroconversion rather than to circulation of wild polio virus, since the incidence of polio decreased during this time.

The 1988 Israel Polio Outbreak

Since 1963, Israel's national routine immunization policy for polio has been to vaccinate infants with four feedings of OPV only. The continued occurrence of endemic polio during the late 1970s, mainly concentrated in five of 15 subdistricts, led to the institution since 1978 of annual spring booster campaigns using type I OPV among all children ages three years and younger in selected areas of these subdistricts.[12,13] Total reported coverage of polio immunization in Israel is over 90 percent.[12–14]

In two other subdistricts in Israel (with 6.7 percent of the total population in 1986), a trial program of three doses of IPV only was carried out between 1982–88.[6,12–14] In 1988, an outbreak of 15 cases of type I poliovirus occurred in Israel; 12 of these cases were from one of the subdistricts (Hadera) using IPV alone. These were largely concentrated in areas of this subdistrict that had serious sanitation problems. The other three cases were temporally and virologically associated with the outbreak, but there was no confirmed physical contact with Hadera. Nine of the 15 cases were 15 years of age or older; nine had received at least three doses of OPV in the past. This outbreak has been described in detail.*[15–17]

The West Bank and Gaza are not immediately adjacent to each other. However, the distances between the most populated areas of the West Bank, Gaza, and Israel average under 50 kilometers. There is very extensive contact between the populations of all three areas (Figure 2).

Despite this, the population of the West Bank and Gaza was unaffected in the 1988 Israel outbreak, although one of the two polio cases in the West Bank may have been epidemiologically connected to the Israeli outbreak. The other case was vaccine associated.** The one case in Gaza in 1988 was epidemiologically unconnected. However, in view of the risk of spread of the disease from Israel to the West Bank and Gaza, a program of administering a booster dose of trivalent OPV to everyone aged 0–40 was carried out, in conjunction with a similar campaign in Israel. This program reached approximately 90 percent of the target population in the West Bank and Gaza.***

The OPV/IPV Debate

The debate on polio policy has been primarily between the Sabin[18] and Salk[19,20] schools who advocate, respectively, OPV-only and IPV-only vaccination policies. The 10-year experience of the West Bank and Gaza reported here suggests that a combined IPV/OPV program can result in polio control even in developing areas with significant sanitation problems and with high potential for the import of wild virus from neighboring areas where wild polio virus is still prevalent.[21]

Oral polio vaccine (OPV) is relatively inexpensive and easy to administer. It induces both humoral and intestinal immunity (secretory antibody response). Due to its oral administration and consequent presence in the intestinal system, it diffuses immunity directly through contact with the vaccinee, and indirectly through the environment, especially in areas with relatively unsanitary conditions.[18,22–29] OPV-induced immunity among contacts of the vaccinee and through the environment is a major advantage over using IPV alone.[25,27]

FIGURE 1: POLIO INCIDENCE RATES
GAZA, WEST BANK, ISRAEL: 1968–1988

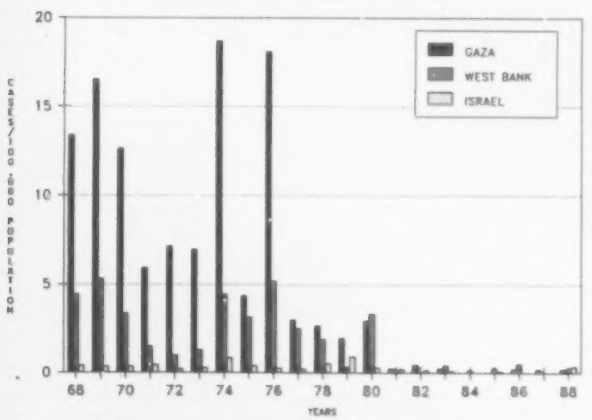

FIGURE 1—Polio Incidence Rates, Gaza, West Bank, Israel: 1968–1988

*Melnick JL, Orenstein WA, Rey M: Report of a consultation on poliomyelitis control in Israel, October 5–6, 1988. Jerusalem: Ministry of Health, 1988 (unpublished); and Slater PE, personal communication, 1989.

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TABLE 1—Confirmed Poliomyelitis by Five-Year Periods: Cases, Average Annual Cases, and Annual Average Incidence Rates, West Bank, Gaza, and Israel, 1968–88

	1968–72	1973–77	1978–82	1983–87	1988
Gaza					
Cases	205	210	37	3	1
Average annual cases	41.0	42.0	7.4	0.6	—
Average annual incidence rate	11.1	10.2	1.6	0.1	0.2
West Bank					
Cases	92	110	40	10	2
Average annual cases	18.4	22.0	8.0	2.0	—
Average annual incidence rate	3.1	3.3	1.1	0.3	0.2
Israel					
Cases	53	67	77	8	15
Average annual cases	10.6	13.4	15.4	1.6	—
Average annual incidence rate	0.3	0.4	0.4	0.04	0.34

NOTE: Incidence rates are cases per 100,000 population.

TABLE 2—Serosurveys for Percent with Protective Antibody Levels by Polio Type, West Bank and Gaza

Area/Age Group	Year	No.	Type I	Type II	Type III
Gaza					
6–36 months	1974	50	56%	74%	66%
	1978	50	60	82	70
	1980	117	100	98	100
	1983	209	89	83	82
West Bank					
6–8 years	1983	240	92	96	97
7 years	1986	266	97	99	96

NOTES: Serosurveys were performed with 1:4 dilutions (1974 in Gaza used 1:10). Survey samples were random (1980 in Gaza was follow-up survey of vaccinees only).

However, successful immunization with OPV requires four doses at appropriate intervals. In addition, because OPV contains an attenuated live virus, there are rare cases (1 per 2.6 million doses in the United States for 1974–84) of vaccine associated disease, in which the vaccinee or a person in contact with the vaccinee incurs paralytic polio from mutated vaccine virus.[22,25,27,30–31] Additionally, there is strong evidence that OPV take is inhibited if the vaccinee is infected with other enteroviruses, resulting in failure of the vaccine to produce satisfactory levels of protective antibodies.[23,24,26,31]

Inactivated polio vaccine (IPV) rapidly produces a high level of protective antibodies. It poses no risk of vaccine-associated disease, and is not susceptible to interference from other enteroviruses.[26] IPV also protects vaccinees (although not necessarily their contacts) from vaccine-associated disease contracted from OPV doses administered subsequently.[25–27] Newly developed enhanced-potency IPV vaccines are even more effective in terms of speed and level of immunogenicity than previous inactivated vaccines.[22,24]

However, IPV produces only limited intestinal immunity. This permits vaccinees who receive only IPV to be acute carriers of polio. The wild polio virus can enter and infect the intestinal tract of vaccinees, who will not themselves contract paralytic polio, but may excrete the virus, spreading it through the environment or directly to other people.*[25–27]

The Combined OPV/IPV Approach

Both vaccine types have proven effective in controlling

FIGURE 2—Map of Israel Showing West Bank, Gaza and Hadera (site of 1988 outbreak)

*Melnick JL, Orenstein WA, Rey M: Op Cit.

polio in developed and developing countries. However, particularly in less than ideal sanitary conditions, neither type used alone seems capable of eradicating the disease. Recently, the combination policy of IPV and OPV administered sequentially has attracted interest internationally.[22,26–27,32–33] Denmark has been using such a policy successfully since 1968.[27,32]

Although poliomyelitis is an acute viral disease affecting humans only, with no animal host and no chronic carrier status, it can be imported by infected persons from outside a given geographical area. Polio virus can then be transmitted by contact or through sewage and waste processing systems.

Developing countries often face great difficulties in vaccinating the numbers of people necessary for adequate herd immunity. In addition, they face problems of interference with vaccine take from other enteroviruses. The policy of combining OPV and IPV is well suited for minimizing the effects of these problems.

As the 1988 polio outbreak in Israel illustrates, an IPV-only vaccination policy for areas with sanitary problems, and where wild polio virus is present, has potential for failure. The 1985 polio outbreak in Finland, even though associated with an inadequately immunogenic IPV (for type 3), further suggests that an IPV-only policy may not produce adequate levels of herd immunity even in areas without serious sanitary problems and where wild polio virus introduction may be unlikely.[21,28] In Holland, where IPV has been used alone, repeated introductions of wild polio virus were associated with relatively large numbers of cases (114 over the period 1974–84), mostly among non-immunized members of religious communities. Introduction of wild polio virus to an area by travelers to and from developing areas is a risk factor that must be considered when determining polio vaccine policy.[25]

The spread of immunity through personal and environmental contact from OPV use is a major advantage; the reduction or elimination of vaccine-associated polio by use of IPV is also a major advantage. The West Bank and Gaza experience show the effectiveness of combining IPV with OPV in areas where OPV alone has a high failure rate.

The combined policy has the further advantage of at least partially mitigating the problem of incomplete vaccination schedules. Giving OPV and IPV at the same time makes it likely that infants, particularly in populations where return visits for further vaccinations cannot be guaranteed, will be protected even after only three visits although a complete schedule is more desirable. The new, high potency IPV's ability to rapidly create high levels of protective antibodies makes seroconversion more likely, even with incomplete vaccination schedules. In the applied field setting, the combined policy can lend itself both to routine campaigns and to the blitz campaign approach used in some developing countries.[24,31,34]

In addition, the combined IPV/OPV policy can reduce or eliminate the risk of vaccine-associated polio, which remains an important factor in the epidemiology of the disease. As an example, four of the eight Israeli polio cases occurring between 1982 and 1987 were vaccine-associated, even though the rate of vaccine-associated polio reported in the United States is considerably lower. For this reason, IPV should be given before or with the first OPV dose, since the greatest danger of vaccine-associated disease comes from the first dose of OPV in individuals previously unvaccinated against polio.[29]

International Implications

The global eradication of smallpox was an outstanding achievement, with enormous benefit to the world. It was achieved by a combination of a safe, effective vaccine, new technology in vaccination, a world consensus on the need to act, and excellent strategies in application of the technologies available.[23] As the World Health Organization (WHO) now has targeted polio for eradication, it is vital to develop the most appropriate strategies in the use of existing and new technologies. Polio eradication will be more complex than was smallpox eradication because of differences in transmission, clinical manifestation, interference, and duration of immunity. Perhaps most importantly, immunity to polio requires multiple doses and boosters, whereas a single dose of smallpox vaccine confers lifelong immunity.[23]

As a result of the importance and complexity of the issue, documented field trials of various approaches to polio control are needed. The West Bank and Gaza experience in use of the combined OPV-IPV approach should be considered for replication in wider field trials in the search for appropriate strategies in polio control/eradication, particularly for developing countries. The Israel Ministry of Health has recently decided to adopt the combined OPV/IPV approach for its future immunization policy. Although the combined approach is more expensive than OPV alone, its advantages make the combined approach an essential tool in the world battle to eradicate polio.

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Business and Nursing Schools Join Ranks to Develop MBA-MSN Degree Programs

Ten of the nation's top-rated graduate schools of business are joining ranks with their graduate schools of nursing to provide future nursing leaders with the same caliber of management, financial, and analytic training usually reserved for the business world. Superior management skills—combined with their clinical skills—are essential to tomorrow's nurse leaders. This unprecedented alliance between the schools of business and nursing "to develop joint MBA-MSN degree programs comes in recognition of the enormous resource allocation, quality control, and employment practice decisions now made by nursing leaders," said Dean Meyer Feldbert of Columbia University Graduate School of Business.

Nursing comprises the largest workforce in the health care industry, and health care is the nation's third largest industry, with expenses approaching $2 billion per day.

Almost 300 nurses with MBA degrees were recently surveyed for The Commonwealth Fund by Louis Harris and Associates, which found that most nurse-MBAs get far more managerial responsibilities as a result of their MBA degree. The most valued skills, according to the nurse-MBAs surveyed, were in finance and marketing. Eighty-eight percent of respondents advised other nurses like themselves to get an MBA degree.

Each of the universities developing the new joint degree programs will receive $100,000 in start-up support from The Commonwealth Fund. The Fund will also provide fellowships of $15,000 each to 25 nurses per year, as further encouragement of this type of education.

The 10 universities developing MBA-MSN graduate degree programs are: University of California (Los Angeles), Case Western Reserve University (Cleveland), Columbia University (New York City), Iowa University (Iowa City), University of Michigan (Ann Arbor), University of Rochester (New York State), Rush University/Northwestern University (Chicago), University of Texas (Austin), Vanderbilt University (Nashville), and University of Virginia (Charlottesville). Courses are due to start in Fall 1990.

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Public Health Briefs

A Comparison of Perceived and Objective CVD Risk in a General Population

Minoo Niknian, PhD, Sonja M. McKinlay, PhD, William Rakowski, PhD, and Richard A. Carleton, MD

Abstract: This report examines correspondence between perceived and objective (American Heart Association's RISKO) risk of heart attack and stroke in a randomly selected sample from two surveys (n = 4,171) conducted in each of two New England cities, in 1981–82 and 1983–84, respectively.

Results confirmed prior reports that people tend to underestimate their CVD (cardiovascular disease) risk and showed that estimates of those at lowest risk were most accurate. (*Am J Public Health* 1989; 79:1653–1654.)

Introduction

Self-assessments of health status have been an integral part of behavioral science research and theory. One question of continuing interest has been the correspondence between "perceived' and "objective" indices of current health. As a general rule, self-assessed health appears to have a basis in actual physical status, using professional or laboratory indices of illness severity as a standard for comparison.[1–3] However, it also seems well-accepted that when discrepancies between personal and objective data occur, individuals are more likely to overestimate their health status.[4–6] This report examines the correspondence between perceived and "objective" risk of heart attack or stroke in a representative community sample.

Methods

Data were collected in two community health surveys, conducted in each of two New England cities in 1981–82 and 1983–84, respectively. Each spanned an entire year to control for seasonal differences. Approximately 5,244 interviews with adults 18–65 years were completed and two blood pressure readings, height, weight, total cholesterol and HDL were measured. Response rate averaged 70 percent.[7]

Two variables were used to construct the dependent variable for this analysis: *Perceived Cardiovascular (CVD) risk* and total *RISKO score* (from the American Heart Association's CVD risk assessment instrument).[8] RISKO was calculated with height, weight, blood pressure, cholesterol and estrogen (for women) as obtained directly from the surveys (n = 4,171). A second, estimated score, using the RISKO substitution rule was analyzed, produced equivalent results and is not reported here.*

RISKO ranges from 0 (lowest risk) to 24 (highest risk) for each of four age × gender groups (<55, ≥55 years; male and female). Three risk categories were constructed from this score: "low risk" was defined as having all except one risk factor at the lowest risk level for those aged <55, producing a maximum score of 5. Similarly, the "high risk" category was defined as having all factors at the third risk level or higher in the younger age category, producing a minimum score of 12. The three objective risk categories, defined on RISKO scores were therefore (0–5, 6–11, 12–24). Because RISKO is conditional on age × sex category, a score of 9 has the same relative meaning of average risk, in each age × sex group.

Perceived risk was assessed as: "Compared with persons of your own age and sex, how would you rate your risk of having a heart attack or stroke within the next five years?" The pre-coded responses were: high (3), average (2), low (1), and don't know. Persons responding to this last category (7 percent) were excluded from subsequent analyses.

Independent variables considered in this analysis were: age (in years), gender, years of schooling, ethnicity (Portuguese, French Canadian, and other), body mass index (Wgt [Kg]/hgt^2 [m^2]); smoking status (yes/no); taking any CVD-related medication (yes/no); and knowledge (whether or not at least one CVD risk factor is mentioned as modifiable).

The discrepant categories between RISKO and perceived risk have possible values (−2, −1, 0, 1, 2), where 0 represents agreement. Using this score as the dependent variable, a stepwise regression[9] was performed on all the independent variables, entering sociodemographic characteristics first. Variables were retained in the model if their individual F-values were at approximately the .01 level and/or induced a relative increase in R^2 of greater than 5 percent. An equivalent discriminant analysis, as a validity check of this approach, produced the same models.* The second stage involved calculation of weighted and unweighted Kappa statistics,[10] which measures agreement between two scoring systems, independently applied to the same subjects.

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*Data available on request to author.

TABLE 1—Unadjusted Kappa and other Agreement Statistics for Comparisons of Objective-Perceived Risk*

Comparison	n	$\hat{K}$	S.e.($\hat{K}$)	% Agreed	% Underestimating Actual Risk	% Overestimating Actual Risk
RISKO	4171	.076	.010	42.8	39.7	17.5

*Note: Reference from all analyses for RISKO using the substitution rule are available upon request.

Results

As expected, educational level had a significant impact on congruence, as did age and gender. Interestingly, knowledge of CVD risk had no association with discrepant scores. Only the two well-known CVD risk factors (smoking and BMI) contributed to explaining discrepant scores, as did use of hypertension medication (diuretic or beta-blocker).

To further investigate the concordance between perceived and actual risk, Kappa statistics were calculated[10] for each level of each of the significant covariates mentioned above as well as the percent of responses in agreement. Females (44.4%) were more likely than males (40.5%), while younger people (44.3%) were more likely than those over age 44 (40.3%) to perceive their risk accurately. Education, dichotomized at 16 years, was positively related to higher agreement (47.1% compared to 42.5%). This is consistent with the regression results using the continuous measure of education. Those at lowest CVD risk [non-smokers (44.6% versus 40.0%) and those with BMI ≤22.5 (49.8% versus 40.7%)] perceived their risk more accurately, as would be anticipated. Those on hypertensive medication also perceived their risk more accurately (45.5%) than those not on medication (42.4%).

Apart from these associations, the overall Kappa was .078 which is low and never exceeds 0.12 indicating that agreement was generally poor. The direction of disagreement was further investigated (Table 1) by considering the percentage under- and overestimating their risk. From these statistics it is evident that twice as many people underestimated their risk, compared to those who overestimated their risk.

Discussion

The analyses presented here lead to two major conclusions. First, there was poor agreement between self-perceived and a more objectively assessed cardiovascular risk, with a high prevalence of risk underestimation. Second, higher levels of agreement between perceived and objective risk are associated with being female, being younger, having more education, being a non-smoker or on hypertensive medication, or having a lower than average BMI. As expected, people at low CVD risk are the most likely to correctly define themselves. Hypertensives, as the clear exception, are sensitized to their higher risk. Knowledge of CVD risk was not independently associated with increased accuracy because of its high correlation with educational level.

The results presented here confirm prior reports that people generally underestimate future risk of negative health outcomes. The important additional contributions of this study are:

- Use of a representative, randomly selected community sample of adults; and
- Use of a validated scoring system (RISKO) to assess cardiovascular disease risk within a specific future period (five years).

In this environment of heightened awareness and decreasing mortality rates, the persistence of CVD risk underestimation is of substantial concern. Clearly there is still considerable room for improvement in health education efforts, at least with respect to CVD.

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hourly employees and the start of programs for salaried employees encouraging early retirement and separation from the corporation.[10] The number of inactive employees (retirees and surviving spouses) grew twice as fast as the active workforce. Dependents, who comprise over half of the GM population,[7] accounted for much of the growth. The number of spouses grew for all employee types except active salaried employees, and grew very rapidly, nearly 3.5 percent per year. In contrast, the number of children hardly grew at all.

Discussion

This is the first study to examine demographic change in a population provided health benefits by a major corporation. It has documented rapid growth, faster than in the US, in this large group. If current trends continue, this population will become larger, older, and have a greater percentage of women.

National health care utilization statistics[11–12] suggest that these demographic changes alone increased GM hospital days of care by 10 percent and physician visits by 7 percent from 1983 to 1987. Consequently, it is not surprising that GM's health care costs rose from $2.2 billion in 1983[5] to nearly $3 billion in 1987.[6]

The trends identified in this study may or may not predict future demographic conditions. Future investigations should estimate the impact on growth rates of employment conditions, sociodemographic processes influencing population growth, and administrative changes.

This study has found that despite increased employment, dependents, particularly spouses, were responsible for most of the population growth. This finding demonstrates that much remains to be learned about how individuals, particularly nonworkers, become eligible for employer-sponsored health coverage. Case studies of employers, such as this one, can illuminate findings from general population surveys about the link between employment and health care coverage.[13] In response to potential legislation that increases the cost of health benefits, employers could pay more attention to who is eligible for these benefits in their efforts to provide cost-effective coverage.

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Fatal Unintended Carbon Monoxide Poisoning in West Virginia from Nonvehicular Sources

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Abstract: Based on medical examiner reports and state vital records, 1978–84, nonvehicular carbon monoxide (CO) caused 62 unintended deaths, representing 42 percent of all unintended fatal CO poisonings in West Virginia. Sources were almost always heating or cooking appliances associated with incomplete combustion of fuels (methane, butane, or propane) not commonly recognized for their potential to produce CO. Hazards included failure to provide recommended venting, neglected maintenance, or use in small areas without natural ventilation. (*Am J Public Health* 1989; 79:1656–1658.)

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Introduction

Carbon monoxide (CO) is the agent most often responsible for fatal, unintended poisoning in the United States.[1] Motor vehicle exhaust causes the majority of cases, but a substantial number occur in domestic or recreational settings[1] where sources are not adequately identified in national mortality statistics[2] or by death certificate review.[3] While heating appliances are recognized as important sources of CO exposure,[3–9] their overall role and specific hazards have not been carefully examined in a population-based study.

Using data obtained principally from the Office of the Chief Medical Examiner (OCME), we have reviewed all deaths in West Virginia attributed to unintended CO exposure from nonvehicular sources during a seven-year period.

Methods

The West Virginia Medical Examiner has jurisdiction over all homicidal, suicidal, accidental, and unattended

natural deaths in the state and maintains a centralized case record file. When indicated, analyses for blood alcohol concentration (BAC) and percent carboxyhemoglobin (COHb) are performed by standard methods.[10,11]

We reviewed the files for the period 1978–84 to identify all certified unintentional deaths attributed to CO from nonvehicular sources, excluding uncontrolled fires. We also reviewed vital statistics for in-state deaths coded "accidental poisoning from gases and vapors" during 1978 (ICD-8 codes E870-7) and 1970–84 (ICD-9 codes E867-9) to identify any additional unintended, nonvehicular CO deaths not reported to the OCME. Data recorded included each victim's age, sex, BAC, and percent COHb, along with date and place of death, and the alleged CO source. Hazards identified at the site of exposure were recorded from information either in the OCME record or acquired through special request to local police or fire departments.

Results

From 1978–84, there were 62 unintentional deaths in West Virginia attributed to CO from nonvehicular sources (two were recorded by vital statistics alone). These deaths occurred in 38 domestic episodes (51 deaths) and in seven recreational settings (11 deaths). On the death certificates, 31 (50 percent) fatalities were not specifically attributed to CO and one was mistakenly associated with motor vehicle exhaust (Table 1).

Postmortem COHb was measured in 47 victims and ranged from 50–88 percent. For eight victims who were not tested, death from CO was determined by association with confirmed cases (three deaths), by CO-positive environmental samples (three deaths), or by elevated premortem COHb (two deaths). For seven remaining victims, CO was assumed the cause based on observations at the scene.

Fatal episodes increased sharply in the fall and peaked during the winter (Figure 1). The male to female victim ratio was almost 4:1 (Table 2). Death rates were highest in young adults and the elderly. Blood alcohol was tested in 40 (70 percent) of the 57 victims ≥15 years of age. Nine (22.5 percent) tested positive, with BAC ranging from 2–46 mmol/L; only three (7.5 percent) had levels consistent with legal intoxication (22 mmol/L).

Residential Settings

Furnaces or space heaters were implicated in 34 (89 percent) of the 38 residential episodes. Cooking ranges were the source of CO in three (8 percent) episodes and a generator was the source in one. Thirty-six units (94 percent) were fueled by natural gas (methane) or butane (only one unit). Table 3 shows that on-site investigations found hazards related to:

- installation of large volume furnaces dependent on direct venting of combustion products to the outside;

TABLE 1—E-Code Classifications on 62 Death Certificates for Unintentional CO Poisoning from Nonvehicular Sources, West Virginia, 1978–84

E-code	Description of Accidental Poisoning	Deaths
ICD-8*		
874	CO from incomplete combustion of domestic fluids	4
875	Other CO	1
ICD-9**		
867	Gas distributed by pipeline	1
868.0	Liquefied petroleum gas in mobile containers	4
868.1	Other and unspecified utility gas	26
868.2	Motor vehicle exhaust gas	1
868.3	CO from incomplete combustion of other domestic fuels	22
868.8	CO from other sources	1
868.9	Unspecified CO	2

*In use for 1978
**In use for 1979–84

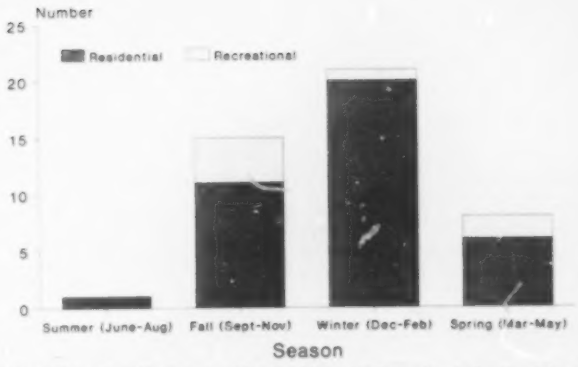

FIGURE 1—Fatal Unintentional Carbon Monoxide Poisonings in Residential and Recreational Settings by Season, West Virginia

TABLE 2—Deaths from Unintentional Nonvehicular CO Exposure by Age and Sex of Victim, West Virginia, 1978–84

Age (years)	Deaths			Rate*
	Male	Female	Total	
0–14	3	2	5	1.6
15–24	13	3	16	6.6
25–44	15	3	18	5.0
45–64	10	3	13	4.7
≥65	8	2	10	6.0
All ages	49	13	62	4.5

*Deaths per 1,000,000 population per year

TABLE 3—Cause of Hazard and Fuel Responsible for Fatal Unintentional Exposure to CO from Nonvehicular Sources by Setting, West Virginia, 1978–84

Setting and Cause of Hazard	Episodes	Fuel
Residential		
Faulty installation		
Failure to install or connect vent	4	Methane (4)
Vent obstructed during installation	3	Methane (3)
Faulty vent design	3	Methane (3)
Neglected maintenance		
Burner malfunction from accumulated debris or faulty adjustment	5	Methane (4), Butane (1)
Damaged vent*	4	Methane (4)
Use in sealed or confined enclosure	13	Methane (12), Kerosene (1)
Miscellaneous	1	Gasoline (1)
Not identified	5	Methane (5)
Recreational		
Use in confined enclosure	7	Butane (2), Propane (3) Charcoal (1), Wood (1)

*Includes combustion product leakage through loose indoor vent joint (three episodes) or backflow caused by downdraft from corroded exterior vent segment (one episode).

- use of smaller units not requiring direct venting but operated in closed rooms. Windows were sealed with plastic in at least four of these 13 episodes;
- neglected maintenance.

Recreational Settings

Five recreational episodes involved the use of butane or propane-fueled heating stoves in poorly ventilated enclosures (two tents, two recreational vans, and a cellar house); one resulted from a smoldering charcoal grill in a recreational van; and one resulted from a poorly vented wood-burning fire in an enclosed outdoor shelter.

Discussion

Despite the elimination of CO from domestic gas since the 1940s and '50s with conversion to natural gas, and the widespread use of other clean burning fuels, domestic and recreational CO exposure remains an important health hazard in West Virginia, contributing 62 (42 percent) of the 149 unintended CO deaths from 1978 through 1984; 87 deaths from motor vehicle exhaust were described earlier.[12] Alcohol use was not a common predisposing factor in these exposures as it was for exposures to motor vehicle exhaust. For surveillance purposes, coded death certificates alone grossly underestimate the problem in nonvehicular settings.

While each of these episodes reflects some failure to adequately remove CO from the living or recreational environment, the vast majority are associated with fuels not commonly recognized for their potential to produce CO.[13] Unlike cruder fuels (gasoline, kerosene, wood, and coal), methane, butane, and propane normally undergo complete combustion in the presence of sufficient oxygen, producing nontoxic carbon dioxide and water vapor.[14] In most instances, CO production by these fuels had at least one obvious explanation:[14]

- operating small volume, unvented gas-burning heaters in closed spaces without natural ventilation (e.g., open windows) for replacing depleted oxygen;
- inadequate venting (removal) of combustion products, displacing oxygen from the ambient supply and/or preventing adequate air flow to the burners. While this was usually associated with faulty installation, it occasionally reflected long-term use and neglected maintenance;
- neglecting to adjust or clean burners, causing inadequate mixing of oxygen and fuel in the combustion chamber.

Prevention therefore depends largely on assuring proper function and appropriate use of gas-burning applicances and can be achieved by professional installation, annual servicing, and education to avoid burning fuels in confined or tightly sealed enclosures. A coordinated educational and inspection program involving health departments, gas companies, and appliance distributors—conducted in the late summer and early fall—would be desirable. Because there is a tendency to conserve heat by closing or sealing windows, terminating the production of unvented units has also been suggested.[7]

Nonlethal CO exposure is a significant health hazard[4,6,7] that can be overlooked when symptoms of headache, nausea, vomiting, and malaise are attributed to other conditions. Several reports of fatalities among persons who had previously consulted a physician[8,9,13] suggest that health care providers can help to identify homes with a CO hazard by recognizing symptoms of exposure.

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Gastrointestinal Effects of Water Reuse for Public Park Irrigation

ROGER DURAND, PHD, AND GERHARD SCHWEBACH, MS, MPA

Abstract: To investigate the gastrointestinal effects of employing recycled water as an irrigation source for urban public parks, we studied subjects active in parks irrigated with potable water, nonpotable water of wastewater origin, and nonpotable water of runoff origin. Wet grass conditions during activity and elevated densities of common indicator bacteria, but not exposure to nonpotable irrigation water per se, were found associated with an increased rate of gastrointestinal illness. (*Am J Public Health* 1989; 79:1659–1660.)

Introduction

Water reuse or recycling has become a widely practiced solution to the water shortage problems of arid and semi-arid regions of the United States.[1–3] To date, however, there is almost no evidence available on the health effects of using recycled water as an irrigation source for public access parks.[4] This paper reports the results of an investigation of the gastrointestinal consequences of employing recycled water for irrigating urban parks. The investigation itself, conducted in the city of Colorado Springs over the period from 1984 to 1987, was designed to answer three research questions:

- Does the irrigation of public access parks with recycled nonpotable, rather than potable, water result in a greater incidence of reported gasrointestinal illness among park users?
- Do higher degrees of exposure to nonpotable water used in irrigation lead to an increased incidence of reported gastrointestinal illness among park users?
- Can an indicator bacteria and bacterial density that is associated with an increased rate of gastrointestinal illness be identified?

Methods

In order to address the research questions, a prospective cohort research design was utilized. Epidemiologic data were collected by means of telephone surveys over two irrigation seasons from randomly selected subjects active in parks irrigated with potable water, nonpotable water of wastewater origin, and nonpotable water of runoff origin. The nonpotable water of wastewater origin utilized for park irrigation was effluent that had been sand-anthracite filtered and chlorinated up to a level of 4–6 mg/L. The nonpotable irrigation water of runoff origin was untreated city runoff that was collected in a pond.

During an initial interview, conducted within two days of park activity, each subject's presence in a park at the scheduled time was confirmed. Each research subject was then asked a battery of questions concerning his/her park activity and exposure to the park's irrigation water. The same subjects were contacted again 10 to 12 days later to determine whether they had experienced an illness in the intervening time period and, if so, the nature of the symptoms they experienced. Following studies sponsored by the Environmental Protection Agency (EPA),[5] "credible symptoms" of gastrointestinal illness were studied as the principal dependent variable. Such symptoms were defined to include reports by subjects of any one of the following accompanying "stomach discomfort": diarrhea, vomiting, cramps, fever, weight loss, excessive gas, or blood in stools.

A total of 2,642 subjects were surveyed on both the initial and follow-up interviews for a completion rate of 94 percent among those potential subjects who could be contacted. Unsuccessful attempts were made to contact an additional 511 subjects. All subjects were screened to avoid including them in the study more than once.

In addition to the survey evidence, microbiological samples of irrigation water were collected periodically in the parks included in the study that were irrigated with nonpotable water. A few samples were also obtained from parks employing potable irrigation water. Analyses were performed on these samples both for common indicator bacteria and for the biological pathogens *Salmonella* and *Shigella* using standard methods of microbial analysis.[6]

Results

Adjusted for the effect of gender, subjects exposed either to wastewater or to run-off water reported credible symptoms of gastrointestinal illness (6 percent and 5 percent, respectively) about as frequently as subjects exposed to potable irrigation water (7 percent) (Table 1). Unadjusted rates for respondents active in parks irrigated with potable water were higher than among counterparts active in parks irrigated with wastewater and runoff at statistically significant levels, but adjustments for variations in the gender and age composition of the parks clientele eliminated this initial difference. Separate controls for type of park activity (soccer and flag football versus golf, softball, and senior citizen games) and for variations in degree of exposure to irrigation water (wet grass, sprayed by a sprinkler, frequency of prior exposure and length of exposure) did not reveal a greater relative incidence of reported symptoms among subjects exposed either to wastewater or to runoff.

Interestingly, wet grass conditions during activity, regardless of type of park irrigation water, were found associated with greater symptoms reporting (adjusted difference = 3%, 95% CI = 1, −1), a finding consistent with some previous research.[7]

To eliminate the effect of interpark mobility or "park switching," we examined the 105 individuals from parks irrigated with potable water who had participated in an activity in a park irrigated with nonpotable water prior to the follow-up survey. The reported rate of illness was the same as in the remaining 966 subjects who did not switch.

Subjects active in parks irrigated with runoff water were found to be exposed to higher average (mean) densities of fecal streptococci, fecal coliforms, and total coliforms than were other subjects. The mean fecal streptococcus and the

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TABLE 1—Reported Symptoms by Park Irrigation Type Adjusting for Demographic Characteristics and Exposure

Park Type/ Adjustment for:	Cases	Total Subjects	Adjusted* Reported Cases (%)	Difference in Adjusted Percentage	95% Confidence Interval
Potable Water					
Unadjusted	90	1071	8	—	—
Gender	90	1071	7	—	—
Age	90	1062	8	—	—
Park activity	90	1071	9	—	—
Wet grass	90	1071	9	—	—
Sprayed	90	1067	8	—	—
Prior exposure	90	1071	10	—	—
Length of exposure	90	1070	10	—	—
Nonpotable wastewater					
Unadjusted	47	1044	5	3	1,5
Gender	47	1044	6	1	−1,3
Age	47	1043	6	2	0,4
Park activity	47	1043	4	5	3,7
Wet grass	47	1044	5	4	2,6
Sprayed	47	1042	4	4	2,6
Prior exposure	47	1043	3	4	2,6
Length of exposure	47	1041	4	6	4,8
Nonpotable run-off water					
Unadjusted	28	527	5	3	0,6
Gender	28	527	5	2	0,4
Age	28	491	5	3	0,6
Park activity	28	527	4	5	2,8
Wet grass	28	527	5	4	1,7
Sprayed	28	522	5	3	0,6
Prior exposure	28	526	7	3	0,6
Length of exposure	28	524	5	5	2,8

*Rate adjustment based on characteristics of total sample.
Gender: male (69), female (31).
Age: 0–15 years (19), 16–65 years (73), over 65 years (8).
Park activity: golf, softball, senior games (74), flag football, soccer (26).
Grass conditions: wet (40), dry (60).
Sprayed: yes (4), no (96).
Prior exposure: frequent (38), infrequent (62).
Length of exposure: less than 3 hours (63), more than 3 hours (37).

mean fecal coliform densities for subjects exposed to wastewater irrigation never exceeded 11.9/100 mL and 5/100 mL, respectively, while those for subjects exposed to runoff exceeded 12,000/100 mL and 3000/100 mL, respectively, in one study year. Similarly, total coliform counts per 100 mL averaged as high as 150 in one year for subjects in parks irrigated with wastewater compared to 60,000 for their runoff counterparts in one of the years. The densities to which subjects active in parks irrigated with potable water were exposed were a constant zero value.

Among subjects exposed to either wastewater or runoff irrigation, the percentage of symptom reports was found to be greater among those active in a park when the 500/100 mL fecal coliform density level was exceeded than when it was not exceeded (Table 2). Identical results were obtained for fecal streptococci at the 500/100 mL level and for total coliforms at the 3,000/100 mL level. Symptom reports were not found to differ significantly at any other bacterial density.

TABLE 2—Credible Symptom Reports by Fecal Coliform Counts/100 mL

	Under Count		Over Count			
Count/ 100 mL	% Reporting Symptom	Cases/ Total Subjects	% Reporting Symptom	Cases/ Total Subjects	Difference in Percent	95% Confidence Interval
200	4	37/922	6	28/439	2	0,4
500	4	37/953	7	28/427	3	1,5
3,000	4	46/1066	6	19/314	2	−1,5

Discussion

These results suggest the utility of fecal coliforms, fecal streptococci, and total coliforms at the identified density levels (500, 500, and 3000 per 100 mL, respectively) in establishing health standards for public park irrigation. Second, they suggest the advisability of park management practices that minimize the exposure of park patrons to wet grass conditions. And finally, they suggest that recycled water can be used for public park irrigation without undue hazard to health, provided that bacteria density levels are kept below those identified here.

ACKNOWLEDGMENTS

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New from nchs

First Births to Older Mothers, 1970–86

STEPHANIE J. VENTURA, AM

The number of first births to women in their thirties has quadrupled in the 16 years, 1970–86.[1] Throughout the 1970s, the increases were largest for women ages 30–34 but, since 1980, the increases in first births and first birth rates have been much larger for women ages 35–39.[1]

In 1986, 181,504 women ages 30–34 gave birth to their first child, more than four times the number (42,404) reported in 1970. Although the typical ages of childbearing continue to be 15–29 years, women in their early thirties accounted for 12 percent of all first births in 1986 compared with just 3 percent in 1970. The rise in first birth rates for these women has been nearly as impressive: 17.5 births per 1,000 women ages 30–34 in 1986 compared with 7.3 in 1970. Increases for women 35–39 were also substantial, although the numbers are considerably smaller. The rate for these women increased from 2.1 to 4.7 during this period (Table 1).

These findings are among those included in a recent report from the National Center for Health Statistics (NCHS) which also includes information on demographic patterns such as educational attainment and marital status of the mother and maternal and infant health characteristics.[1] The data are drawn from the live birth certificates of all States and the District of Columbia.

The increases during the early 1970s in first-time childbearing by women in their thirties occurred at the same time that there were steep reductions in higher order birth rates or, to put it another way, a decline in large families for older women. In the five years from 1970 to 1975, rates for fourth and higher order births for women aged 30 years and older fell by about one-half. As a consequence of these declines, which more than offset the slowly increasing first birth rates, the overall birth rate for women in their thirties dropped during the 1970s (Figure 1).

The reversal of these declines beginning in the mid-1970s is associated entirely with the sizable increases in first-birth rates, while higher order rates continued to decline but at a much slower pace. Higher order births comprised only 20 percent of all births to women aged 30 and older in 1986 compared with 55 percent in 1970.

The dramatic increases measured since 1970 in first-time childbearing by women aged 30 and older could have been anticipated because of several important demographic trends. First, there has been the widespread postponement of marriage by young people and the growing rate of dissolution of their marriages.[2,3] Thus, in 1986, 36 percent of women ages 25–29 years were not married (i.e., never married, widowed, or divorced) compared with 15 percent in 1970.[3]

A second important change has been the sharp decline and then leveling off in first birth rates for women in their twenties, particularly well-educated women, leaving large numbers and proportions of women still childless at age 30 and older. Between 1970 and 1975, first birth rates for women in their early twenties declined by nearly one-third; among women who were college graduates the rates fell by one-half. Some of this decline was made up for by small increases in birth rates for women ages 25–29. But the rates for women ages 25–29 stabilized by the late 1970s and, for college graduates, the rates have fallen since 1980. Because of these changes, large numbers and proportions of women have reached age 30 without having had any children. One-fourth of women ages 30–34 in 1986 were childless compared with 12 percent of comparably aged women in 1970.

A third important factor is the substantial growth in the number of women ages 25–39, from 18.4 million in 1970 to 30.9 million in 1986, a 68-percent increase.[4,5] These women, now ages 25–39 years, were all born in the baby boom years following World War II.

Fourth is the fact that national surveys show that most women intend to have at least one child; only about 10 percent of women in their early thirties expect to remain childless.[6]

Because of the interaction of these demographic patterns it was perhaps inevitable that the first birth rates for women in their thirties would surge upward in the late 1970s and 1980s. That is what has happened.

The educational attainment of women who became mothers at relatively older ages has dramatically increased; a larger proportion of these women than of women in any other age group has completed college. Since 1980, close to half of the women in their thirties having their first child were college graduates, compared with one-fourth of comparable women in 1970 (Figure 2). Birth rates specific for educational attainment provide further evidence that the postponement of childbearing and the making up of delayed first births have been primarily confined to well-educated women. For example, the first birth rate for college graduates ages 20–24 fell from 81 in 1970 to 29 in 1985, and for those ages 25–29 the rate fell from 80 to 58. In contrast, the first birth rate for college graduates ages 30–34 rose from 19 to 36 during these years and for women ages 35–39, the rate increased from 5 to 9 per 1,000 women (Table 2).

Greater educational attainment is associated with more timely receipt of prenatal care and a better outcome in terms of birthweight. For example, of first births to college graduates ages 30–34, only 5.8 percent were low birthweight (less than 2,500 grams or 5 pounds 8 ounces) compared with 7.5 percent of all first births to mothers in this age group.[1] In addition to seeking prenatal care very early in pregnancy, well-educated women are much more likely to be well-nourished and to gain

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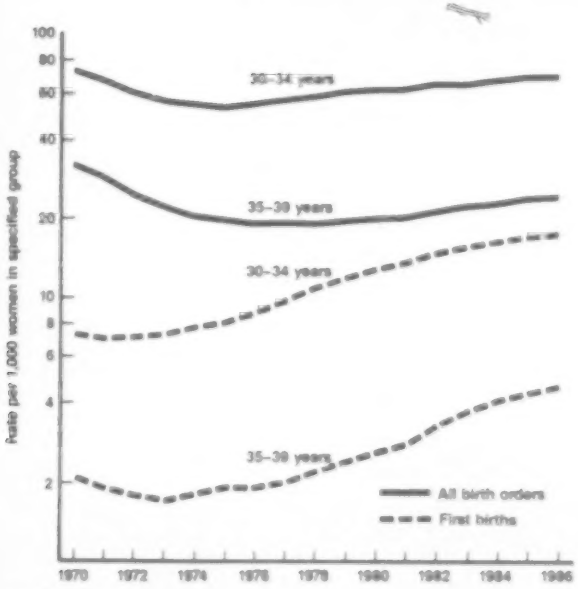
Copies of the full report, Trends and Variations in First Births to Older Women, United States, 1970–86, Vital and Health Statistics Series 21, No. 47, are available from the U.S. Government Printing Office, Washington, DC 20402. Order stock no. 017-022-01081-0, price is $1.75.

TABLE 1—Number of First Births and First Birth Rates by Age of Mother: United States 1970, 1980, 1985, 1986

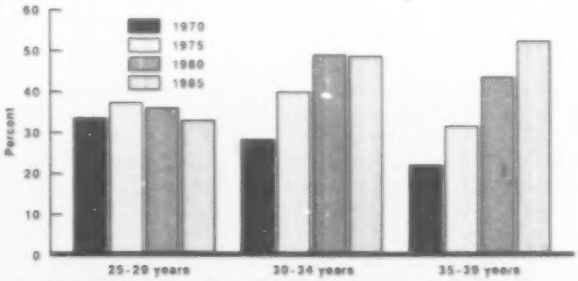
Year	Age of Mother (years)						
	15–44[a]	15–19	20–24	25–29	30–34	35–39	40–44
	Number						
1970	1,430,680	498,388	652,530	212,102	4[illegible],404	11,704	2,442
1980	1,545,604	425,676	605,183	371,859	112,964	18,241	1,964
1985	1,554,788	359,272	552,974	418,658	170,686	39,447	3,804
1986	1,553,751	355,233	534,121	424,088	181,504	44,427	4,419
	Rate[b]						
1970	34.2	53.7	78.2	31.2	7.3	2.1	0.4
1980	29.5	41.4	57.3	38.2	12.8	2.6	0.3
1985	27.6	39.7	53.0	38.8	16.9	4.4	0.5
1986	27.2	39.1	52.7	38.8	17.5	4.7	0.6

[a]Numbers include births to women under age 15 years and ages 45–49 years which are not shown separately. Rates computed by relating first births regardless of age of mother, to women aged 15–44 years.
[b]First births per 1,000 women in specified group.
SOURCE: Reference 1, Tables 1 and 2.



SOURCE: Reference 1, tables 2 and 4

FIGURE 1—Birth Rates for Women Ages 30–34 and 35–39 Years, All Births and First Births, United States, 1970–86



NOTE: Reporting areas increased from 38 states in 1970 to 47 states and the District of Columbia in 1985.
SOURCE: Reference 1, table 7

FIGURE 2—Percent of Women Having Their First Child Who Are College Graduates, by age
NOTE: Reporting areas increased from 38 states in 1970 to 47 states and the District of Columbia in 1985.
SOURCE: Reference 1, Table 7

TABLE 2—First Birth Rates for Women Who are College Graduates, by Age of Mother: United States 1970, 1975, 1980, 1985

Year	Age of Mother (years)			
	20–24	25–29	30–34	35–39
1970	81.1	80.3	19.1	5.0
1975	40.1	64.4	20.1	4.8
1980	39.7	65.8	29.8	6.6
1985	28.9	58.1	35.6	9.4

(Rates are first births per 1,000 women in specified age and educational attainment group)
Source: Reference 1, Table 8.

adequate weight during pregnancy as well as to curtail behaviors such as smoking which could affect pregnancy outcome.[7–9]

In recent years the number of babies born in the United States has been increasing because the number of women in the childbearing ages has risen. Birth rates by age of mother, however, have risen only for relatively older women.[10] The fact that a growing fraction of women giving birth for the first time are 30 years old or older may have implications for health care providers. For example, cesarean deliveries are more likely for older mothers.[11] Older mothers are at greater risk for certain complications,* and their babies are at higher risk of being of low birthweight and of having certain congenital anomalies.[10,12] Additionally, the much greater labor force participation rate of these older mothers may have implications in terms of child care needs. About two-thirds of first-time mothers ages 30–44 were in the labor force in June 1986 compared with half of women having their first child at ages under 25 years.[6] The data in this report can provide a basis for decisions on these medical, health, and child care issues.

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Errata

An error in the printing of Appendix Table 2 was recently found in the article by Sempos, *et al*, in the November 1988 Journal.[1] For 1979, the sum of the age-adjusted mortality rates of Ischemic Heart Disease and Hypertensive Heart Disease for black females should be 129.1. Also for 1979, the sum of the age-adjusted mortality rates of Ischemic Heart Disease, Hypertensive Heart Disease, and Cardiovascular Disease Unspecified for white males, white females, black males, and black females should be 244.9, 111.2, 254.2, and 151.0, respectively.

Also, the SOURCE should read: "Prepared by authors using data from the NCHS, Division of Vital Statistics." The editorial staff of the Journal regrets this error.

REFERENCE

1. Sempos C, Cooper R, Kovar MG, McMillen M: Divergence of the recent trends in coronary mortality for the four major race-sex groups in the United States. Am J Public Health 1988; 78(11):1422–1427.

* * *

In the paper by Lurie, *et al*,[1] May 1989 issue of this Journal, there is an error in the Table 2.

In column three, headed "Diff. (95% CI)," under the main heading of "Free", the % with natural impairment having an eye examination should read (3, 13) rather than (3, 3) as appeared in the published version. The authors regret that this error was not caught in the galleys.

REFERENCE

1. Lurie N, Kamberg CJ, Brook RH, Keeler EB, Newhouse JP: How free care improved vision in the health insurance experiment. Am J Public Health 1989; 79(5):640–2.

* * *

Concerning the letter to the Editor *Comments Received on Walker's 'Odd Man Out' Approach* published in the June 1989 issue of the *American Journal of Public Health* (Vol. 79, No. 6, p 781): The first sentence in the last paragraph should have read "It is not our aim . . ." rather than "It is our aim. . . ." Although it was felt that most readers can tell from the tone of the paragraph that the authors of the letter were not trying to detract from the Walker, *et al*, paper, they nonetheless requested that the Journal notify the readers of this error and express their regret over its occurrence.

Public Health

Featuring. . .

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Effect of Disinfectants on Pseudomonads Colonized on the Interior Surface of PVC Pipes

Roger L. Anderson, PhD, Betty W. Holland, Janice K. Carr, BS, Walter W. Bond, MS, and Martin S. Favero, PhD

Abstract: We investigated the effect of disinfectants on microbial contamination present on the interior surface of polyvinyl chloride (PVC) pipes filled with 600 ml of water contaminated with *Pseudomonas pickettii* and *P. aeruginosa*. After eight weeks, water was removed, and the test pipes exposed to various types of aqueous disinfectants. Disinfectant samples were removed, neutralized, and examined for recovery of microorganisms by membrane filtration. After seven-days exposure, disinfectant solutions were removed and pipes filled with sterile distilled water. Water was examined by membrane filtration at seven-day intervals to determine whether the organisms had survived in the pipes. Colonization of PVC surfaces were examined during each study phase by scanning electron-microscopy (SEM). *P. aeruginosa* was isolated directly from iodophor disinfectant, phenolic germicide, and iodophor antiseptic solutions. After addition of sterile water, *P. aeruginosa* was recovered from PVC pipes previously exposed to chlorine, phenolic, quaternary-ammonium, and iodophor disinfectants; *P. pickettii* was recovered from water in pipes treated with iodophor disinfectant, chlorine, and ethanol. The existence of glycocalyx-like cellular masses on the interior wall of PVC pipes most likely protected embedded organisms from the microbicidal action of some of the disinfectants tested and served as the reservoir for continuous contamination. (*Am J Public Health* 1990; 80:17–21.)

Introduction

The resistance of microorganisms to antimicrobial agents in general and chemical germicides in particular is controlled by a number of factors including the culture history and strain of the microorganism, the nature of the suspending menstruum as well as a variety of physical factors such as temperature, pH, and hardness. Further, microorganisms in their naturally occurring state have been shown to be significantly more resistant to chemical germicides than microorganisms that have been subcultured on artificial culture media.[1,2]

In the last several years, however, there have been reports of bacteria surviving in concentrations of chemical germicides that go significantly beyond the perceived limits of resistance. For example, recent epidemiologic and microbiologic investigations have documented intrinsic microbial contamination of iodophor antiseptic solutions.[3–6] Pseudobacteremia caused by *Pseudomonas cepacia* has been associated with the use of contaminated povidone-iodine, and *Pseudomonas aeruginosa* peritoneal infections has been attributed to the use of contaminated poloxamer-iodine. Presently, epidemiologic and laboratory investigations involving a contaminated povidone-iodine solution are in progress. Peritoneal infections in infants and false positive blood cultures from intensive-care unit patients with *P. cepacia* have been associated with the use of this intrinsically contaminated povidone-iodine antiseptic solution.[7] In addition, the prolonged survival of *P. aeruginosa* in iodophor solutions has been demonstrated after the iodophor was exposed to the inside surfaces of naturally contaminated PVC water distribution pipes.[8,9] When these organisms were isolated from iodine solutions and then tested, high numbers ($>10^6$ per ml) of *P. aeruginosa* were inactivated by iodophor solutions in minutes. More recently, studies have been completed which show recovery of *P. aeruginosa* from iodophors after unused or new PVC, stainless steel, copper, and glass pipes were artificially contaminated with water containing these organisms and subsequently exposed to iodine antiseptic solutions.[8]

The isolation and extended survival of microorganisms in these iodine-containing solutions may be due to extracellular glycocalyx-like material that microorganisms form and deposit on a variety of different types of surfaces.[10–12] Microbial colonization on the interior surfaces of PVC pipes and subsequent protection of organisms from the microbicidal action of antiseptic solutions may be due to the formation and adherence of extracellular matrixes to PVC surfaces. The end result is the production of thick masses of cells and extracellular material (biofilms). Once these masses are formed, antibacterial agents such as disinfectants or antiseptics must saturate the matrix before they can kill bacteria within adhesive biofilms.

Because PVC pipes are commonly used to distribute water in many pharmaceutical and manufacturing plants, in hospital dialysis units, and in various laboratory facilities, we initiated laboratory studies to determine: 1) the effects of various chemical germicidal disinfectants on *Pseudomonas* spp. colonizing the interior wall surfaces of new PVC pipe; 2) the observation and importance of thick cellular masses on PVC surfaces by SEM; and 3) if bacteria survive in and recolonize test pipes after exposure to chemical disinfectants. The purpose of these studies was to formulate a model system to determine the reason for the extraordinary ability of *P. cepacia* and *P. aeruginosa* to survive for extended periods of time after exposure to chemical germicides.

Methods

Preparation of PVC Test Pipes and Sample Remnants

Schedule 80 PVC pipe (4.1 cm in diameter) was used in these investigations. Test pipes consisted of two 30.4 cm PVC sections attached to a 90° PVC elbow. Straight pieces of PVC were separately attached to the elbow by pipe putty (Rectorseal, The Rectorseal Corp, Houston, TX), teflon tape (Teflon Thread Seal, DuPont, Wilmington, DE), organic solvents (Oatey PVC Cement, Oatey Co, Cleveland, OH), or dresser couplings (Flo-Control, Burbank, CA). Thus, four

From the Hospital Infections Program, Center for Infectious Diseases, Centers for Disease Control, Atlanta. Address reprint requests to Roger L. Anderson, PhD, Hospital Infections Program, Centers for Disease Control, 1600 Clifton Road, 1-B341, Atlanta, GA 30333. This paper, submitted to the Journal February 6, 1989, was revised and accepted for publication July 17, 1989.

Editor's Note: See also related editorial p 13 this issue.

sections of PVC (distinguished by the type of attachment) were included in each test group. All PVC test pipes were thoroughly rinsed with tap water, dried, and separately packaged before ethylene oxide sterilization; rubber stoppers used to plug the pipe ends were steam sterilized.

Remnant samples for SEM were prepared by cutting small (1.5 by 1.5 cm) squares of PVC pipe. A small hole was bored at one end and a single 30.5 cm piece of monofilament fishing line tied to each PVC square. Remnant samples were packaged and sterilized with ethylene oxide before suspending them in PVC test pipes.

Microbiologic Conditioning of Test PVC Pipes and Remnant Samples

PVC test pipes (two test groups evaluated each week for four weeks) were filled with 600 ml of water containing a mixed, naturally occurring bacterial population which included only *P. aeruginosa* and *P. pickettii*. These organisms were originally present in naturally contaminated manufacturing plant water and served as the inocula for maintaining a source of contaminated water for these investigations. A reservoir of contaminated water was maintained by inoculating an 18-liter plastic carboy of sterile deionized water with 500 ml of contaminated plant water. Water in the carboy was held at ambient temperature (25°C), and contained a total microbial level of 10^4 colony-forming units (CFU) per ml at the beginning of this study. Initially, contaminated water present in the PVC test pipes contained 10^2 *P. aeruginosa* and 10^2 *P. pickettii* per ml of water.

Five remnant samples were suspended in rubber-plugged pipes filled with contaminated water. Pipes were then stored for eight weeks at 25°C to effect microbial colonization of the interior PVC surfaces. Microbial populations in the pipe waters were sampled weekly using membrane filtration procedures as described. Ten-fold water dilutions were made and passed through sterile sampling tubes attached to bacteriological membrane field monitors (0.45 micron; Millipore Corp, Bedford, MA). PVC remnants were removed at various time intervals for SEM observation of microbial colonization (i.e., formation of cellular and extracellular material accumulations).

Scanning Electron Microscopic Examination of PVC Remnants

For SEM studies, the PVC remnants were fixed in 1 percent glutaraldehyde in Sorenson's PO_4 buffer (pH 4.9) for 16 hours at room temperature, then rinsed three times in PO_4 buffer (pH 7.0). Postfixation was in osmium tetroxide (1 percent in PO_4 buffer, pH 7.0) for four hours at room temperature. The remnants were dehydrated in a graded ethanol series and then placed in a critical-point drying apparatus. Critical-point drying was carried out with CO_2 as the transitional fluid-drying gas. Specimens were loaded into the unit while immersed in 100 percent ethanol, and drying was accomplished by successive changes of liquid Co_2 while the apparatus was chilled by an ice/water bath. The CO_2 was removed by the temperature and pressure being raised to the critical point (32°C and 1100 psi) and slowly exhausted. Specimens were sputter-coated with gold palladium and examined in a Philips SEM 515.

Disinfection of Contaminated PVC Test Pipes

After eight weeks of incubation, contaminated water in the PVC test pipes was removed and cultured quantitatively using ten-fold dilutions and membrane filtration procedures as previously described. Two disinfectants were evaluated each week in two groups of PVC test pipes. Four contaminated PVC test sections (representing the four types of pipe attachment), or one PVC test group, were totally filled (*ca.* 600 ml) with each of the following disinfectants: 1) a 1:213 aqueous dilution (pH 2.9) of an iodophor detergent-germicide (Wescodyne, West Chemical Co, Lynbrook, NY); 2) a 1:128 aqueous dilution (pH 9.2) of a phenolic germicidal detergent (Vesphene II, Vestal Laboratories, St. Louis, MO); 3) a 1:256 aqueous dilution (pH 8.9) of a quaternary-ammonium germicidal detergent (Hi-Tor, Huntington Laboratories Inc, Huntington, IN); 4) a stock solution (pH 5.0) of an iodophor antiseptic (Prepodyne, West-Agro Chemical Co, Inc, Westwood, KS, manufactured for AMSCO Medical Products Division, Erie, PA); 5) 2 percent glutaraldehyde (Cidex, Surgikos, Inc, Arlington, TX) of pH 8.0; 6) a aqueous dilution (pH 7.5) of purified-grade sodium hypochlorite (Fisher Scientific Co, Fair Lawn, NJ) having 10–50 mg of free available chlorine per liter, as determined by the DPD colorimetric test method (Hack Company, Loveland, CO); 7) a 2 percent aqueous dilution (pH 3.4) of formaldehyde (Whiteworth Inc, Gardena, CA); and 8) 70 percent (vol/vol) aqueous ethyl alcohol (Warner-Graham Co, Cockeysville, MD) of pH 5.4. Two control PVC pipes were evaluated with each disinfectant test group: one noncontaminated PVC pipe with disinfectant and one water-contaminated PVC pipe with sterile distilled water.

Aliquots (10 to 50 ml) of the disinfectant were removed and sampled using membrane filtration procedures at 0, 2, 4, and 6 hours and 1, 2, 3, 5, and 7 days. After sampling, each pipe was refilled with an equal amount of the appropriate disinfectant. Filters were rinsed with 250 ml of brain heart infusion broth with 0.5 percent beef extract and the appropriate neutralizer. The filters were placed on blood agar plates. The following chemicals (i.e., in final percent concentration) were added to the enriched broth to neutralize any residual disinfectant: 0.5 percent sodium thiosulfate for iodophors and chlorine solutions; 0.5 percent Tween 80 for phenolics; 0.7 percent lecithin (soybean) for quaternary ammonium compounds; and 0.25 percent sodium bisulfite for aldehydes. Blood agar plates with filters were incubated for 24–48 hours at 35–37°C. The microbial count was recorded per aliquot sampled and subsequently expressed as CFU per ml. To insure that *P. aeruginosa* and *P. pickettii* were the only organisms in the mixed microbial population that was used to condition the PVC pipes, two representative colonies of each morphologic type were picked from the filters and inoculated to triple sugar iron agar slants. Organisms showing nonfermentative reactions on these slants were identified by standard biochemical tests.[13]

Recolonization of PVC Pipes Previously Exposed to Disinfectants

After the contaminated pipes were exposed to disinfectants for seven days, the pipes were emptied and each was refilled with 600 ml of sterile distilled water. Pipes were resealed with fresh sterile rubber stoppers. Water was removed at seven-day intervals and various ten-fold dilutions were examined by membrane filtration procedures as previously described to determine reestablishment of microbial contamination in PVC test pipes previously exposed to chemical disinfectants. After dilutions were filtered, the membrane filters were placed directly on blood agar plates (no neutralizer rinse) and incubated.

Results

Microbiologic Conditioning of PVC Test Pipes

Over the eight-week conditioning period, water counts

for *P. pickettii* (biovar Va-1) were higher (10^5 to 10^6 CFU per ml) than counts observed for *P. aeruginosa* (10^3 to 10^5 CFU per ml). The microbial count for each organism increased each week during the eight-week sampling time. Total viable counts were approximately ten-fold higher in PVC test pipes that used pipe putty (a pipe-threading compound) to attach individual straight pieces to the 90° PVC elbow than pipes that did not have putty.

SEM Examination of PVC Remnants Exposed to Contaminated Water

The presence of bacterial colonization (i.e., formation of cells embedded in extracellular material) on the interior surface of PVC sample remnants was observed by SEM after remnant exposure to contaminated water (Figure 1). Microorganisms were not observed on control PVC remnants (samples not exposed or conditioned with contaminated water).

Recovery of Organisms from Test Disinfectants

P. aeruginosa was recovered directly from an iodophor disinfectant and a phenolic germicidal detergent in 48 hours and from an iodophor antiseptic solution in five days (Table 1). *P. pickettii* was not isolated from any of the test chemicals through seven days of sampling. *P. aeruginosa* and *P. pickettii* organisms were recovered from each of the 32 sterile water controls (i.e., four controls for each test disinfectant). The method of attaching PVC sections to the 90° PVC elbow did not correlate with recovery of organisms from test disinfectants.

Reestablishment of Organisms in Sterile Water

P. aeruginosa was recovered from water placed in PVC pipes previously exposed for seven days to use dilutions (i.e., diluted product as recommended by the manufacturer) of either an iodophor disinfectant, a phenolic germicidal detergent, a quaternary-ammonium compound, an iodophor antiseptic, or a dilution of sodium hypochlorite; *P. pickettii* was recovered from the pipes treated with an iodophor disinfectant, a chlorine solution, or 70 percent ethanol (Table 2). There was no evidence of microbial contamination during 42 days of sampling from contaminated PVC pipes exposed to 2 percent glutaraldehyde or 2 percent formaldehyde and refilled with sterile water. In some instances, organisms were found in water present in the PVC test pipes but were not recovered from the corresponding disinfectant initially used to disinfect that group of pipes.

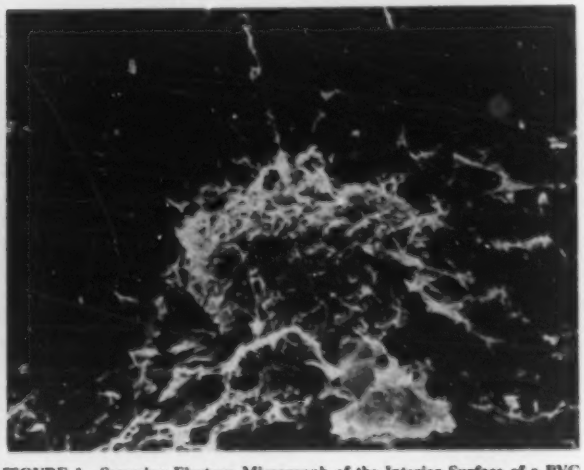

FIGURE 1—Scanning Electron Micrograph of the Interior Surface of a PVC Remnant Sample after Eight Weeks of Exposure to Contaminated Water. × 3,800

Discussion

Previous investigations have demonstrated the prolonged survival of *P. aeruginosa* in naturally contaminated PVC water distribution pipe segments after they were exposed to iodophor antiseptic solutions.[8,9] In one study, *P. aeruginosa* was isolated from a poloxamer-iodine solution 48 hours after membrane-filter sterilized poloxamer-iodine was added to the interior surface of naturally-contaminated PVC water pipe. Continuous exposure of poloxamer-iodine to this pipe resulted in a level of 10^4 CFU per ml of *P. aeruginosa* at nine days. This contaminated solution of poloxamer-iodine was removed from the pipe, stored at 25°C, and served as the reservoir for prolonged microbial survival studies. Using this contaminated stock solution of poloxamer-iodine, we found that *P. aeruginosa* (10^4 CFU per ml) survived for as long as 98 days in stock solution after removal from the PVC pipe.[8] Based on this investigational approach, a suitable laboratory model for induction of survival of these organisms and for studying the mechanism of *Pseudomonas* resistance to iodophors was developed.

In addition, studies have been done in which unused pipes of different composition were artificially contaminated by exposure to contaminated water containing *P. aeruginosa*.[8] After seven days, the water was removed from each pipe, and all were filled with membrane-filtered poloxamer-iodine. *P. aeruginosa* was recovered from poloxamer-iodine after exposure to artificially-contaminated PVC, stainless steel, copper, and glass pipes, but not galvanized steel pipe.

Thus, the newly developed model of using both naturally and artificially contaminated PVC pipes proved useful in demonstrating iodophor-resistance of *P. aeruginosa*. To date, the addition of iodophor antiseptic solutions to contaminated PVC pipe is the only laboratory method now available to show recovery and survival of *P. aeruginosa* from iodophor antiseptics.

We report experiments designed to determine: 1) the effect of various germicides on *Pseudomonas* spp. colonizing PVC pipe; 2) the existence of glycocalyx-like structures as a potential survival mechanism for these organisms; and 3) the reestablishment of *Pseudomonas* spp. in sterile distilled water added to contaminated and disinfectant-treated PVC pipes. *P. aeruginosa* was recovered from three test germicides during a seven-day exposure period: an iodophor disinfectant and phenolic germicide in 48 hours and an iodophor antiseptic in five days. *P. aeruginosa* was not recovered from either a quaternary-ammonium germicidal detergent, 2 percent glutaraldehyde, 10–50 ppm chlorine, 2 percent formaldehyde, or 70 percent ethyl alcohol; *P. pickettii* was not isolated from any of the eight disinfectants tested. The variable microbicidal capabilities of the germicides tested could be due to the type and amount of bacterial colonization present in the PVC pipes, the presence of morphologically abnormal bacterial cytoplasm in cells, or the specific mode and site of action of test disinfectants on bacterial cells (such as microorganisms embedded in a matrix of extracellular material).

Recent review articles have discussed contaminated antiseptics and disinfectants and the various types of orga-

TABLE 1—Recovery of Organisms from Chemical Disinfectants Exposed to Contaminated PVC Test Pipes

Test Pipe Group and Disinfectant Used	Recovery of Organisms per Aliquot of Chemical Sampled:		Recovery of Pseudomonads from Sterile Water Controls:	
	P. aeruginosa	*P. pickettii*	*P. aeruginosa*	*P. pickettii*
A[a]-Iodophor Disinfectant	48h, 2:50 ml	–[b]	+[c]	+
B-Phenolic Germicide	48h, 4:50 ml	–	+	+
C-Quaternary Ammonium Comp.	–	–	+	+
D-Iodophor Antiseptic	5d, 39:10 ml	–	+	+
E-2% Glutaraldehyde	–	–	+	+
F-Chlorine (10-50 ppm)	–	–	+	+
G-2% Formaldehyde	–	–	+	+
H-70% Ethanol	–	–	+	+

[a]Each group consisted of four test PVC pipes.
[b]No recovery after seven days of chemical exposure.
[c]Recovery of organisms from sterile water controls of each disinfectant test group (a total of 32 positive controls).

TABLE 2—Reestablishment of Organisms in Sterile Distilled Water after Exposure of Contaminated PVC Pipes to Disinfectants

Test Pipe Group and Disinfectant Used	Recovery of Organisms from Water after Pipes Were Exposed to Chemicals	
	P. aeruginosa	*P. pickettii*
A[a]-Iodophor Disinfectant	7d, 3.1×10^5/ml	7d, 7.5×10^4/ml
B-Phenolic Germicide	7d, 7.5×10^5/ml	–[b]
C-Quaternary Ammonium Comp.	7d, 25:50 ml	–
D-Iodophor Antiseptic	7d, 2.8×10^2/ml	–
E-2% Glutaraldehyde	–	–
F-Chlorine (10–50ppm)	7d, 2.1×10^3/ml	7d, 3.5×10^5/ml
G-2% Formaldehyde	–	–
H-70% Ethanol	–	21d, 1.4×10^6/ml

[a]Each group consisted of four test PVC pipes.
[b]No recovery after 42 days of water sampling.

nisms associated with these contaminated chemical solutions[14] as well as the resistance of bacteria to antiseptics and disinfectants.[15] A more definitive answer as to why *Pseudomonas* may survive or even grow in germicidal solutions will most likely not be available until laboratory studies are completed which clarify the mechanism of germicidal activity and determine the biologic and ecologic characteristics of *Pseudomonas* spp. that permit their tolerance to or utilization of various chemical compounds.

Massive concentrations of bacterial cells were observed by SEM on the surface of PVC remnant samples after incubation in contaminated water. These cellular masses (or glycocalyx-like structures) appeared to develop in greater numbers as the length of exposure time to contaminated water increased. As suggested in this investigation, the existence of glycocalyx-like structures and the shedding of these structures from interior PVC wall surfaces most likely served to shield *P. aeruginosa* from the microbicidal action of iodophor and phenolic germicides (Table 1).

The protective effect of the bacterial glycocalyx has been suggested by several investigators.[8–12,16] But it should be noted that our preparative techniques for SEM observations were not sufficient to preserve the entire glycocalyx matrix as described by Coseterton, *et al.*[10] Although we observed a considerable degree of microbial adhesion accompanied by notable accumulations of extracellular material on the PVC surfaces, our visual observations are likely an underestimation of the actual amount of physical mass. The prolonged survival of *Serratia marcescens* in 2 percent chlorhexidine was most likely due to these microorganisms being embedded in a thick matrix which adhered to the walls of storage jugs containing this antiseptic solution.[17] Viable *S. marcescens* was recovered from 2 percent chlorhexidine during a storage period of 27 months. The recovery of *P. aeruginosa* from unopened bottles of poloxamer-iodine could be the result of contaminated water and PVC product distribution pipes from a manufacturing plant.[16] SEM micrographs of these pipes demonstrated large concentrations of rod-shaped and coccobacillary cells embedded in interior deposits of the pipe. Also, large numbers of *P. aeruginosa* (10^4 CFU per ml) survived for as long as 98 days in a stock solution of poloxamer-iodine after exposure and removal from naturally contaminated PVC water distribution pipe.[8]

In the present study, microorganisms were observed to survive and reestablish in sterile water placed in PVC pipes previously exposed to chemical germicides. Although *Pseudomonas* spp. were not recovered from some of the test disinfectants, they were isolated from water that was placed in the corresponding chemically-treated test pipes. This reestablishment of microbial contamination suggests a continuous reservoir of organisms adhering to and shedding from the interior PVC wall surface. Organisms showing survival in five PVC test pipes were *P. aeruginosa* (after exposure to chlorine or a quaternary ammonium compound) and *P. pickettii* (after exposure to a iodophor disinfectant, chlorine, or 70 percent ethanol). This is strong evidence that these organisms survived within the colonized PVC pipes after exposure for seven days to a variety of chemical treatments. The physical thickness of cellular and extracellular material that form on PVC pipe surfaces could protect organisms from the action of germicidal chemicals and serve as a continuous reservoir for microbial contamination in test pipes and of water flowing through pipes.

Other investigators have hypothesized the importance of cell masses such as the glycocalyx as a potential reservoir of microbial contamination.[18,19] Several recent investigations have implicated *P. aeruginosa* as the etiologic agent of whirlpool-associated infections (i.e., folliculitis). These organisms are capable of growing in water and colonizing various whirlpool surfaces including water pipes. Such cellular and extracellular material accumulations adhering to whirlpool surfaces could protect bacteria from the action of chlorine and provide a constant microbial reservoir resulting in the contamination of water after a chemical treatment.

The public health significance of this investigation and its findings are widespread. PVC pipes are commonly used in

laboratory facilities, pharmaceutical and manufacturing plants, hospitals, therapeutic and public whirlpool-type hot tubs, and homes to distribute water for various usages. In 1984, during an investigation of a manufacturing plant which produced iodophor antiseptics, *P. aeruginosa* contamination was recovered throughout the PVC water distribution system.[16] Subsequently it was found that intrinsic contamination of a poloxamer-iodine antiseptic solution resulted when the formulated iodophor was allowed to stand in contaminated PVC pipes (pipes between the mixing tank and storage tank and between the storage tank and the bottling area) prior to bottling. As remedial measures, the company removed all PVC pipes, installed new stainless pipe, and initiated a hot water sanitizing procedure (60°C water for one hour) of all process water and product distribution pipes. In addition, better and more frequent microbiological quality control procedures on municipal and process water were instituted.

These types of laboratory studies are important in identifying possible mechanisms by which microorganisms could survive in disinfectant solutions for extended periods of time and to determine how bacterial organisms could continuously contaminate the various parts within a water distribution system. The best way to maintain a water distribution system in a safe condition is to limit the number of microorganisms by proper and scheduled maintenance and to routinely sanitize pipes or tubing that transport water. The laboratory findings presented here add to development of effective strategies for disinfecting water or product distribution lines and holding tanks in manufacturing plants and in laboratory, medical, and pharmaceutical facilities where microbial contamination by pseudomonads or other naturally-occurring gram-negative water bacteria is a problem.

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Public Health Workforce Consortium Recommended

Development of a Public Health Workforce Consortium with public and private representatives—to study health personnel implications of strategic public health issues and improve data collection efforts—has been recommended by a work group established by the Health Resources and Services Administration (HRSA).

The HRSA-supported group recommended the study of selected issues such as those delineated by the Year 2000 Health Objectives for the Nation. It also recommended an incremental approach to the eventual creation of an analytical system capable of assessing the public health work force.

Information on the Public Health Workforce Consortium project and its recommendations is available from B. Jerald McClendon, Division of Associated and Dental Health Professions, Bureau of Health Professions, Room 8C-09, DHHS/PHS, 5600 Fishers Lane, Rockville, MD 20857. Telephone: (301) 443-6757.

First Nursing Home Admissions: Time Spent at Home and in Institutions after Discharge

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Abstract: We tracked 96 percent of a sample of 1,942 nursing home patients admitted to a nursing home for the first time in 1982–83. Patients discharged alive from the nursing home were followed for two years or until death. The relative time spent at home, in hospitals, and in skilled nursing facilities is reported.

Of the 705 patients discharged from their initial nursing home admission to homes in the community, about 50 percent made only one transfer and only 15 percent made four or more transfers. Of the 509 discharged to a hospital, 26 percent died there and 37 percent of the 374 survivors made four or more moves in the next two years. In all, 1,332 patients were discharged alive and they spent almost two-thirds of the subsequent two years, or their remaining lifetimes, in the community. Of those who transferred only once, over two-thirds of their follow-up time was spent in their own homes. Policies concerned with long-term care should use some type of actuarial data base to successfully plan and implement long-term care insurance. (*Am J Public Health* 1990; 80:22–24.)

Introduction

As the United States population continues to age, there is growing concern over the costs of long-term care and the importance of developing mechanisms to finance such care.[1,2] Projections of the costs of an insurance program should be based on data that includes patients' use of all types of services and facilities in the health care system, their survival, and the resources required to provide services to maintain them at home. Despite the size of the long-term care segment of the health care system, and the need for its services, relatively little is known about patients' continued use of services, especially non-institutional services, until death.[3]

Our previous longitudinal studies of defined nursing home populations examined the immediate discharge outcomes of nursing home care in three cohorts of elderly patients (1980, 1982–83, 1984).[4–6] Over time there were no changes in casemix or immediate discharge outcomes of persons admitted to nursing homes for the first time (first admissions).[5] However, a comparison of first admissions and readmissions showed that readmissions were sicker and had worse short-term outcomes.[6] A comparison of readmissions in 1980 and 1984 showed 1984 readmissions were more debilitated than those in 1980 and less likely to return home.[7] Further, readmissions who had been admitted from hospitals in 1984 were almost twice as likely to be rehospitalized within 30 days compared to all readmissions in 1980.

Using a small cohort of patients in 1980, we did a preliminary examination of the number of post-discharge transfers for two years after the initial discharge from the nursing home.[8] In the present study, we provide a detailed report on the continued use of services including time spent at "home" and in institutions, over a two-year period using a 1982–83 discharge cohort of first admissions to nursing homes.

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Methods

A cohort of 1,942 patients discharged in 1982–83 from 45 non-hospital based skilled nursing facilities (SNFs) in the urban areas of San Bernardino/Riverside/Ontario in Southern California comprised the total sample. A detailed account of the data collection methods for this first nursing home admission sample has been reported.[5]

The initial discharge outcomes were classified as:

- returned to home in the community,
- transferred to another nursing home,
- died in the nursing home,
- transferred to the hospital and died there, and
- transferred to the hospital and survived (i.e., subsequently discharged alive from the hospital to a nursing home or to a home in the community).

Patients discharged alive were followed for a period of two years or until death following their initial discharge from a nursing home. The location and associated follow-up information for patients discharged alive were obtained from hospital and nursing home medical records, correspondence with medical record librarians, and telephone interviews with family and friends. This patient relocation information was obtained for two years after the initial nursing home discharge by recording each patient's change of setting: length of stay in that setting, and whether the patient was discharged alive or dead from that setting. Time at home in the community was separated into days spent in the patient's "own home," which included their personal home or that of a relative or friend, days in a retirement home, and days spent in a board and care facility.

Length of life was calculated from the time of discharge from the initial nursing home until death or the end of the two-year follow-up period. Deaths were verified in the county vital statistics records.

This paper describes the associations between number of transfers (0, 1, 2, 3, and 4, or more) and patients' immediate discharge outcomes. It also provides detailed documentation on patients' length of stay in the initial SNF, all subsequent SNFs, hospitals, and homes, including their own (or that of friends or relatives), as well as retirement and board and care homes, according to the number of transfers. Because distributions of survival are skewed, both medians and means are reported.

Results

We successfully tracked 96 percent (N = 1865) of the

total sample of patients (N = 1,942) for the two-year follow-up. Those patients lost to follow-up (N = 77) were excluded from the analysis. At two years, a review of county death records showed that none of the 77 patients lost to follow-up had died in the study area.

Table 1 shows the association between the initial nursing home discharge outcome and the number of subsequent transfers (0–4 or more) for two years. Over one-fourth (N = 533) of the total patients tracked (N = 1,865) died in the nursing home. Almost 30 percent (N = 550) made only one transfer; of these, 11 percent moved to other SNFs, and 64 percent returned to homes in the community. The remainder (25 percent) went to hospitals where they died.

About 18 percent (N = 342) of all patients made two transfers after discharge from the SNF. Of these, 50 percent were discharged to hospitals and survived; 44 percent initially returned to homes in the community; the rest moved to another SNF. Ten percent (N = 187) of all patients made three transfers; of these 16 percent initially went to other SNFs; 35 percent were discharged to hospitals and lived; and almost half returned to homes in the community. The remainder of the patients (N = 253 or about 14 percent) made four or more transfers; only 3 percent of this group were discharged to other SNFs; 55 percent went to hospitals and survived; and 42 percent returned initially to homes in the community.

Put another way, of the 705 patients discharged from their initial nursing home admission to homes in the community, 354 (about 50 percent) made only one transfer and only 15 percent (106/705) made four or more transfers. Of the 509 discharged to a hospital, 26 percent died there; and of the 374 survivors, 37 percent made four or more moves in the next two years.

Table 2 summarizes the survival time of each group making at least one transfer, the proportion alive at two years, and the relative distribution of days of life spent in various locations, i.e., SNFs, hospitals and homes in the community after discharge from the initial nursing home.* An overall summary is also given for all patients discharged alive. The median survival time after discharge for the entire cohort of patients was 157 days. After eliminating those individuals who died in the nursing home, the median survival time was 617 days. Patients discharged alive from the nursing home spent 65 percent of their remaining time during the two-year follow-up in the community. Following discharge from the nursing home, these individuals spent approximately 33 percent of their time in SNFs, 3 percent in hospitals, 11 percent in board/care homes, 3 percent in retirement homes, and 51 percent in their "own" homes.

The median survival in days was 293 for those who transferred twice,and 691 days for those who made three transfers.[9] Because of censored data related to missing information on long-term survivors, we were unable to compute the median survival for individuals who made one and four or more transfers.

As indicated in Table 2, only those making either two or four or more transfers spent the majority of their lives during the two-year follow-up period in institutions. Of those making only one transfer, 53 percent were alive at two years, and as a group they spent over 86 percent of their follow-up days in the community, with over 67 percent of those community days being spent in their "own" homes. For those making three transfers, over half of their post SNF discharge days of life during follow-up were also spent in the community, and the vast majority of that time in the community was spent in the patients' "own" homes. Of the 1,332 patients in Table 2 who spent variable proportions of their remaining days of life at home, 56 percent were disoriented, 46 percent were incontinent of stool and/or urine and 76 percent were dependent in activities of daily living.

*Note that "any home" in the Table includes board/care, retirement and "own" homes.

TABLE 2—Distribution of Time Spent in Various Locations after SNF Discharge and Survival for Two Years According to Number of Transfers

	Number of Transfers				
	1	2	3	≥4	All
No. of Patients	550	342	187	253	1,332
Median LOS in Initial SNF	33	28	36	29	32
% Alive at Two Years:	53	33	49	53	47.2
Median Survival in Days:*	—	293	691	—	617
(Average)**	444	360	483	538	448
% Days Spent In:					
SNF	13.0	49.5	38.1	50.1	32.6
Hospital	0.5	3.1	4.6	5.5	2.7
Board/Care	15.0	7.2	7.6	7.0	10.4
Retirement Home	4.0	2.5	3.0	1.0	2.9
Own Home	67.5	37.7	46.7	36.4	51.4
Any Home***	86.5	47.4	57.3	44.4	64.7

*—Unable to compute median due to censored data related to long-term survivors.
**Censored data causes average survival to be biased because information is lacking on long-term survivors.
***"Any Home" includes all types of homes, i.e., board and care, retirement patient's own home, and the home of a friend or relative.

TABLE 1—Association between Initial Nursing Home Discharge Outcome and Number of Post Discharge Transfers for Two Years

				Transferred from SNF to:			
					Hospitals		
Number of Transfers	Number of Patients	Percentage of Patients	Died in SNF	SNF	Died	Lived	Home*
0	533	28.6	533	0	0	0	0
1	550	29.5	0	61	135	0	354
2	342	18.3	0	19	0	171	152
3	187	10.0	0	29	0	65	93
≥4	253	13.6	0	9	0	138	106
Totals	1,865	100%	533	118	135	374	705

*Home includes all types of homes, i.e., board and care, retirement, patient's own home, and the home of a friend or relative.

Discussion

This study documents the subsequent number of transfers and resource utilization of a discharge cohort of 1,865 patients admitted to skilled nursing facilities for the first time. Almost 30 percent of the sample died in the nursing home, but among those who survived, an unexpected and interesting finding was the length of time many of these patients spent in homes (usually their "own") in the community after being discharged.

The transfer of the frail elderly with variable needs for continued care from one setting to another is a function of several factors. Clearly health status or requirements for care affect placement, especially for those with the greatest need for 24-hour care. The supply of beds, reimbursement policies and patients' economic resources interact to limit length of stay in different facilities. Finally, the preferences of patients and families may well be the determinant of placement at home or in a long-term care facility for those with the personal and or economic resources to exercise this option. This study clearly demonstrates the magnitude of "at home" care utilized by those who survive their first nursing home admission. There are no reported studies, to date, of the relative importance of the variables listed above in determining the mobility of this population, and the non-institutional costs of providing care for them.

Thirty percent of these patients made just one transfer after discharge, and then spent over two-thirds of the succeeding two years, or their remaining lifetime in their own home or that of a friend or relative. There is no reported information related to the family and community resources required for this amount of home care. Over one-half of the individuals making one transfer survived the two-year follow-up period. This represents a considerable amount of time at home. Given the burden of care requirements these patients present, one can only speculate about the degree to which their nursing care requirements were met, the impact of their need for care on family resources, and how (already mentioned this in terms of degree) the challenge was partitioned among family, friends, and other home care providers. Clearly, the need for a more detailed study of patients' continuing care needs (and how these were met) is apparent. Whether or not the families of these individuals managed independently to care for their loved ones or used community nursing agencies is important information for those concerned with policy. Another economically relevant issue that deserves consideration is lost work time for family caregivers.

Information on the specific care requirements for the significant portion of the persons in the sample who were living at home two years after discharge is particularly essential. Such data (while not collected in this study) can best be derived from longitudinal tracking of patients, a research design not commonly used in nursing home studies.[10]

As indicated in previous publications, the area for this study represents the 37 standard statistical metropolitan (SMSA) areas throughout the country with the exception that it has a lower proportion of Blacks and individuals below the poverty level. It may or may not represent the population in the eight urban SMSAs, e.g., New York City, Los Angeles, San Francisco, etc.

This data set does provide a fairly detailed picture of the natural history of continuing care for a modest sample of patients for two years after discharge from nursing homes. Estimates of costs may be derived from the number of days spent in various institutional settings (hospitals, skilled nursing facilities, board and care homes). Advocates of long-term care insurance will require some type of actuarial data base of individual patients' utilization of facilities and resources to successfully plan and implement long-term care insurance. However, what is conspicuously missing from this report is information on the services provided to these individuals and their social and economic costs during those intervals spent at home. More information of this type is essential to planning for the care of the rapidly growing numbers of frail elderly, and assisting families to cope with the burdens of their care.

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Cocaine and the Risk of Low Birth Weight

Diana B. Petitti, MD, MPH, and Charlotte Coleman, BA

Abstract: In a population-based case-control study, we assessed the relation between cocaine use and the risk of low birth weight in Alameda County, California, excluding women who used heroin and/or methadone during pregnancy. After adjustment for confounders, the relative risk of low birth weight in users of cocaine throughout pregnancy was 4.0 (95% CI = 1.8, 8.9). Cocaine use was estimated to account for 10 percent of cases of low birth weight in Black women in Alameda County. (*Am J Public Health* 1990; 80:25–28.)

Introduction

Cocaine use affects pregnancy outcome adversely in animals.[1,2] In humans, it has been associated with an increased risk of abruptio placenta[3,4] and of intrauterine growth retardation and pre-term delivery.[5–11] In a population-based case-control study of low birth weight in Alameda County, California, we estimated the relative risk of low birth weight in relation to use of cocaine and the contribution of cocaine use to the problem of low birth weight in this urban population.

Methods

Subjects were identified using information from Alameda County, California birth certificates. Eligible were singleton infants without congenital anomalies whose mother and father were both Black or both White, non-Hispanic, born in Alameda County between January 1, 1987 and December 31, 1987 to residents of Alameda County. Cases weighed 500 through 2499 grams. Controls were chosen at random from among infants with weights of 3000 or more grams, the number of controls in each ethnic group being equal to the number of cases.

We identified 500 Black cases and 500 Black controls and 268 White, non-Hispanic cases and 268 White, non-Hispanic controls for the study, but nine records were subsequently judged ineligible. After obtaining permission from the California Vital Statistics Advisory Committee to use information from the confidential section of the birth certificate and the approval of our study protocol and interview instrument by two local committees for protection of human subjects, we used information on the birth certificate to locate women, who were given a detailed explanation of the study's aims, of the risks and benefits of participation, along with assurances of the confidential nature of the information provided. Consenting women were interviewed in their homes by trained interviewers. Ultimately, 766 of 994 eligible Black mothers (77.1 percent) and 462 of 533 eligible White mothers (86.7 percent) were interviewed.

Women were separately queried about use of cocaine and of "crack" or freebase cocaine, which is the smoking of pure crystalline cocaine.[12] Information about other potential confounders was also collected by interview.

Cases of low birth weight were sub-classified into three groups: ≤1500 grams (very low birth weight), 1501–2500 grams and <37 menstrual weeks of gestation (low birth weight, pre-term), and 1501–2500 grams and ≥37 menstrual weeks of gestation (low birth weight, term, intrauterine growth retarded) according to suggestion of Yerushalmy.[13]

The relative risk of low birth weight was estimated using the exposure odds ratio. The chi-square statistic was used for polychotomous variable. Ninety-five percent confidence intervals were estimated using the method of Cornfield,[14] except when these failed to converge, in which case the method of Kleinbaum and Kupper was used.[15] Logistic regression as implemented on SAS[16] was used to estimate relative risk controlling for confounders.

The population attributable risk percent was estimated using methods described by Walter.[17] The population prevalence of cocaine use in Whites and Blacks was estimated as the sum of the number of cases who used cocaine and the number of controls who used cocaine multiplied by the relevant sampling ratio (26.32 for Whites and 7.75 for Blacks) divided by the estimated total number of White or Black deliveries in Alameda County.

Results

Seven Black women and four White women who used heroin and/or methadone during pregnancy, and four Black women with incomplete information on cocaine use, were excluded from all analyses. After these exclusions, both the use of cocaine in forms other than crack throughout pregnancy and the use of crack throughout pregnancy were associated with large increases in the risk of low birth weight (Table 1).

No White women admitted to use of crack or cocaine in other forms throughout pregnancy, and multivariate analysis was confined to Blacks. Because of the small number of controls who admitted to use of cocaine or crack throughout pregnancy, we combined the categories for cocaine and crack use throughout pregnancy. After adjustment for confounders, the increased relative risk of low birth weight in women who used cocaine and/or crack throughout pregnancy persisted but was attenuated (Table 2).

Low pre-pregnancy weight and low weight gain may be caused at least in part by use of cocaine, and a history of having a prior low birth weight infant may be related to use of cocaine in the prior pregnancy. Thus, a multivariate statistical model that includes these factors may "over

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TABLE 1—Estimated Relative Risk of Low Birth Weight by Cocaine Use in Blacks and Whites

Variables	Blacks N Cases	Blacks N Controls	Blacks RR (95% CI)	Whites N Cases	Whites N Controls	Whites RR (95% CI)
Self-Reported Cocaine Use during Pregnancy						
Did not use at all	297	375	1.0[a]	218	234	1.0[a]
Used cocaine and/or crack, not throughout pregnancy	14	5	3.5 (1.2, 11.4)	2	4	0.5 (0.1, 3.0)
Used crack with or without cocaine in other forms throughout pregnancy	32	4	10.1 (3.5, 28.9)	0	0	—[b]
Used cocaine but not crack throughout pregnancy	23	5	5.8 (2.1, 17.6)	0	0	—[b]

[a]referent
[b]cannot be estimated

TABLE 2—Results of Three Multiple Logistic Regression Analyses Examining Cocaine/Crack Use and the Relative Risk of Low Birth Weight Controlling for Various Other Variables[a]

Variables	Full Model RR (95% CI)	Excluding Weight Gain <15 Pounds RR (95% CI)	Excluding Weight Gain <15 Pounds, Low Pre-Pregnancy Weight RR (95% CI)
Self-Reported Cocaine/Crack Use during Pregnancy			
Not at all	1.0[b]	1.0[b]	1.0[b]
Used cocaine and/or crack but not throughout pregnancy	1.8 (0.5, 6.4)	2.4 (0.7, 7.9)	2.3 (0.7, 7.6)
Used cocaine and/or crack throughout pregnancy	4.0 (1.8, 8.9)	4.4 (2.0, 9.7)	4.4 (2.0, 9.6)
Cigarette Smoking during Pregnancy			
Not at all	1.0[b]	1.0[b]	1.0[b]
Used but quit	1.3 (0.6, 2.6)	1.3 (0.7, 2.5)	1.3 (0.7, 2.5)
Used throughout	3.1 (2.1, 4.5)	3.2 (2.2, 4.7)	3.3 (2.2, 4.7)
Maternal Age (years)			
<17	1.7 (0.7, 3.8)	1.8 (0.8, 3.9)	1.8 (0.8, 3.8)
17–34	1.0[b]	1.0[b]	1.0[b]
35+	1.9 (1.0, 3.5)	2.0 (1.1, 3.7)	1.9 (1.0, 3.5)
Prior Live Births			
0	2.3 (1.5, 3.3)	2.1 (1.4, 3.1)	2.2 (1.5, 3.2)
1–2	1.0[b]	1.0[b]	1.0[b]
3+	1.4 (0.8, 2.5)	1.2 (0.7, 2.2)	1.3 (0.7, 2.3)
Alcohol Use during Pregnancy			
<3 drinks/day	1.0[b]	1.0[b]	1.0[b]
3+ drinks/day	1.7 (0.7, 4.1)	1.5 (0.6, 3.6)	1.4 (0.6, 3.5)
Socioeconomic Status Index[c]			
High	1.0[b]	1.0[b]	1.0[b]
Middle	1.1 (0.7, 1.7)	1.2 (0.8, 1.8)	1.2 (0.8, 1.9)
Low	1.5 (0.9, 2.3)	1.7 (1.1, 2.7)	1.7 (1.1, 2.7)
Prior Low Birth Weight Baby			
No	1.0[b]	1.0[b]	1.0[b]
Yes	6.9 (3.7, 13.2)	7.5 (4.0, 14.0)	7.6 (4.1, 14.1)
Low Pre-pregnancy Weight[d]			
No	1.0[b]	1.0[b]	—[e]
Yes	2.2 (1.4, 3.6)	2.2 (1.3, 3.5)	—[e]
Weight Gain <15 Pounds			
No	1.0[b]	—[e]	—[e]
Yes	4.6 (2.6, 7.8)	—[e]	—[e]

[a]Analysis confined to Blacks.
[b]Referent.
[c]Index takes into account education (4 categories) and income (5 categories) and places equal percentages of the total Black and White population in three tertiles.
[d]Pre-pregnancy Quetelet Index (wt in pounds/height in inches^2 × 100) ≤2.7.
[e]Variable not included in this model.

TABLE 3—Estimated Relative Risk of Various Sub-Categories of Low Birth Weight by Cocaine Use[a]

Variables	Controls N	≤1500 g Cases N	≤1500 g RR[b] (95% CI)	1501–2500 g <37 weeks Cases N	1501–2500 g <37 weeks RR[b] (95% CI)	1501–2500 g ≥37 weeks Cases N	1501–2500 g ≥37 weeks RR[b] (95% CI)
Self-Reported Cocaine Use							
Did not use at all	375	78	1.0[c]	116	1.0[c]	91	1.0[c]
Used cocaine and or crack cocaine, but not throughout pregnancy	5	3	2.9 (0.7, 12.4)	5	3.2 (0.9, 11.3)	4	3.3 (0.9, 12.5)
Used cocaine and or crack cocaine throughout pregnancy	9	12	6.4 (2.4, 17.2)	20	7.2 (3.0, 17.6)	19	8.7 (3.6, 21.6)

[a]Analysis confined to Blacks; excludes 19 women who could not be classified because gestational age was unknown.
[b]Reference group for this analysis is all Black controls.
[c]Referent.

control" by adjusting for these factors. In multivariate analyses that excluded these variables and adjusted for confounders, the relative risk of having a low birthweight infant in women who used cocaine and/or crack throughout pregnancy was still elevated by a factor of about 4 to 5 (Table 2).

The use of cocaine and/or crack throughout pregnancy increased the relative risk of very low birth weight, pre-term low birth weight, and intrauterine growth retardation in Blacks by about the same amount (Table 3).

Based on an estimate that the prevalence of use of cocaine and/or crack throughout pregnancy is 3.9 percent among Black women in Alameda County and an estimate that the relative risk of low birth weight in users of cocaine and/or crack throughout pregnancy is 4.0 after adjustment for confounding, the attributable risk of low birth weight for cocaine and/or crack use throughout pregnancy in Black women in Alameda County can be estimated to be 0.10.*

Discussion

This study joins a growing body of research[3–11] that links maternal cocaine use with poor pregnancy outcome. The study is unique in being population-based. Results show that cocaine use increases the risk of both pre-term delivery and intrauterine growth retardation, a finding that also is in keeping with the findings of most other similar studies,[5–11] that of Chasnoff, *et al*,[4] being an exception.

This study has some important limitations:

- We did not interview all of the women eligible for the study, although consideration of the characteristics of the non-interviewed women, who mostly could not be located, suggest they would be *more* likely than those we interviewed to use cocaine.
- Information on cocaine use was based on self-report. It is unlikely that women would overreport use of an illegal drug, thus spuriously increasing the risk of low birth weight in association with cocaine use, but we cannot exclude this possibility entirely.
- Mothers of cases of low birth weight might have selectively overreported cocaine use because of concern or guilt about an adverse outcome, or for other reasons. The number of mothers of cases who selectively overreported cocaine use would have to be very large to explain our findings.
- Women may have reported cocaine use but not use of drugs, such as heroin, that are known to reduce birth weight. It is not easy to understand why women would report cocaine use and not use of other illegal drugs, but this explanation for our results cannot be excluded.
- Our study is limited in its ability to draw conclusions about dose-response, because our definition of cocaine use is imprecise. We do not have information about the amount of cocaine use or the timing of its use during pregnancy, because we did not ask these questions.

Considering these limitations and in the context of a large body of data linking cocaine use with intrauterine growth retardation and pre-term delivery,[3–11] we conclude that cocaine use is an important contributor to the public health problem of low birth weight in the Black population of Alameda County, and probably in other urban settings where there is substantial use of the drug. Women who cease cocaine use in the first trimester of pregnancy have been shown to have no increase in the risk of pre-term delivery or growth retardation.[9] Even though specific programs to reduce cocaine use during pregnancy have not been evaluated, the evidence linking cocaine use with poor pregnancy outcome appears sufficient to warrant interventions that provide counseling and a broad-based community effort to identify women early who use cocaine in their pregnancies. The magnitude of the increase in the risk of poor pregnancy outcome in cocaine users suggests that the success of efforts to improve infant and perinatal mortality in settings where use is common may be dependent on addressing the problem of cocaine use successfully.

*Data available on request to author.

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U-MI Offers Non-Residential Graduate Programs in Health Administration and Policy

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For additional information and application forms on either program, contact: David Perlman, MPH, OJ/OC/Pew Program Manager, School of Public Health, University of Michigan, M3525 SPH II, Ann Arbor, MI 48109-2029. Telephone: (313) 936-1191.

Cigarette Smoking as a Risk Factor for Sudden Infant Death Syndrome: A Population-Based Study

BENGT HAGLUND, DRMEDSC, AND SVEN CNATTINGIUS, MD

Abstract: Risk factors for sudden infant death syndrome (SIDS) were examined in a prospective study based on Swedish births between 1983 and 1985. All infants surviving the first week of life were included (279,938). The overall rate of SIDS was 0.7 per 1,000 first week survivors. Elevated relative risks were associated with low maternal age, multiparity, maternal smoking, and male infants. Smoking doubled the risk and a clear dose-response relation by amount smoked was observed. Maternal smoking also seemed to influence the time of death, as infants of smokers died at an earlier age. In countries like Sweden, smoking may be the single most important preventable risk factor for sudden infant death syndrome. (*Am J Public Health* 1990; 80:29–32.)

Introduction

Although syndromes similar to sudden infant death syndrome (SIDS) have been known since biblical time, progress in solving the enigma of SIDS has been slow. The incidence of SIDS varies widely, being low in Scandinavia[1] and higher in other industrialized countries, e.g., in North America. In Sweden[2] and Finland,[3] the incidence of SIDS has doubled since the 1970s, despite reductions in the infant mortality rate of almost 40 percent.[2]

SIDS and other causes of infant death share some risk factors, but the age at death distinguishes SIDS from the other causes of death.[4] SIDS victims usually die within one to six months of age; for unknown reasons, there are two peaks of age at death: eight to nine weeks, and 13 to 15 weeks.[2,5]

Low social class and maternal smoking have previously been associated with SIDS.[1,3,4] Since smoking is more frequent in lower social classes,[6] it is uncertain whether the increased risk for SIDS should be ascribed primarily to smoking or low social class.

In 1973, a medical birth registry was set up by the National Board of Health and Welfare in Sweden,[7] and, since 1982, information has been collected prospectively for each pregnancy from the first antenatal visit. The registry provided an excellent opportunity to investigate the influence of maternal smoking and other risk factors on SIDS in an unselected, homogeneous low-risk population.

Methods

The Swedish National Board of Health and Welfare receives demographic data, previous reproductive history and pregnancy outcome on all hospital births. This is transmitted as copies of the standardized antenatal and obstetrical records, which are generally used in Sweden. In the first trimester, midwives at the antenatal care clinics record previous reproductive history, smoking habits, and family situation. Practically all Swedish women visit these clinics regularly during pregnancy.[8] All births reported to the birth registry are validated every year against a population registry at Statistics Sweden, which receives information from the parishes about all births and deaths. This cross-checking is done by using the mother's personal identification number. The Medical Birth Registry covers more than 99 percent of all births in Sweden.[9]

During the study period from 1983 to 1985, 280,256 infants survived the first week of life. The study was restricted to births of mothers between 15 and 44 years of age. The 318 births for which maternal age was either unknown or outside this interval were excluded, leaving a total of 279,938 infants for study. Ninety-five percent of these infants were born to Scandinavian women.

In Sweden, the death certificate includes information about when the death occurred, possible causes of death, and whether or not an autopsy was performed. In the present investigation, 698 infants were reported to have died between one and 51 weeks of age. SIDS was reported as the only cause of death in 190 infants (International Classification of Diseases, ICD 8, 1968, 795,99). Autopsies were performed on 98 percent of the cases (94 percent forensic and 4 percent clinical). Most deaths from other causes also had autopsies (58 percent clinical and 12 percent forensic).

Parity was defined as the number of live births plus stillbirths. Smoking habits were categorized as non-smoker (i.e., non-daily smoker), moderate smoker (smoking between one and nine cigarettes per day), or heavy smoker (10 cigarettes or more per day). Since it is common in Sweden for young couples to live together without being married, information about family situation was recorded as whether or not living with infant's father.

Multiple logistic regression analyses were performed in order to calculate associations of the independent variables on SIDS as estimated by the relative risk.[10] The independent variables used in the analyses were maternal age, parity, family situation, smoking habits, type of birth (single or multiple), and sex of infant. The continuous variables were categorized in order to avoid assumptions about linearity. The statistical analyses were made using the SAS program package.[11,12]

Results

Mortality from SIDS, 190 cases or 0.7 per 1,000 first week survivors during the three years, accounted for about 27 percent of the total infant mortality after the first week of life. Table 1 shows that maternal age was inversely associated with risk for SIDS. Other characteristics of the mothers associated with an excess number of SIDS were high parity, not cohabiting with infant's father and the number of cigarettes smoked per day. Higher risks for boys and for multiple births were also seen.

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TABLE 1—Number of Infants Surviving the first Week of Life and SIDS Death Rate by Characteristics of the Mother and Infant, Sweden, 1983–85

Characteristics	Infants No.	SIDS No.	SIDS Death-rate per 1000
Maternal Age at Delivery (years)			
15–19	9,574	14	1.46
20–24	66,582	53	0.80
25–29	103,527	68	0.66
30–34	68,838	41	0.60
35–39	27,155	12	0.44
40–44	4,262	2	0.47
Parity			
1	112,994	52	0.46
2	101,323	75	0.74
3	47,333	38	0.80
≥4	18,288	25	1.37
Family Situation			
Living with father	253,588	164	0.65
Not living with father	14,436	17	1.18
Not available	11,914	9	0.76
Smoking Habits			
Non-smoker	181,218	88	0.49
Daily smoker			
1–9 cigarettes	48,465	45	0.93
≥10 cigarettes	31,225	46	1.47
Not available	19,030	11	0.58
Sex of Infant			
Boy	143,981	110	0.76
Girl	135,391	79	0.58
Not available	566	1	...
Type of Birth			
Single	274,526	184	0.67
Multiple	5,407	6	1.11
Not available	5	0	...
Total	279,938	190	0.68

Smoking habits varied with age and family situation: half of the teenage mothers smoked compared with 25 to 29 percent of mothers aged 25 or more. More than half of women who were not living with infant's father were daily smokers, compared to 29 percent of those living with infant's father.[13] We therefore adjusted the estimates for the effects of other variables using a multiple logistic regression model (Table 2).

As seen in Table 2, high parity strongly increased the risk for SIDS, but high maternal age reduced the risk of SIDS. It is plausible that the effect of parity varies with age, but we detected no interaction between age and parity ($p = 0.6$). Infants not living with their father had 50 percent greater SIDS mortality than those living with their father (95% CI = 0.8, 2.5).

Maternal smoking was strongly related to SIDS even when controlling for other risk factors. Smoking up to nine cigarettes per day doubled the risk of SIDS and smoking 10 cigarettes or more per day nearly tripled the risk of SIDS, compared to non-smokers. We examined whether smoking increased the risk of SIDS mortality more for certain women than others, but no clear interactions were detected (smallest p-value = 0.11).

Age at Death of SIDS Infants

The age at death from SIDS ranged widely with a peak incidence at nine to 12 weeks. The age distribution reported from other studies—two peaks during the first six months of life[2,5]—was not clearly observable in our study. Only when the age of death was grouped by three-week intervals was there a weak tendency toward an early and a late peak.

The SIDS victims were stratified into two groups of equal size. However, before this was done, the 10 percent with the longest survival time were excluded in order to obtain groups including only cases with typical ages at death. Early SIDS were defined as deaths occurring between seven and 67 days of age and late SIDS were those between 68 and 145 days of age. Although the magnitude of the risks differed between the two models, they were in general rather similar (Table 2). However, moderate maternal smoking almost tripled the risk for early SIDS, while no increase in the risk for late SIDS appeared. A similar but less marked pattern was true for infants not living with their father.

For further analyses of the difference between early and late SIDS, a logistic regression model based only on the SIDS cases was performed, to estimate the probability of dying before 68 days of age among the SIDS victims. The analysis showed that moderate maternal smoking was strongly associated with increased risk for early SIDS (RR 1.7, 95% CI = 1.2, 2.1). SIDS victims whose mothers were heavy smokers (RR 1.4, 95% CI = 0.9, 1.4), or not living with infants father (RR 1.8, 95% CI = 0.9, 2.3), also had an increased risk for short survival, while infants of mothers 35 years of older seemed to have higher probability for longer survival (RR 0.4, 95% CI = 0.1, 1.1).

The influence of maternal smoking on the time of dying from SIDS, is illustrated in Figure 1. Infants of non-smoking mothers had a symmetric age distribution with a mode value of nine to 12 weeks. Infants of smoking mothers however, had a right-skewed age distribution with a shorter modal time of survival, five to eight weeks.

Discussion

In the present study, the diagnosis of SIDS was based on cases where the death certificate stated SIDS as the only cause of death. As 98 percent of these cases underwent autopsy (and 94 percent forensic autopsy), the diagnostic accuracy in these cases must be considered as sufficient. The diagnostic difficulties of SIDS, when using death certificates, mainly concern underregistration,[2] so the incidence estimated (0.7 per 1000) may be an underestimate.

Maternal smoking has earlier been reported as a risk factor for SIDS.[5,14,15] In the present study, a strong association between maternal smoking and SIDS was found. We have no direct measures of confounders associated with smoking that might adversely affect the infant. For instance, we have no information on whether there were other smokers in the household. However, adjustment for the effects of maternal age, parity, and family situation only marginally reduced the estimates. Such socio-demographic variables are also related to behavioral and lifestyle characteristics that could adversely affect the infant. It therefore seems unlikely that unmeasured confounders would appreciably influence the association. The present results were based on a prospective study, including about 280,000 infants. The population studied was a relatively homogeneous, low-risk population, which further suggests a limited influence of possible confounders.

The information on smoking habits was based on the mothers' reports to the midwives at their first visit to antenatal care. As practically all women in Sweden regularly visit these clinics during pregnancy, the influence of possible selection bias seems negligible.[8] Since pregnant women today are exposed to anti-smoking information, this may have influenced the validity of self-reported smoking habits.

TABLE 2—Relative Risks* of Sudden Infant Death Syndrome Based on a Logistic Regression Model (257,361 Infants Surviving the first Week of Life in Sweden 1983–85)

	All SIDS (1–51 weeks) Relative risk (95% CI)	Early SIDS (7–67 days) Relative risk (95% CI)	Late SIDS (68–145 days) Relative risk (95% CI)
Maternal Age (years)			
15–19	2.0 (1.0, 4.0)	1.5 (0.5, 4.4)	2.6 (0.9, 7.6)
20–29 (ref. group)	1.0	1.0	1.0
30–34	0.7 (0.5, 1.0)	0.6 (0.3, 1.0)	0.8 (0.5, 1.5)
35–44	0.5 (0.2, 0.8)	0.3 (0.1, 0.8)	0.8 (0.4, 1.7)
Parity			
1 (ref. group)	1.0	1.0	1.0
2–3	2.2 (1.5, 3.3)	2.8 (1.6, 4.8)	2.2 (1.2, 3.9)
≥4	4.8 (2.7, 8.4)	5.0 (2.1, 11.9)	5.0 (2.2, 11.4)
Family Situation			
Living with father (ref. group)	1.0	1.0	1.0
Not living with father	1.5 (0.8, 2.5)	1.9 (0.9, 4.0)	0.7 (0.2, 2.2)
Smoking Habits			
Non-smoker (ref. group)	1.0	1.0	1.0
Daily smoker			
1–9 cigarettes	1.8 (1.2, 2.6)	2.8 (1.7, 4.8)	1.0 (0.5, 1.8)
≥10 cigarettes	2.7 (1.9, 3.9)	3.6 (2.1, 6.2)	2.0 (1.1, 3.5)
Sex of Infant			
Boy	1.4 (1.1, 1.9)	1.2 (0.8, 1.9)	1.7 (1.1, 2.7)
Girl (ref. group)	1.0	1.0	1.0
Type of Birth			
Single (ref. group)	1.0	1.0	1.0
Multiple	1.5 (0.7, 3.4)	2.1 (0.8, 5.8)	1.1 (0.3, 4.5)

*Estimates are adjusted for all other variables.

Moreover, women may also have stopped smoking during pregnancy. It has been reported elsewhere that 15 to 20 percent of pregnant smokers quit "on their own" during pregnancy.[16,17] Thus, if a causal relationship between maternal smoking and SIDS exists, the true relative risk may be even greater than estimated herein.

Other studies have reported low birthweight to be associated with SIDS[3,18] and smoking to be associated with low birthweight.[17] Because the effect of maternal smoking on SIDS could be mediated by low birthweight, we also conducted an alternative analysis controlling for low birthweight (<2500 grams). We found almost the same risk estimates for smoking (RR = 1.7 and RR = 2.5 for moderate and heavy smoking, respectively) indicating that smoking affects birthweight-specific as well as overall SIDS mortality.

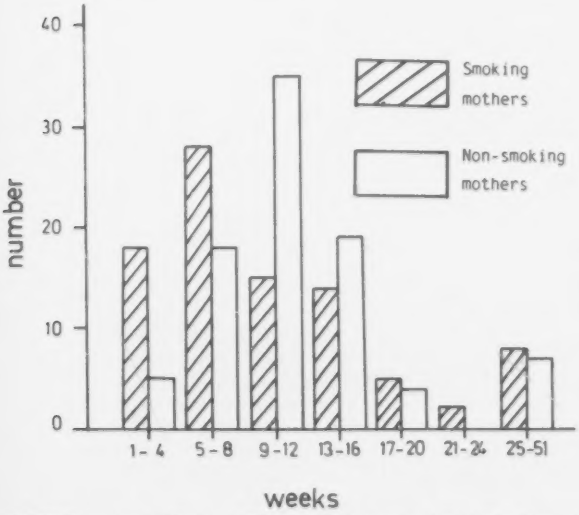

FIGURE 1—Age in Weeks of SIDS Infants in Sweden 1983–85 by Maternal Smoking Habits

The socioeconomic factor included in this study (cohabiting vs not cohabiting parents) has previously been associated with SIDS in Scandinavia,[2,19] but information about maternal smoking was not included in those studies. In the present study, the crude relative risk of SIDS was 1.9 in non-cohabiting families, but daily smoking was more common among non-cohabiting women. Adjusted for the effects of differences in smoking habits, the relative risk was reduced to 1.5 (95% CI = 0.8, 2.5). These data suggest that smoking is a confounding factor, through which at least some of the effects of social class on SIDS rate are mediated, at least in the homogeneous populations of Sweden, where poverty and malnutrition are practically non-existent.

The previously reported bimodal age distribution of SIDS[2,5] led us to stratify the SIDS cases into early and late SIDS. The most striking difference in risk factor pattern between early and late SIDS was the influence of maternal smoking, a finding in accordance with Golding, *et al.*[5] Early SIDS was more strongly related to maternal smoking. We suggest that the reported bimodal age distribution may partly reflect differences in maternal smoking.

Today, smoking is probably the most important preventable risk factor for late fetal death.[13,20] The present results suggest that this may also hold true for SIDS. Assuming that maternal smoking could be eliminated and infants delivered by mothers who gave up smoking faced the same mortality risk as their non-smoking peers (i.e., same maternal age, parity, sex, etc.), we estimated that SIDS death rate would be reduced by 27 percent. In terms of number of SIDS deaths, this means a reduction from 190 deaths to approximately 140 deaths during a three-year period in Sweden.

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An earlier version of this study was presented orally in Swedish at the annual meeting of the Swedish Medical Association in December 1988.

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Past Pregnancy Outcomes among Women Living in the Vicinity of a Lead Smelter in Kosovo, Yugoslavia

Mary J. Murphy, MD, MPH, Joseph H. Graziano, PhD, Dusan Popovac, MD, PhD, Jennie K. Kline, PhD, Ali Mehmeti, MD, Pam Factor-Litvak, MS, Gemail Ahmedi, MD, Patrick Shrout, PhD, Biljana Rajovic, MD, Dragoslav U. Nenezic, MD, and Zena A. Stein, MB

Abstract: This analysis compares the rates of spontaneous abortion among women living in the vicinity of a lead smelter with those of women living in a town where blood lead levels were low. Data derive from the obstetric histories of both groups of women obtained while seeking prenatal care for a later pregnancy. A total of 639 women (304 exposed, 335 unexposed) had at least one previous pregnancy and lived at the same address since their first pregnancy. The geometric mean blood lead concentrations in the sample at the time of the interviews were 0.77 μmol/L in the exposed town and 0.25 μmol/L in the unexposed town.

The rates of spontaneous abortions in first pregnancies were similar, with 16.4 percent of women in the exposed town and 14.0 percent in the unexposed town reporting loss. The adjusted odds ratio relating town of residence to spontaneous abortion was 1.1 (95% CI = 0.9, 1.4). This analysis represents the first systematic attempt to seek an association between environmental lead exposure and spontaneous abortion. As such, the failure to find a positive association strongly suggests that at the levels of exposure represented in our sample, such an association does not exist. (*Am J Public Health* 1990; 80:33–35.)

Introduction

Historical anecdotes have suggested that occupational exposure to lead may be detrimental to reproductive outcome. Pregnant women working in the pottery and white lead industries in England in the nineteenth century were reported to be more likely to miscarry than women not so employed; other reports from Italy, Germany, France, and Japan also describe high rates of loss among pregnant women exposed to lead as compared to women without exposure.[1] While most samples lack both precise measures of exposure and information on potentially confounding factors, taken together they suggest that high levels of lead might be harmful to the developing conceptus. This suggestion is consistent with a series of reports from Sweden, where Nordstrom and co-workers found a significantly increased frequency of spontaneous abortion among female employees working at a lead smelter.[2] Similar observations were made on populations living in close proximity to that smelter.[3]

In contrast, a recent prospective cohort study of pregnant women living near a lead smelter in Australia reported no association between maternal blood lead (BPb) and spontaneous abortion.[4] That study, however, may have suffered from incomplete ascertainment of the event.[5] The present report examines the rates of spontaneous abortion in two comparable populations, one living in the vicinity of a lead smelter and the other in a town 25 miles away without significant environmental lead exposure.

Methods

The subjects of this analysis were recruited for a prospective cohort study of environmental lead exposure, reproductive outcome, and infant development. Further description of the population and methods of data collection have been presented in a recent publication.[6] The past reproductive histories of these women provide the data for this report. In brief, the study took place in Titova Mitrovica (T. Mitrovica), Yugoslavia, where there is significant environmental exposure to lead from a lead smelter, refinery, and battery plant, and in Pristina, Yugoslavia, a town without significant lead exposure. The prospective cohort study recruited women in mid-pregnancy. At that time an interview inquiring about medical and reproductive history and sociodemographic factors was administered.

A total of 1,502 women was recruited at mid-pregnancy. This analysis draws on data from the first 1,032 recruited during the period of May 1985–May 1986. Of the 1,032 women evaluated, 777 (332 in T. Mitrovica and 445 in Pristina) had had at least one previous pregnancy. In T. Mitrovica, the current BPb varies with distance from the smelter, such that women living close to the smelter have, on average, higher blood lead (BPb) levels than those living further away.[6] Since it is reasonable to assume that this variation in BPbs with distance from the smelter was also true in the past, it is only among women who have not moved since their first pregnancy that we can be assured of previous lead exposure during that pregnancy. Accordingly, this analysis was confined to 639 women (304 in T. Mitrovica and 335 in Pristina) who had lived at their current address at least since their first pregnancy.

In these analyses, spontaneous abortion was defined as a fetal loss prior to the seventh month of gestation; stillbirth was defined as a fetal loss occurring in months seven through nine of gestation. Only the first pregnancy of each woman was used in the analysis. This strategy avoids the problem of nonindependence of reproductive outcomes within individuals.[7,8] Maximum likelihood logistic regression analysis was used to assess the influence of lead on spontaneous abortion, after adjustment for the effects of potentially confounding variables.[9]

Results

Table 1 shows the demographic characteristics of the 639 women with at least one previous pregnancy who lived in the same home since their first pregnancy. Mean parental ages at

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TABLE 1—Demographic Characteristics at Study Recruitment

Characteristics	T. Mitrovica	Pristina
Number of women	304	335
Mean maternal age at first pregnancy	21.7 (3.9)*	22.1 (3.4)
Mean paternal age at first pregnancy	26.0 (4.8)	25.6 (4.0)
Mean maternal education (years)	8.7 (4.0)	8.7 (3.9)
Mean paternal education (years)	11.3 (2.7)	11.2 (3.0)
Mean number of previous pregnancies	2.8 (2.2)	2.6 (2.0)
% Current cigarette smokers	23.0	26.3
% Current alcohol users	5.9	10.5
% Same address since birth	31.0	30.2
Mean years at current address	13.6 (10.1)	7.0 (6.4)
% Husbands employed in Pb industry	28.8	—
Ethnicity		
% Albanian	59.3	63.3
% Serbian	21.5	25.6
% Other	19.2	11.1

*Standard deviation.

first pregnancy, mean parental years of school at the later index pregnancy, and gravidity were similar in the two towns. Women in Pristina compared to women in T. Mitrovica were slightly more likely to be current smokers and twice as likely to be current alcohol users. While approximately 30 percent of women had lived in their respective towns since birth, women in T. Mitrovica had lived an average of six years longer at their current address than women in Pristina. Within this sample, the proportions of women of Albanian and Serbian origin were higher in Pristina.

Hematologic findings at the time of study recruitment revealed the expected differences in BPb and erythrocyte protoporphyrin (EP) (Table 2). Mean BPb in T. Mitrovica was 0.77 μmol/L as compared to 0.25 μmol/L in Pristina. The mean hemoglobin concentrations were within normal limits in both towns.

The rates of spontaneous abortions were similar in T. Mitrovica (16.4 percent) and Pristina (14.0 percent) (Table 3). Stillbirth rates were also similar across towns. When all fetal losses were combined, the rate in T. Mitrovica was 19.7 percent; the rate in Pristina was 17.9 percent. These rates remained essentially unchanged when adjusted for the number of induced abortions in each town.[10]

Adjustment for the effects of maternal age at first pregnancy, current smoking, ethnic group, and maternal education by maximum likelihood logistic regression produced little change in the odds ratio relating town of residence and spontaneous abortion (odds ratio 1.1) (Table 3). Refinement of the exposure variable using distance of the home from the smelter showed no consistent trend in the odds ratio using Pristina as the reference group. For women living within 2 km of the smelter, the adjusted odds ratio was 1.2 (95% confidence interval = 0.7, 1.9); for those within 2–4 km it was 2.1 (0.8, 5.3); for those within 4–8 km it was 0.5 (0.2, 1.2); and for those beyond 8 km it was 0.8 (0.6, 1.6). Analysis of the data restricted to women who reported never drinking alcohol showed similar results.

TABLE 2—Current Blood Lead and Hematology Findings

	T. Mitrovica	Pristina
Number of women	304	335
Blood Lead (μmol/L)[a]	0.77 (0.32, 1.83)	0.25 (0.12, 0.51)
Hemoglobin (g/L)[b]	125 (103, 147)	123 (94, 151)
EP (μmol/L)[a]	0.71 (0.21, 2.41)	0.50 (0.22, 1.17)
Serum Ferritin (μg/L)[a]	12.5 (1.9, 82.9)	10.8 (1.4, 85.0)

[a]Geometric means (95% confidence interval)
[b]Arithmetic means (95% confidence interval)

TABLE 3—First Pregnancy Outcome Rates

	T. Mitrovica	Pristina	Odds Ratio[c]
	% (number)	% (number)	(95% CI)
Spontaneous Abortions[a]	16.4 (50)	14.0 (47)	1.1 (0.9, 1.4)
Stillbirths[b]	3.9 (10)	4.5 (13)	1.0 (0.6, 1.5)
Livebirths	80.3 (244)	82.1 (275)	—

[a]Denominator excludes reported induced abortions, ectopic pregnancies, and pregnancies where the outcome was unknown.
[b]Denominator excludes spontaneous abortions.
[c]Adjusted for maternal age, current smoking, ethnic group, and maternal education.

Discussion

These data indicate that lead exposure, at the levels encountered in T. Mitrovica, is not a risk factor for spontaneous abortion. The adjusted odds ratio was 1.1 with an upper bound of 1.4 suggesting larger effects are unlikely. There was 80 percent power to detect an odds ratio of 1.6 (alpha = 0.05, two tailed).

In order to increase our confidence that this result is real and not due to bias, we examined carefully the basis for measuring exposure, outcome, and confounding variables. Town of current residence is the index of exposure in this analysis and the population of women examined lived at least since their first pregnancy in the same home. While current blood lead is a marker of recent exposure, the striking differences in current blood leads between these two groups, together with the fact that these women had not moved since their first pregnancies, make it likely that exposure at first pregnancy similarly differed across towns. Moreover, previous blood and air lead survey data from these two towns indicate that the levels of exposure in T. Mitrovica were probably higher in the past.[11] Thus, previous levels of lead exposure such as might have occurred in the first pregnancies of these study women are likely to have been higher than those recorded at present. We remain assured that the index of exposure is an appropriate one, that the exposure differs across towns, and that the levels of exposure in T. Mitrovica among currently pregnant women are significantly higher than those in Pristina. We note, however, that the level of lead exposure of our subjects in T. Mitrovica does not nearly approach that typically found among women exposed to lead through occupation.

In reviewing this study, one could speculate about the possible effects of random or differential misclassification of spontaneous abortion. Insofar as spontaneous abortion rates are similar to those reported in other samples, the stillbirth rates comparable to those found in less developed countries, there is no basis for positing high levels of misclassification. Differential misclassification might be produced if recall differed between towns. In order to produce the null result observed here, we must posit bias in opposite directions for the two towns: underreporting in T. Mitrovica and overreporting in Pristina. Again, since the rates of loss in both towns fall well within the bounds usually reported, such biasing effects seem unlikely.

Previous work which examined the relation of lead exposure to spontaneous abortion revealed inconsistent find-

ings. The initial historic reports which suggested an association are largely based on anecdotal reports of women working in lead-related industries where more severe and multiple occupational exposures may have been operating. These observations gave rise to the hypothesis of a causal relation, but did not provide systematic evidence of such an association. The Swedish smelter studies,[2,3] although suggestive of an effect of environmental exposure on spontaneous abortion, were largely ecologic and thus do not provide strong support for an association at the individual level.

The only prospective cohort study to address the issue of lead and reproductive outcome assessed 831 pregnant women living near a lead smelter in Australia.[4] In that study, mean blood lead levels (std error) among the exposed were relatively low, ranging from 0.51 ± 0.01 to 0.54 ± 0.01 μmol/L. Blood lead and other risk factors were examined in relation to pregnancy outcome and no association was found between maternal BPb and spontaneous abortion. However, the number of abortions ascertained was small (n = 23) raising the question of incomplete ascertainment of the event.[5] The failure to find a positive association in the current study strongly suggests that, at the levels of exposure represented in our sample, such an association does not exist.

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Call for Proposals: Improving Service Systems for the Physically Disabled

The largest single commitment of private funds ever provided to benefit people with disabilities has been announced by the Robert Wood Johnson Foundation of Princeton, NJ. The Foundation will award up to $8.4 million to community-based organizations run by and for the disabled. As many as 12 one-year planning grants of up to $100,000 will be made under the Foundation's new program, "Improving Service Systems for People with Disabilities."

The grant funds are intended to help community organizations such as Independent Living Centers create comprehensive service systems so that the disabled can live independently. Community organizations that receive the planning grants also will be eligible to compete for three-year grants of up to $600,000 to carry out their plans to coordinate available health and supportive services, develop new services, and identify public and private financing sources for these services.

The application deadline is January 16, 1990. Organizations intending to apply should contact Lex Frieden, Independent Living Research Utilization, 3400 Bissonnet, Suite 101, Houston, Texas, 77005 or call 713/528-0504. The grants awards will be announced in the fall of 1990.

The Effect of a Patient Education Program on Emergency Room Use for Inner-City Children with Asthma

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Abstract: An educational program for children with asthma designed to reduce emergency room (ER) use enrolled all eligible children (n = 253 primarily low-income Black) within a health maintenance organization (HMO) who had used the hospital or ER for asthma during the pre-enrollment period and randomized them into two groups. Twenty-four of the experimental group patients had 55 ER visits and 18 of the control patients had 39 ER visits during the first 12 months post-intervention. This program did not achieve its goal. (*Am J Public Health* 1990; 80:36–38.)

Introduction

Asthma is a common disease that leads to considerable morbidity and expense.[1] Lethal asthma is a serious problem and continues to increase in frequency, especially for minority patients.[2] The years of potential life lost due to asthma almost doubled for minority males between 1979 and 1983.[3] One of the strategies to reduce morbidity, mortality, and expense of asthma has been patient education. The program evaluated in this paper was designed to improve patients' knowledge and behaviors related to asthma in order to improve their control of the disease, and thereby reduce emergency room (ER) use.

Methods

Subjects

Subjects were drawn from two facilities of an urban health maintenance organization (HMO) with a combined enrollment of over 50,000. Approximately 80 percent of these patients were Black and all had at least one working adult in the family. The median household income for the HMO service area was 6 percent below the overall city median. Patients were eligible for inclusion in the study if they were 18 years of age or younger and had at least one emergency room visit or hospitalization for asthma during the prior four-year period. A total of 253 subjects met eligibility requirements. All eligible patients were enrolled in the study.

Randomization

For randomization purposes, patients were listed in order of their past emergency room use and descending this list patients were allocated randomly using a table of random numbers to either experimental (n = 127) or control (n = 126) groups. The random assignment was balanced so that out of every 10 assignments, five were to the experimental group. The experimental and control groups were virtually identical on several measures as shown in Table 1.

Data Collection

The HMO collected information on hospital days, emergency room visits, and office visits including the related diagnoses during its normal course of business. Once a patient dropped out of the HMO, no further information was available. The enrollment and dropout of patients were monitored by the HMO. Utilization outside the HMO while a member was recorded because virtually all other emergency rooms and hospitals would have billed the HMO for their services.

TABLE 1—Profile of total Group [N = 253]

	Experimental Group	Control Group
Number of Patients	127	126
Mean Age (years)	8.5	9.0
Sex (% male)	61%	65%
ER Use before entry (# visits in 1 year)	1.2	1.2
Months in HMO before entry	20.5	21.2
Percent registered at largest HMO center	48.0	49.2
Percent with one or more hospital days for asthma in prior year†	18.2	23.6

†Percent shown for patients for whom data are available.

Educational Program

The program consisted of two components: classes conducted in group settings for patients and their parents, and telephone instruction by protocol to patients or parents by nurse clinicians to provide additional instruction and reinforcement. The classes and calls covered 23 instructional objectives grouped into four content areas: 1) prevention of asthma attacks, 2) medication management, 3) intervention during asthma attacks, and 4) utilization of health care resources. Instruction dealt with knowledge, skills, and behavior. Four classes were offered to each patient and his or her parents on weekday evenings and Saturday mornings with make-up sessions scheduled periodically to accommodate patients who missed classes. Each class lasted 1.5 hours. Following classes, telephone instruction by protocol to patients and their parents was done at least each season. The calls typically lasted 30 minutes.

The program was offered to all 127 experimental group patients. Of the patients who remained in the HMO for 12 or more months, 38 percent attended one or more classes. Telephone instruction by protocol improved patient contact to 81 percent. The mean number of educational contacts (classes plus calls) was 4.4. The intervention increased the frequency of self-reported mastery on most of the instructional objectives. The post-intervention mastery of specific educational objectives varied from 80 percent of patients for avoidance of over-the-counter drugs, 75 percent of patients for quitting smoking, 35 percent of patients for recognition of

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the early signs of an asthma attack, to 30 percent of patients for using medicines correctly. Detailed descriptions of the program content, instructional techniques, and educational outcomes for these patients have been reported previously.[4,5]

Exposure of control group patients to the intervention was unlikely because the program was performed apart from the primary care providers and the program staff avoided routine contact with those providers. The control group continued to receive their usual medical care.

Results

Of the 253 patients who began the program, 205 remained within the HMO for the first 12 months and 194 remained within the HMO for the entire study period, up to 29 months. Dropouts were evenly distributed between experimental and control groups. Participants and dropouts were compared according to age, sex, and ER use before entry. The differences were small and not statistically significant.

There was no significant difference between experimental and control groups in post-intervention health care utilization for respiratory related illnesses for a one-year period as shown in Table 2. This analysis excludes dropouts and eliminates any seasonal bias, since each patient was enrolled for 12 continuous months. Hospital days and office visits also are compared. Further analysis (not shown in this table) revealed no differences in utilization for non-respiratory illnesses. The results were similar when this analysis was repeated for the total time period of the study with all dropouts included. The probability of a Type II error for a clinically significant difference was calculated and found to be negligible.

Discussion and Conclusions

The program did not achieve its goal of decreasing emergency room visits for children with asthma. This occurred despite careful program design, implementation by clinicians with expertise in pulmonary disease, and an average of $96 expended per patient per year (1984 dollars) for the asthma education.

Other authors have shown other programs to be effective with children with asthma through such outcome measures as reduced emergency room visits, hospital use, sick days, and days lost from school.[6–10] Why have these other interventions been successful while this program was not? The inability of this intervention to demonstrate a reduction in ER utilization does not appear to be due to any of the following methodological problems: randomization problems, dropouts, exposure of control patients to intervention, omission of traditional program content, or accuracy of outcome measures.

TABLE 2—Respiratory-Related Utilization for Patients Enrolled Entire 1st 12 Months [N = 205]

	Experimental Group	Control Group
Number of Patients	101	104
Patient months enrolled	1212	1248
Number of ER visits	55	39
Distribution of ER visits		
0	77	86
1–2	17	13
3 or more	7	5
Hospital Days		
mean	0.27	0.22
standard deviation	1.34	0.74
Office Visits		
mean	1.63	1.86
standard deviation	2.28	4.24

The program probably failed for two sets of reasons: failures of implementation, and failures of program effectiveness. The socioeconomic, educational, and racial profile of the patients probably contributed to problems with both program implementation and program effectiveness. Programs need to be tailored to their target populations. Furthermore, the program was difficult to implement because it was offered to all eligible children with asthma, not just volunteers. This undoubtedly accounted for some of the difficulties in obtaining participation in classes by patients and parents. Implementation was further hindered by the enrollment of many patients with moderate asthma who were probably less motivated and less likely to benefit than patients with more severe disease.[11]

Problems with program effectiveness took several forms. The behavioral component of this program was less intensive than that of other successful programs.[6] It has been demonstrated for asthma[12] and other diseases[13–15] that increases in patient knowledge alone, without instruction on how to change behavior, will not improve patient compliance or health outcomes. Although the importance of behavioral change was discussed with patients in this program, specific behavior modification techniques such as reinforcement and contingency contracting were not utilized. Secondly, less mastery of the educational material was attained by the patients than anticipated, even though considerable resources were used in the program. Less mastery occurred partially because class attendance was less than expected. Furthermore, although telephone instruction alone improved the rate of contact and increased the level of mastery of certain educational objectives, it did not lead to the level of mastery desired.[5] Thirdly, those instructional sessions with parents alone may not have been as effective as those sessions including the children.

Although this study did not show any impact on health care utilization, it is possible that the intervention had a positive impact on functional measures such as days lost from school or sick days. These measures should be included in future research.

This study identifies problems that must be addressed during the widespread implementation and funding of patient education for asthmatic patients. To the inexperienced, patient education seems deceptively simple. However, unless the complexity of patient education is appreciated, a program may fail and may have unintended outcomes. The findings of this study suggest that programs should: 1) include a strong behavioral component, 2) be targeted at key groups who will actively participate and benefit, 3) be monitored to assure that intermediate goals are reached, and 4) be validated for specific socioeconomic groups before implementation.

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New NCHS Report Gives Trends in Low Birthweight

The National Center for Health Statistics recently released its latest findings in vital and health statistics. The report—*Trends in Low Birthweight: United States, 1975–85*—analyzes national and regional trends in rates of low, moderately low, and very low birthweight, and examines the relative risk by maternal characteristics such as age, race, education, and marital status. Some of the findings:

- Low birthweight (LBW) which had been on the decline for the past two decades, increased 2 percent between 1985–87.
- Approximately 262,000 LBW babies were born in 1987 (69 per 1000 live births).
- Rates of LBW and very low birthweight (VLBW) are substantially higher for Black than for White births. In 1987, Blacks were almost three times as likely as Whites to have a VLBW infant and more than twice as likely to have an LBW baby.
- Rates of low, very low, and moderately low birthweight (MLBW) are lowest for mothers in their late twenties and rise as maternal age increases or decreases.
- All LBW rates are substantially higher for young women having a second or higher order birth than for first-time mothers, reflecting the close spacing of many second and subsequent births.
- More than half of all LBW and MLBW babies and 90 percent of VLBW babies are born before 37 weeks of gestation (preterm). Of LBW babies who were full term, only 3 percent weighed less than 1500 grams, but 27 percent of those born preterm weighed this little. For both preterm and full-term LBW babies, the proportion weighing less than 1500 grams is higher for Black than for White babies.
- For all characteristics examined, rates of LBW, MLBW, and VLBW are substantially higher for Black than for White births. The racial differential is most pronounced for VLBW. In 1985, the VLBW rate was 2.8 times as high for Black as for White babies, and rates of LBW and MLBW were 2.1–2.2 times as high for Blacks as for White births.

The NCHS report, by Selma Taffel, is: *Trends in Low Birthweight: United States, 1975–85*, Vital Health Statistics Series 21, No. 48, DHHS Pub. No. (PHS) 89-1926. For further information about this report, or for a list of titles of reports published in the Vital and Health Statistics series, contact: Scientific and Technical Information Branch, NCHS, Hyattsville, MD 20782, tel: (303) 436-8500.

Post-Hospital Support Program for the Frail Elderly and Their Caregivers: A Quasi-Experimental Evaluation

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Abstract: This paper evaluates the Post-Hospital Support Program for the frail elderly and their caregivers using a quasi-experimental design. The program goals were to reduce stress in the caregivers, improve functioning and reduce mortality in patients, and reduce health service utilization in patients. Subjects were patients at the Johns Hopkins Hospital in Baltimore, Maryland, age 65 or over, who were returning home to the care of a non-paid caregiver and who had extensive post-hospital care needs which were expected to continue for at least one year. The 93 comparison group patient/caregiver pairs were discharged between May 15, 1983 and May 14, 1984. The 98 treatment group pairs were discharged between May 15, 1984 and May 14, 1985. Interviews were conducted with patients and caregivers at 1, 3, 6, 9, and 12 months after discharge. Results showed a slight reduction in caregiver stress and a substantial reduction in hospital days used by the treatment group. When confounding and history effects are taken into consideration, an average difference of 6.5 days per patient remains. The data also suggest that the treatment program may have postponed some deaths and nursing home placements. The results suggest that support services for the frail elderly and their caregivers can be cost efficient by reducing hospital length-of-stay. Savings were estimated at $4,585 per patient per year in this study. However, further work is needed to design programs which more effectively reduce caregiver stress. (*Am J Public Health* 1990; 80:39–46.)

Introduction

The elderly population is expanding rapidly and, at the same time, growing proportionately older.[1] This means more and more elderly will be in need of some type of long-term care.[2] The cost for long-term care, especially for institutional care, has been growing considerably faster than the rate of inflation.[3] The $33 billion average annual expenditure (1986–90) for nursing home care is expected to expand to over $120 billion by the year 2020.[4] Nor are the projected increases limited to nursing home care. For every frail elderly person now in a nursing home, there are two equally frail elderly living in the community.[5] Providing home care services for this population is increasingly recognized as an important goal while, at the same time, the potentially explosive costs are greatly feared. A key to achieving good care for the community-based frail elderly population, and avoiding costly institutional care, is the family caregiver, who currently provides 80–90 percent of in-home care in the United States. The Informal Caregivers Survey, conducted as a part of the 1982 Long-Term Care Survey, showed that the typical caregiver of the frail elderly is a 57-year-old married female who is in poor health;[6] substantial proportions experience emotional distress and social isolation.[7,8] A number of programs designed to support these family caregivers have sprung up around the country, but most of them offer no more than a brief "support group"; these programs have not been systematically evaluated.[9] We do not yet know whether a program designed to provide support for the family caregiver can be effective in reducing the negative effects of caregiving, and whether this strategy will lead to a reduction in the use of institutional care. This paper describes a study designed to determine the effectiveness of a support program for caregivers of the frail elderly following hospital discharge. The program goals were to reduce stress in the caregivers, improve functioning in patients, reduce mortality in patients, and reduce cost by reducing health service utilization.

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Method

Treatment Program

The Post-Hospital Support Program provided both medical and social services to the frail elderly and their families for one year following hospitalization. A coordinated approach was provided by a project team made up of a nurse and a social worker. Services included assessment, case management, skilled nursing, counseling, referrals, respite, education, support group, medical back-up, and on-call help. All cases received a minimum of one nurse and one social work visit per month. However, the amount of service depended on need. The service differed from what is usually available in the Baltimore area not only in the amount of service, but also in its coordinated medical-social approach. Focus was on the caregiver/patient configuration and not primarily on the patient; service was closely coordinated with the hospital inpatient and outpatient services; and service extended for one year post-discharge, regardless of the health status of the patient. Additional services were available, such as respite, some "gap filling" money (used primarily for incontinence supplies), and a support group. A more detailed description of the treatment program is provided in Appendix A.

Subjects

Subjects were inpatients at the Johns Hopkins Hospital who gave informed consent and who met the following eligibility criteria:

- Post-hospital care would be provided by a non-paid caregiver.
- Post-hospital needs were comparable to those of patients in "skilled" nursing facilities. (A measure, used by the State of New York—the DSM-1[10]—to determine eligibility for "skilled" care, was used to determine level of need. Patients with scores of 180 or more were eligible for the study.)
- Post-hospital care needs were chronic (expected to last at least one year) and long-lasting (patient expected to live at least one year).

- Patient residence was within 20 miles of the Hospital.

Design and Procedures

The design was quasi-experimental.[11] Patients who were discharged from the hospital between May 15, 1983 and May 14, 1984 constituted the comparison group; 93 patients and caregivers who met the eligibility criteria and agreed to participate during the first year of the study formed the comparison group. Patients discharged between May 15, 1984 and May 14, 1985 constituted the treatment group; 98 cases who met the eligibility criteria and agreed to participate in the second year formed the treatment group. The refusal rate was about 10 percent for both groups. Both patients and caregivers were interviewed within one month of discharge, and again at 3, 6, 9, and 12 months post-discharge. At the final interview, there were 50 patient/caregiver pairs in the comparison group and 62 patient/caregiver pairs in the treatment group. The interviews, which were identical for the two groups, were conducted by trained interviewers. Interviewers attended weekly supervisory sessions. Although there was no way to blind the interviewers, they were not aware of the pattern of the design, and did not seem to differentiate between comparison and treatment groups.

Measurement

Caregiver Stress—Caregiver stress was measured in three ways: The 27-item General Health Questionnaire (sensitivity = 88 percent, specificity = 84 percent)[12] was used to measure overall stress level. We also used a symptom checklist for measuring physical health (alpha = 82, inter-rater reliability = .88).[13] A measure of the extent to which caregiving was having a negative impact on the social functioning of the caregiver (Negative Impact of Caregivers Scale) was also included.[14] Finally, caregiver utilization of health services was measured with caregiver report.

Patient Functioning—Patient functioning was measured with the Katz Activities of Daily Living Scale (ADL)[15] (coefficient of reproducibility = .94–.98), three items* from the Instrumental Activities of Daily Living Scale (IADL)[16] (reproductibility = .94) and the Mental Status Questionnaire (MSQ)[17] (alpha = .84, test-retest reliability = .8). The ADL and IADL were based on caregiver report. The MSQ was given directly to the patient.

Patient Utilization of Services—At the 3, 6, 9 and 12 month interviews, utilization of a wide range of health services was determined (by caregiver report) including: physician and emergency room visits, nursing home days, use of home health nurse, home health aide, respite, transportation, social work, and other in-home services. In addition, hospital records were examined to determine hospital days used by the patient.

Data Analysis

Caregiver stress and patient functioning were measured with proportional change scores in each of the four time periods (0–3 months, 3–6 months, 6–9 months, and 9–12 months). That is, the time one score was subtracted from the time two score. This difference was then divided by the maximum change possible given the time one score. Thus if a patient scored "8" on the Mental Status Questionnaire, the maximum positive change would have been "2" because the maximum score on the scale is "10". If the change was negative, it was divided by the maximum negative change possible—in this case "−8". The resulting score ranges from −1 to +1. A score of "0" represents no change. "+.50" represents an improvement of 50% of the possible improvement, given the time-one score. The use of a proportional change score compensates for different amounts of possible change based on any uneven distribution of initial scores. All cases who were available at the two time points were included in the analysis. Thus a patient who died seven months after discharge would be included in the 0–3 and 3–6 month measures but not in the 6–9 or 9–12 month ones. Thus the number of cases decreases over the four time periods. A 0–12 month figure is also included for those who remained in the program for 12 months.

Mortality data are analyzed using the Mantel-Haenszel odds ratio for each of the four time periods and the total 12-month period. A Kaplan-Meier survival curve is also presented. In analyzing service utilization, descriptive measures (e.g., percents, means) are used for each of the time periods. For hospital days and outpatient visits, means are calculated for the entire group and again for the patients who stayed in the study for 12 months. This is to eliminate the effect of any differential mortality or institutional placement rates in the two groups.

Results

Sample Characteristics

The 191 patients in the study were predominantly female (61 percent) Black (76 percent) widowed (53 percent). The mean age was 76 years. Mean annual income was $3,880. One-third were on Medicaid; 63 percent had less than eight years of education. The mean DMS-1 score for the study population was 320. An example of a patient with this score would be Mr. L., "a 71-year-old man with coronary artery disease, diabetes mellitus, hypertension, permanent pacemaker, and glaucoma. He was admitted to the study after being hospitalized for left femoral peroneal bypass. He had extensive wounds extending from the groin to the achilles area requiring daily soaks, dressings and monitoring." In spite of this level of dependency, only 10 percent had poor prognosis; 52 percent had "guarded" or "fair" prognosis, and 38 percent had a good prognosis. The caregivers, 80 percent of whom were female, had a mean age of 53; 30 percent were spouses of the patient, and 35 percent were daughters; 73 percent had been caring for the patient before the hospital episode in which the patient was identified for the study.

Comparison of Treatment and Control Groups

The two groups were similar on most patient and caregiver demographic characteristics and on medical and functional status (Tables 1 & 2), with two exceptions. In the treatment group, more caregivers were new to the task, and fewer patients had a "good" prognosis.

Comparison of Services Received

In the Baltimore area, a variety of services were available to the comparison group during the course of the study. For example, home health service (limited to the period of skilled nursing need) including visiting nurse, home health aide, and social work services were widely available (Table 3). There was also a case management service (continuation of the program evaluated in the National Channeling Demonstration).[18] Transportation was available for Medicaid patients only. A comparison of the rates of service utilization

*The items were: handling the telephone, handling own medication, and handling own finances.

TABLE 1—Demographic Characteristics of Patients and Caregivers in the Comparison and Treatment Groups

Demographic Characteristics of Patients	Comparison Group (N = 93)	Treatment Group (N = 98)
% female	60.2	61.2
% Black	78.5	74.5
% widowed	54.8	52.0
% <8th grade education	64.8	61.3
% Protestant religion	76.3	74.5
Mean age (years)	76.5	76.3
Mean income*	$5,436.74	$5,747.88
% lives alone	9.8	9.3
% on Medicaid	37.4	28.4
Demographic Characteristics of Caregivers		
% female	82.8	77.6
% of caregivers who are the spouse of patient	28.6	31.9
% of cases where caregiver provided care prior to the index hospitalization	60.4	83.7†

*Adjusted for inflation rate between 1983 and 1984 of 4.3 percent.
†Difference (60.4–83.7) = 23.3. 95% confidence intervals of the difference −35, −12.

TABLE 2—Medical and Funcational Characteristics of Patients in the Comparison and Treatment Groups

	Comparison Group (N = 93)	Treatment Group (N = 98)
Mean DSM-1, score	305.7	333.4
% of "Good" prognosis	46.2	29.6*
% Taking four or more medications	56.1	55.1
Mean ADL score on initial interview	5.0	4.3
Mean MSQ score on initial interview	5.5	6.1
Mean IADL score on initial interview	1.4	1.2
Mean # hospital days in year preceding	7.2	9.5

*Difference (46.2–29.6) = 16.6. 95% confidence intervals 3.6, 29.6.

in the comparison and treatment groups by caregiver report shows that, in all time periods, more patient/caregiver pairs in the treatment group received visiting nurse service and social work service, home health aids and transportation from the third to the twelfth month post-discharge, but not more respite care.

Impact on Caregiver Stress

A comparison of the proportional difference scores on caregiver stress for the treatment and comparison groups is shown in Table 4. Most of the proportional difference scores are negative, indicating a reduction in stress over time. The exception is on the Negative Impact on Caregiving Scale, which shows a sharp reduction in the 0–3 month period followed by a sharp increase in the 9–12 month period. Overall, the differences between the two groups are very small. The confidence interval includes zero for all but two comparisons: Number of Caregiver Illnesses (6–9 months) and Negative Impact of Caregiving (0–3 months). There is a consistent pattern of greater reduction in the General Health Questionnaire scores (indicating less mental disorder) for

TABLE 3—In-Home Service Utilization by Comparison and Treatment Groups at 3, 6, 9 and 12 Months Post Discharge

	Comparison Group		Treatment Group		% Difference	95% Confidence Interval
	N	% (a)	N	% (b)	(a − b)	
Visiting Nurse						
0–3 months	66	51	81	96	−45	−31, −59
3–6 months	51	28	73	95	−67	−50, −84
6–9 months	50	28	69	97	−69	−52, −86
9–12 months	50	16	62	95	−79	−61, −97
Home Health Aide						
0–3 months	66	41	81	48	−7	+10, −24
3–6 months	51	25	73	45	−20	−3, −37
6–9 months	50	25	69	46	−21	−3, −39
9–12 months	50	24	62	42	−18	0, −36
Social Worker						
0–3 months	66	23	81	93	−70	−54, −86
3–6 months	51	12	73	96	−84	−67, −100
6–9 months	50	11	69	91	−80	−62, −98
9–12 months	50	14	62	81	−67	−48, −86
Transportation						
0–3 months	66	30	81	40	−10	+6, −26
3–6 months	51	23	73	43	−20	−3, −37
6–9 months	50	26	69	49	−23	−5, −41
9–12 months	50	28	62	48	−20	−1, −39
Respite Care						
0–3 months	66	14	81	12	+2	+13, −9
3–6 months	51	12	73	15	−3	+96, −15
6–9 months	50	9	69	6	+3	+13, −7
9–12 months	50	14	62	6	+8	+19, −36

treatment group caregivers, but none is large enough to inspire confidence.

No clear relations were found between prognosis and the caregiver stress variables. However, previous caregiving experience was found to be related to the proportional difference in caregiver illness at 6–9 months ($r^2 = .06$). (Those with prior caregiving had greater reductions in numbers of illnesses.) A pooled proportional difference score was calculated, based on the weighted scores of those caregivers who did and did not have prior experience. The pooled difference score was +.13, CI +.04, +.22. There is some evidence overall for a small treatment effect on caregiver stress.

Impact on Patient Functioning

A comparison of the proportional difference scores on patient functioning for the treatment and comparison groups is shown in Table 5. There is no evidence that the Post-Hospital Support Program had any effect—positive or negative—on patient functioning. Patients in both groups showed early improvement in Activities of Daily Living (ADL), followed by a leveling off. There was very little change in the Instrumental Activities of Daily Living (IADL) scores of either group. Both groups showed some improvement in mental status in the first part of the year, with a leveling off in the 9–12 month period.

Mortality

One year after hospital discharge, 27 patients in the treatment group had died (27.5 percent), compared to 28 in the comparison group (30 percent). While there is no evidence that the "Post-hospital Support Program" decreased

TABLE 4—Comparison of Treatment and Comparison Groups on Mean Proportional Difference in Caregiver Stress Variables at Four Time Periods*

	Comparison Group		Treatment Group		Difference	95% Confidence Intervals of Difference
	a	(N)	b	(N)	a − b	
General Health Questionnaire						
0–3 months	−.23	(68)	−.32	(82)	+.09	−.06, +.24
3–6 months	−.13	(55)	−.17	(74)	+.04	−.10, +.18
6–9 months	−.25	(51)	−.28	(68)	+.03	−.14, +.20
9–12 months	−.11	(48)	−.15	(61)	+.04	−.11, +.19
Total: 0–12 months	−.30	(48)	−.35	(61)	+.05	−.14, +.24
# Caregiver Illnesses†						
0–3 months	−.22	(68)	−.25	(82)	+.03	−.09, +.14
3–6 months	−.13	(55)	−.13	(74)	+.00	−.10, +.10
6–9 months	−.09	(51)	−.21	(69)	+.13	+.04, +.22
9–12 months	−.21	(48)	−.15	(62)	−.06	−.19, +.07
Total: 0–12 months	−.26	(48)	−.20	(62)	−.06	−.20, +.08
Negative Impact of Caregiving						
0–3 months	−.19	(66)	−.31	(82)	+.12	+.03, +.19
3–6 months	−.03	(55)	+.03	(74)	−.05	−.11, +.01
6–9 months	−.06	(49)	−.03	(69)	−.03	−.12, +.06
9–12 months	+.24	(47)	+.28	(61)	−.04	−.11, +.03
Total: 0–12 months	+.00	(48)	+.04	(61)	−.04	−.12, +.04

*On all of the caregiver stress variables, a high score indicates an unfavorable outcome.
†Adjusted for previous caregiving experience.

TABLE 5—Comparison of Treatment and Comparison Groups on Mean Proportional Difference in Functional Status at Four Time Periods*

	Comparison Group		Treatment Group		Difference	95% Confidence Intervals of Difference
	a	(N)	b	(N)	a − b	
Activities of Daily Living (ADL)						
0–3 months	−.13	(66)	−.18	(82)	+.05	−.09, +.19
3–6 months	+.01	(55)	−.05	(74)	+.06	−.06, +.18
6–9 months	+.05	(50)	+.03	(69)	+.02	−.11, +.15
9–12 months	−.04	(47)	−.06	(62)	−.02	−.13, +.09
Total: 0–12 months	−.12	(48)	−.20	(62)	−.08	−.26, +.10
Instrumental Activities of Daily Living (ADL)						
0–3 months	−.02	(65)	−.05	(82)	−.07	−.23, +.09
3–6 months	+.08	(54)	.00	(73)	+.08	−.19, +.35
6–9 months	−.13	(50)	−.04	(69)	−.09	−.23, +.05
9–12 months	+.02	(47)	+.03	(62)	+.01	−.18, +.16
Total: 0–12 months	−.05	(48)	+.10	(62)	−.15	−.37, +.07
Mental Status (MSQ)						
0–3 months	+.13	(69)	+.04	(82)	+.09	−.05, +.23
3–6 months	+.25	(57)	+.20	(74)	+.05	−.11, +.21
6–9 months	+.08	(53)	+.09	(69)	−.01	−.17, +.15
9–12 months	.00	(50)	+.02	(62)	−.02	−.17, +.13
Total: 0–12 months	+.04	(50)	+.08	(62)	−.04	−.22, +.14

*On the ADL scale, a high score indicates that more assistance is needed with activities of daily living, hence, an undesirable outcome. On the IADL and MSQ indices, a higher score indicates better functioning.

mortality, there is some evidence that the program may have kept patients alive longer than those in the comparison group.

The Mantel-Haenszel odds ratios for four time periods show that, in the first nine months of the program, the odds of surviving were higher in the treatment group (OR 1.25 to 1.46). In the last three months of the 12-month follow-up period, the treatment group had slightly lower odds of surviving than did the comparison group (OR = .92). The

overall OR is 1.16. These differences are small, and the confidence intervals include an odds ratio of 0 in all time periods. A Kaplan-Meier survival curve (Figure 1) shows this pattern graphically. Thus, while we cannot conclude that the treatment program reduced mortality, the data suggest that some deaths may have been postponed.

Utilization of Health Services

Hospital Days—On the basis of the hospital charts, the patients in the study used an average of 33.9 hospital days during the study. This includes the index hospital episode where the patient was picked up for the study, which averaged 18.4 days. The index hospitalization is included because the PHS (post-hospital support) program began to impact on the situation well before discharge as PHS staff worked with hospital floor staff to plan for the discharge. Thus the program may well have impacted on the length of this initial hospitalization. Table 6 shows that there was a substantial difference in the number of days patients in the treatment and comparison groups were hospitalized. Patients in the comparison group averaged 38.5 days in the hospital (of which 21 days were for the index hospitalization). Patients in the treatment group were hospitalized an average of 29.1 days (of which 16 days were for the index hospitalization). Thus the patients in the Post-Hospital Support Program had fewer hospital days, both in the initial hospitalization and in the subsequent year. Because the mortality experience of the two groups was somewhat different, these averages were also calculated, excluding all patients who did not complete 12 months in the study. These results (line 4, Table 6) serve to strengthen the conclusion.

Because confounding was a possibility in this study, the potentially confounding variables ("good" prognosis and prior caregiver experience) were correlated with hospital days. Surprisingly, prognosis was not related to hospital days. However, prior caregiver experience showed a possible relationship ($r^2 = -.04$), suggesting that patients with caregivers who had been caring for them prior to the index hospitalization had fewer hospital days. Patients of caregivers with prior experience averaged 31.5 hospital days, compared to 39.9 hospital days for new caregivers. The pooled difference score in this case was 8.2 days CI +.7, +15, suggesting that the difference was not due to confounding.

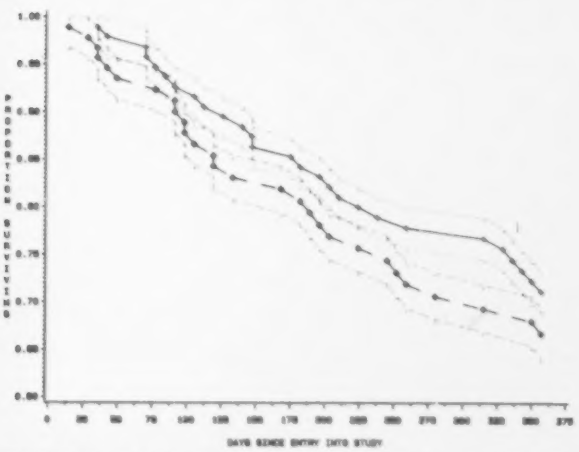

FIGURE 1—Survival Curves (with 95% confidence intervals) for Patients in the Treatment (PHS) and Comparison Groups

Treatment Group
95% confidence intervals for Treatment Group
Comparison Group
95% confidence intervals for Comparison Group

We conclude that the patients in the Post-Hospital Support Program spent fewer days in the hospital than did patients in the comparison group. The total difference averaged about eight days. About half of this savings was due to earlier discharges following the initial hospitalization. It seems probable that hospital staff were comfortable discharging patients with high levels of need earlier, knowing that a comprehensive support program would be available. However, the savings also continued in the year following discharge. The fact that there was no meaningful difference between the groups in the average number of hospitalizations (comparison group $\bar{X} = .66$, treatment group $\bar{X} = .78$) suggests that the same factor was operating in subsequent hospitalizations. Thus the treatment program probably did not prevent hospitalizations; it shortened them.

Table 6 also shows that the patients in the treatment program used fewer nursing home days than did those in the comparison group. However, the confidence interval is too wide to conclude that this difference was not due to chance. Since the proportion of patients who entered nursing homes was about the same in the two groups (12 percent in the comparison group, 10 percent in the treatment group), the difference in nursing home use is probably due to some postponement in entry. In fact, the mean number of nursing home days for those patients in the comparison group who entered nursing homes was 146, compared to only 86 for those who entered nursing homes from the treatment group. This represents an average postponement of about two months.

Not surprisingly, the treatment patients who were in the study at 12 months had slightly more outpatient physician visits than did those in the comparison group. This probably reflects some shifting of services from inpatient to outpatient.

Cost Implications

The Post-Hospital Support Program aimed to reduce costs by reducing unnecessary health service utilization. The figures presented in Table 6 have important economic implications. In order to estimate costs under the two conditions (treatment and comparison) average costs at the Johns Hopkins Hospital (or in the Baltimore area) during the study period were gathered. At the Johns Hopkins Hospital, the average cost of a hospital day in 1985 for patients over 65 years was $1,100. It represents an average of the more expensive first part of a hospitalization and the less expensive later part. This figure includes room, board, and medical services. The average nursing home ran about $65 per day. An outpatient visit averaged $50. Applying these figures to the data for Table 6, a 6.5 day reduction in hospital days means an average saving of $7,150. Eight less nursing home days represents a saving of $520. Two more outpatient visits means an extra cost of $100 per patient. This totals to a savings of $7,670 per patient per year.

This potential saving must be balanced against the program costs. The Post-Hospital Support Program actual costs averaged $4,584.67 per patient. While the cost of similar services to the comparison group are not as readily available, they can be estimated. Using the percents receiving visiting nurse services, we estimate 1.23 home health episodes per comparison group patient. Since an average home health episode at the Johns Hopkins Home Health Agency costs $1,116, the cost of home health services can be estimated at

TABLE 6—Utilization of Health Services by Patients in the Comparison and Treatment Groups

	Comparison Group Mean (std dev.) (a)	Treatment Group Mean (std dev.) (b)	Difference (a − b)	95% Confidence Interval of the Difference
Hospital Days				
Mean days in index hospitalization	21 (16.6)	16 (14.6)	+5	+.4, +9.6
Mean days in hospital from discharge to exit from study	17 (22.6)	12 (19.8)	+5	−1.4, +11.4
Total mean days in hospital (All cases)	39 (29.9)	29 (25.5)	+10	+1.6, +18.4
Total mean days in hospital (12 months cases only)	38 (30.5)	26 (25.3)	+12	+1, +23
Nursing Home Days				
Mean number of nursing home days	17 (59.3)	9 (32.9)	+8	−.56, +21.6
Outpatient Visits				
Mean number of outpatient clinic visits	5 (15.2)	7 (12.2)	−2	−1.9, +5.9
Mean number of clinic visits (12 month cases only)	8 (20.0)	10 (14.3)	−2	−3, −1

$1,372.68 per comparison group patient. Since we cannot calculate costs for transportation (received by 23–30 percent of the comparison group patients) or respite (received by 9–14 percent of comparison group patients), we will round the figure upwards to $1,500 to roughly account for these additional costs. Using this figure, the Post-Hospital Support Program cost about $3,085 more per patient per year. If we compare this to the $7,670 saved in the lower health service utilization, we see that there remains a cost saving of $4,585 per patient per year after additional program costs are taken into account.

Discussion

The Post-Hospital Support Program for the Frail Elderly and Their Caregivers was only partially successful in meeting its goals. There was evidence of some reduction in caregiver stress, but it was not impressive. There was no evidence of improved patient functioning. There was, however, a suggestion that the program may have postponed both deaths and nursing home placements. There was also a clear pattern of evidence suggesting that hospitalizations were shortened for treatment group patients, resulting in substantial cost reductions. These went well beyond the additional program costs.

Because our design did not control the threat of history to internal validity, we cannot overlook the fact that there were substantial changes taking place in the health care system during the course of the study. Because of the introduction of DRGs (diagnostic related groups) by the Medicare Program, hospital stays were being reduced for the elderly at this time.[19] It is possible that the reduction we observed was due to the difference between the years of observation (1983–84 vs 1984–85) rather than to the effect of the treatment program. However, the average reduction in hospital length of stay for patients 65 years of age and older between 1983 and 1985 was one day.† Since, in our study, patients had an average of 1.73 hospitalizations over the study period, we would have expected a reduction of 1.73 days on the basis of history alone. Since the difference we found was almost five times this large, it is highly unlikely that the prospective pricing system could have accounted for the differences found. Also because the 1.73 days related to all patients over age 65, and the patients in our study were a very frail subset of the over 65 population, the 1.73 day figure may well be high. Even if we subtract the 1.73 expected days shorter for the treatment group, we still find at least 6.5 days difference.

Because of the quasi-experimental design and the turbulent hospital environment which existed at the time of the study, the results should be subjected to more rigorous tests before firm conclusions are drawn.

The program's lack of success in reducing caregiver stress suggests a need to reexamine both the program offered and the assumptions behind it. Low attendance at caregiver support groups may have been due to program failure (e.g., lack of skilled leadership, of adequate transportation, or respite) or to a lack of interest in this type of service by caregivers. Recent results from respite programs for caregivers show similar findings,[20–22] suggesting that perhaps the whole notion of caregiver support needs to be reexamined.

In the Post-Hospital Support Program, there were frequent conflicts between the staff and patients/caregivers. These were usually because the staff wanted the medical regimen to be followed, or wanted a higher standard of

†Personal communication. Hyattsville, MD: Scientific and Technical Information Branch, National Center for Health Statistics, Nov. 1, 1987.

personal care to be provided, and these issues may not have been important goals for patients or caregivers. Also, when the interest of patient and caregiver conflicted, the staff tended to become advocates for the patient in spite of program goals. At staff meetings, staff frequently expressed frustration with caregivers who were not doing a "good-enough" job. It is possible that frequent visiting by this staff may have been more stress-producing than stress-reducing for caregivers. Another possibility is that the amount of support available may have been too small considering the nature of the task. One social worker told how she had spoken with a caregiver who complained that she was "so tired!" The social worker worked for several weeks to arrange help: some day care, home health aide and increased family help. When she called back to see how things were going, the caregiver again complained that she was "so tired!" Perhaps we need to examine in more depth the nature of the caregiving tasks, the interactions of patient, caregiver and agency,[23] and the psychology of mid-life women. This type of analysis may lead to the development of totally different types of "support" services.

A final note on caregiving. The current study underestimated the complexity of the caregiving situation. For example, the design designated one person as a "caregiver" and one as a "patient". In fact, a number of patients had multiple caregivers—sometimes sharing responsibilities, and sometimes caring for the patient in a "serial" pattern. In some situations, it was difficult to distinguish "caregiver" and "patient" as the year progressed. When two ill spouses care for each other, people shifted in and out of these roles as their illness waxed and waned. An index, such as the Negative Impact of Caregiving, was hardly appropriate when the designated caregiver was now in the patient role. The study results also suggest that the amount of previous caregiving experience was a critical variable. Future support programs would do well to consider what type of caregiver their services are aimed at. Programs for new caregivers might need to be quite different than those for long-term providers of care.

Although this was not the program's primary focus, the reduction in health service utilization suggested by the results is intriguing. It raises the possibility that the key to reducing hospital costs lies not in what happens in the hospital itself (e.g., discharge planning) but in what happens in the community. By strengthening the caregiver, the Post-Hospital Support Program may have allowed caregivers to take on even more responsibility. This may explain why stress levels were not reduced further.

The results are promising in that they suggest that programs designed to support the frail elderly population and their caregivers in the community can be cost efficient by reducing levels of health service utilization. However, further work is needed to design programs which are also beneficial to both patients and caregivers.

ACKNOWLEDGMENTS

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APPENDIX A

Description of the Treatment Program

Treatment began in the hospital with assessments by both the Project nurse and social worker. At that point, an initial service plan would be established and caregiver education would begin. Because the initial adjustment to home care was often difficult, and patients were often very sick at this point, many patients needed frequent nursing visits in the weeks following discharge. If dressing changes were involved, up to three nursing visits a day were provided. Patients with complex medication schedules needed close monitoring and nurses helped caregivers to develop systems to simplify regimens and watched carefully to see the effects. Often they spent many hours communicating back and forth with the physicians until an appropriate medication schedule was established. If there were family problems, the social worker would become involved, helping families to develop caregiving patterns which they would manage. This often meant adding outside support. The Post-Hospital Support Program (PHS) made funds available for caregivers to hire helpers for personal care, monitoring, day care, and respite. The number of hours needed, and the supervision of outside help would be negotiated with the family, and an initial contract signed. Also, in rare cases, PHS would cover the cost of environmental modifications (e.g., bars, ramps, stair railings). Physical therapy was also provided if the staff thought it would be helpful, and it could not be covered by an outside payor.

After the initial adjustment period, each case received at least one nurse and one social work visit per month. In many cases, stable periods where only minimal assessment and monitoring were needed alternated with crisis periods, when very intensive levels of outside support were needed. The average was four nurse visits per month, and the maximum was a case which received 198 nursing visits in a year. The nurses carried a maximum of 20 cases, and the social workers 30 cases. If rehospitalization was needed, the staff would follow the patient's progress and prepare another plan for post-discharge care. Support group sessions were offered about once a month. Each session had a specific topic (e.g., "dementia", "handling caregiver stress") which opened with some prepared remarks by staff or invited speakers and ended with discussion among the caregivers. Food (usually lunch) was served, and respite and transportation arrangements were made to facilitate attendance. In spite of this, only a minority of caregivers attended those meetings (usually 10–15 caregivers). Meetings usually began with each of the caregivers introducing themselves and telling their "story". Each introduction was followed by an enthusiastic round of applause and many encouraging comments from the other caregivers (e.g., "Aren't you something!" "Your father is lucky to have you!" "You're doing everything you can.") Although not heavily attended, these groups sessions were real morale boosters for both caregivers and staff. Caregivers loved being treated "special" by the hospital. Staff enjoyed watching the caregivers interact, and they also received very heartfelt appreciation ("I don't know what I would have done without Ms. Gina [nurse]").

Throughout the duration of the program, a weekly staff meeting was held. This was attended by all staff nurses and social workers, a geriatric fellow, a psychogeriatric nurse, the researcher, and the project administrator. Each case was discussed on a periodic basis, but most of the time went to discussing problem cases, usually cases experiencing a medical crisis, or cases where the caregiving situation was deteriorating.

The program sought the best possible situation for each patient. Wherever possible, the staff tried to arrange things so that the patient could remain in the community as long as this was in the patient's best interest. In cases where the caregiving situation was poor, and did not improve in spite of repeated attempts to "prop it up" with education, added help and support, the staff would work actively to remove the patient from the home, even if it meant a nursing home placement. Although this type of situation occurred infrequently, deciding when a patient would be better off outside of the home was one of the most difficult aspects of the program for the staff.

As the end of the one year service period grew near, the staff arranged for a "discharge plan" which would provide ongoing care if needed. Those in need of skilled nursing care would be referred to Home Health Care services. Those who only needed somewhere to call if problems arose would be connected to a case-management service.

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State Funding for AIDS Patient Care Increased 160% in 1989

State government funding for AIDS patient care in fiscal year 1989 totaled nearly $65 million or 26 percent of the total non-Medicaid state funds for AIDS, according to a report released October 17 by the George Washington University AIDS Policy Center. This represents a 160 percent increase from the $25 million appropriated in fiscal year 1988. Based on information from state officials nationwide, patient care will continue to absorb a significant share of state funds as the epidemic grows and more people become infected with the human immunodeficiency virus (HIV). Twenty-six states budgeted funds for at least one type of patient care service, whereas in fiscal year 1988, only nine states budgeted funds for that purpose.

Four states (California, Florida, Massachusetts, New York), allocated more than $5 million in state-only non-Medicaid funds for patient care programs for fiscal year 1989. Eight jurisdictions (District of Columbia, Georgia, Illinois, Louisiana, Maryland, Michigan, Missouri, Pennsylvania) appropriated $1–5 million.

The high cost of Azidothymidine (AZT) has prompted at least 10 states, including such low-prevalence states as Hawaii and Utah, to set aside funds to supplement federal allocations for AZT.

The AIDS Policy Center, a component of GWU's Intergovernmental Health Policy Project, is a legislative and policy research center that provides AIDS information and related research to legislators, policymakers and the media. This report, based on the Center's national survey of state AIDS funds released August 30, 1989, is the first of several analyses of states' allocation of non-Medicaid funds for various program areas. Other reports will examine education/information, testing and counseling, research, surveillance activities, support services and administrative activities. The survey and data analyses were supported by a grant from the American Foundation for AIDS Research (AmFAR). For a copy of this report, contact the AIDS Policy Center, (202) 676-8144.

Epidemiologic Features of Treated End-Stage Renal Disease in a Large Prepaid Health Plan

JUAN D. ORDOÑEZ, MD, MPH, ROBERT A. HIATT, MD, PHD, AND CHARLES P. QUESENBERRY, PHD

Abstract: We examined the incidence of treated end-stage renal disease (ESRD) among the two million members of the Kaiser Permanente Medical Care Program of Northern California from 1973 through 1985. The age-adjusted incidence rates rose from 33.9 to 81.7 per million person-years and showed no signs of stabilization. Rates were higher in men than in women and rose with age. Marked upward trends ($p < .001$) in the rates of treated ESRD associated with diabetic nephropathy and hypertension were noted. (*Am J Public Health* 1990; 80:47–49.)

Introduction

Since the enactment of Medicare legislation on end-stage renal disease (ESRD) in 1973, the cost to the United States government of caring for these patients has far exceeded expectations. The number of persons enrolled increased from 16,000 in 1974 to 99,000 in 1986, while the costs rose from $229 million to over $2.8 billion.[1]

Few reports document the incidence of ESRD in defined US populations, and those available from the Health Care Financing Administration (HCFA) have been limited by discontinuity in the data collection process[2] and lack of compliance with the patient history form.[1] Other regional studies encompass very short periods of time[3] or include no data on trends in incidence.[4–7]

This report describes the population treated for ESRD within the membership of the Kaiser Permanente Medical Care Program (KPMCP) of Northern California from 1973 through 1985. It is an extension of an earlier study derived from the same data sources for the years 1973 through 1977.[7]

Methods

The Kaiser Permanente Health Plan provides comprehensive hospital and inpatient services to subscribers and their dependents. The membership increased from 1.1 million in 1973 to about 1.9 million in 1986 and comprises about 25 percent of the population in the San Francisco Bay Area. The age distribution of the membership remained reasonably unchanged during the period of study. The demographic characteristics of this population in the San Francisco-Oakland Metropolitan Statistical Area are similar to those enumerated by the US census for the same area, except for some underrepresentation of persons in the extremes of the socioeconomic scale.[8] Dialysis and transplantation are generally subcontracted to outside providers.

Patient Identification

Patients were all health plan subscribers with ESRD treated with maintenance hemodialysis, continuous ambulatory peritoneal dialysis, or kidney transplantation from 1973 through 1985. Complete ascertainment was possible because patients requiring ESRD treatment were either treated at a dialysis unit in the Kaiser Permanente San Francisco facility or referred to external facilities through a central health plan office.

Medical Record Review

Medical records of all patients referred for ESRD treatment from 1972 through 1977 were reviewed in 1978,[7] those referred from 1978 through 1981 were reviewed in 1982,[9] and those referred between 1981 and 1985 were reviewed in 1986. In each study, methodology was similar. Medical records in all three periods were reviewed by a trained medical record analyst to obtain: demographic characteristics, renal diagnosis and date, whether a kidney biopsy was performed, date of referral for ESRD treatment, location of the referring facility and, when available, date of first dialysis.

Analysis

The date of first treatment was used to determine the annual age-adjusted incidence of ESRD. Only cases first treated after joining the health plan were included in the calculation of incidence rates. Rates were age-adjusted using the direct method with the 1980 US census used as standard. Time trends in the age-adjusted rates were examined using weighted least squares regression techniques.

Results

From 1973 to 1985, 1,134 patients were treated for ESRD, including 68 (6.0 percent) who were first treated before becoming health plan subscribers. Among the 1,066 diagnosed while subscribers (Table 1), the most common primary diagnoses were glomerulonephritis (25.4 percent), diabetic nephropathy (22.2 percent), primary hypertensive disease (13.4 percent), and polycystic kidney disease (7.9 percent).

The age-adjusted incidence rose in a continuous trend from 33.9 per million members in 1973 to 81.7 per million in 1985 (Figure 1). Although the most marked relative increase in incidence was among persons older than 65 years (40.0 per million in 1973 to 220.4 in 1985), the 15- to 44-year-old age group contributed the largest absolute number of patients (Figure 2).

A higher incidence in men was seen for all age groups after the first decade of life (Figure 3). The incidence in men increased steadily with age, in contrast to the incidence in women, which peaked in the 60- to 69-year-old age group.

Upward trends in the annual age-adjusted, disease-specific incidence were greatest for diabetic nephropathy ($p < .001$) and hypertension ($p < .001$). The incidence of polycystic kidney disease (PCK) increased slightly (trend, $p = .033$) and that of glomerulonephritis showed no evidence of a trend. The incidence of ESRD for all other diagnoses ranged between seven and 15 per million without any clear

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TABLE 1—Mean Age-Adjusted Incidence Rates of Treated ESRD by Diagnosis, KPMCP, 1973–85

Diagnosis	ESRD per Million Person-Years (N)		
	Men	Women	Total
Glomerulonephritis	18.3 (161)	11.0 (110)	14.6 (271)
Diabetic nephropathy	11.6 (124)	10.2 (113)	10.9 (237)
Primary hypertension	11.3 (94)	5.6 (49)	8.3 (143)
Polycystic kidney disease	4.5 (44)	3.9 (40)	4.2 (84)
Pyelonephritis	1.5 (12)	2.8 (26)	2.2 (38)
Obstructive uropathy	2.7 (20)	1.3 (16)	2.0 (36)
Collagen disease	.7 (6)	1.6 (17)	1.2 (23)
Congenital renal disease	1.2 (14)	.6 (7)	1.9 (21)
Drugs	1.2 (6)	1.0 (9)	1.1 (15)
Tumors	1.3 (11)	.4 (5)	.8 (16)
Interstitial disease	.6 (4)	.9 (9)	.7 (13)
Acute renal failure	.5 (5)	.6 (7)	.5 (12)
Other	1.7 (10)	1.6 (14)	1.6 (24)
Unknown	13.0 (99)	3.9 (34)	8.3 (133)
Total	70.0 (610)	45.6 (456)	57.2 (1066)

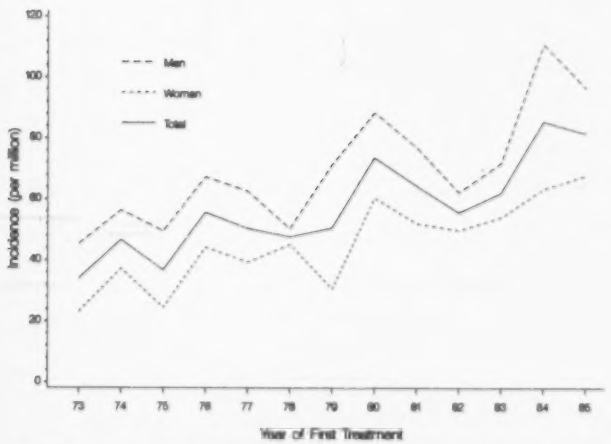

FIGURE 1—Annual Age-Adjusted Incidence of Treated ESRD in the KPMCP, 1973–85

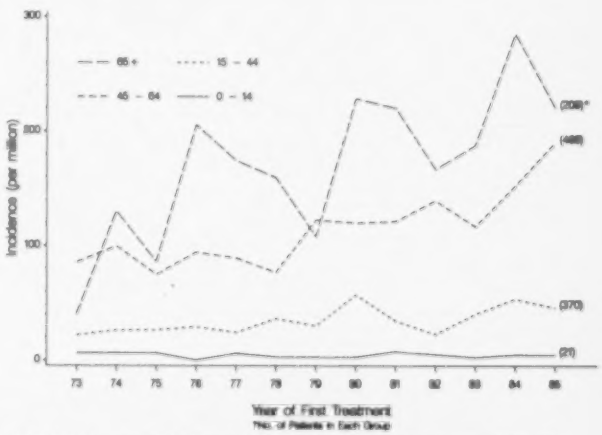

FIGURE 2—Annual Incidence of Treated ESRD by Age Group, 1973–85

trend over time.* The higher incidence of treated ESRD in the oldest age group (>65 years) was primarily associated with diagnoses of glomerulonephritis (23.4 percent) and hypertension (24.4 percent), whereas in the 45- to 64-year-old group, PCK and diabetic nephropathy comprised the largest proportion of patients (38.6 percent).

A small decrease occurred in the proportion of Blacks treated for ESRD in 1982–85 (18 percent), compared with 1973–77 (21 percent) and 1978–81 (24 percent). During the 1973–81 period, glomerulonephritis was the most frequent diagnosis among Whites and Hispanics (29 percent) and among Asians (46 percent) treated for ESRD; hypertension was the most frequent diagnosis among Blacks (26 percent). During the 1982–85 period, diabetic nephropathy became the most frequent diagnosis among all racial groups except Asians (Figure 4).

Discussion

The demographic characteristics of the patients treated for ESRD within the KPMCP in Northern California are similar to those in previous reports,[1,10] based on HCFA data. The incidence of treated ESRD in this health plan population has increased steadily from 1973 to 1985. This observation is compatible with those of Eggers, *et al*,[1] and Weller, *et al*,[11] and suggests that any conclusion that rates have stabilized[10] may be premature.

The most likely reason for the increasing incidence of treated ESRD is the continued liberalization of the criteria for the use of dialysis and transplantation by physicians, which is a consequence of improved survival and decreasing morbidity following treatment of ESRD in recent years.[12,13] Older patients with diseases more likely to be associated with systemic complications, such as diabetes and hypertension, may have benefited the most from the liberalization in criteria, because healthier patients were more likely to have been accepted for treatment in previous years. A possible increase in the incidence of insulin-dependent diabetes may also be contributing to the upward trend in incidence.[14] Finally, the improved medical management of diabetes and hypertension may have decreased competing mortality from coronary artery disease, thus increasing the number of diabetic patients reaching ESRD.

The higher incidence of treated ESRD in older age groups is compatible with the progression of chronic underlying processes that lead to terminal renal failure. The high

*Data available on request to author.

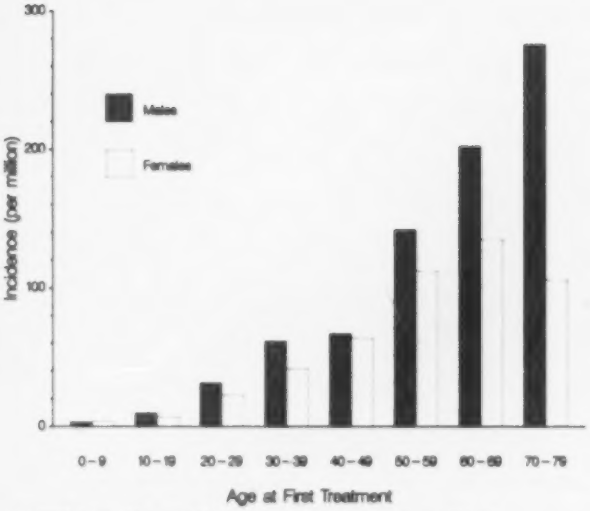

FIGURE 3—Incidence of Treated ESRD by Age and Sex, 1973–85

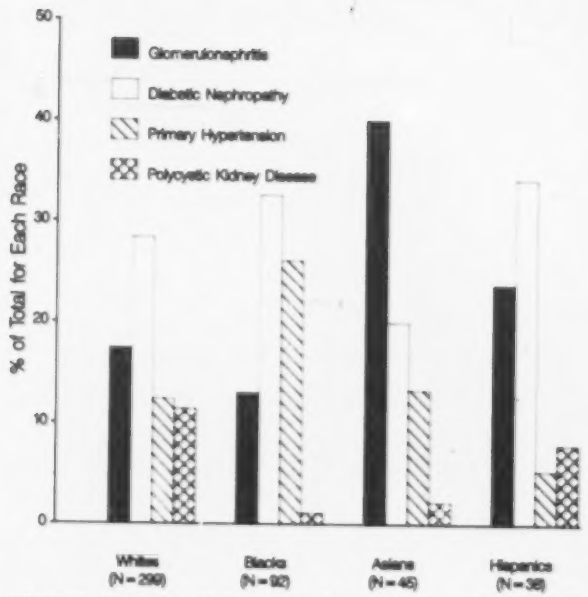

FIGURE 4—Distribution of Primary Renal Diagnosis among Treated ESRD Cases by Race, 1982–85

incidence noted in the group older than 70 years contrasts with national data from HCFA[1] and suggests that age may be less of a deterrent to use of hemodialysis among the KPMCP membership.

We were not able to calculate incidence rates by race because of the lack of data on racial composition of the health plan membership. However, the proportion of Blacks and that of Whites among those treated for ESRD may be decreasing in this Northern California population, while the proportion of Asians (and possibly of Hispanics) is increasing.[15] A higher incidence of diabetes has been reported among Hispanics, compared with other racial groups.[16]

ACKNOWLEDGMENTS

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An Epidemiologic Postmarketing Surveillance Study of Prescription Acne Medications

David P. Facklam, MS, Jacqueline S. Gardner, PhD, Gary L. Neidert, PhD, and Margaret M. Westland, MD, MPH

Abstract: After the Food and Drug Administration approved a topical antibiotic for the treatment of acne, we began a postmarketing surveillance study to measure the frequency of antibiotic-associated colitis and diarrhea in acne patients treated with topical antibiotics and low doses of oral antibiotics. Pharmacists recruited 13,465 patients who presented a prescription for an acne medication, and we obtained detailed information on the use of the medications, perceived acceptability, and the occurrence of new health events from 6,453 with computer-assisted telephone interviews. Three cases (<0.1%) of antibiotic-associated diarrhea and one case (<0.1%) of antibiotic-associated colitis were confirmed. We conclude that this methodology can provide further information about the safety of a drug once it is in customary use, at a fraction of the cost of Phase III clinical trials. (*Am J Public Health* 1990; 80:50–53.)

Introduction

In November 1981, the Upjohn Company's Medication Monitoring Program℠ initiated a postmarketing surveillance study of prescription acne medications. Prior to the initiation of the study, Upjohn introduced a topical clindamycin phosphate product (CLEOCIN T® Topical Solution) for the treatment of acne. Many other antibiotics are available for the treatment of acne. Since pseudomembranous colitis and severe diarrhea can occur with the administration of antibiotics,[1–6] with serious or even fatal consequences, it is important to measure whether the risk of developing colitis or severe diarrhea is increased in patients with acne being treated with topical antibiotics or low doses of oral antibiotics. Information from clinical trials, informal surveys, and case reports indicates that the risk in acne patients is rather low,[7–9] but estimates of the risk under normal use are lacking. We therefore conducted a postmarketing surveillance study to estimate the frequency of colitis and diarrhea in a sample of acne patients presenting prescriptions to pharmacies.

Methods

Recruitment of Pharmacies

A pharmacy-based model of postmarketing surveillance was developed by the Medication Monitoring Program.[10] Retail pharmacies, pharmacies associated with a dermatology clinic, university student health centers, and pharmacies on military bases were recruited to participate in the study, based on willingness to participate and medication dispensing profile. Fifty-eight pharmacies participated in the study, which represented a broad geographic distribution. Pharmacists were nominally reimbursed for their participation.

To create a registry of acne medication users from which to sample for further follow-up, we asked cooperating pharmacists to explain the study to all individuals who presented a prescription for an acne medication and invite them to participate. A total of 13,465 patients were registered by pharmacists and 10,282 (76.4 percent) signed a consent form agreeing to further follow-up and access to their medical records. Participation rates within pharmacies ranged from 9.3 percent to 100 percent and did not vary markedly by type of pharmacy.

Interviews

Participants were assigned different interview forms, based upon the drug or drugs on the registry, to save cost. Patients prescribed topical antibiotics were given the complete interview, which included patterns of use, assessment of acceptability and efficacy, and the occurrence of new health events (including non-hospitalized events, such as changes in bowel habits). Participants prescribed an oral antibiotic and any other prescription acne medication were randomly assigned to the complete interview module, to a short interview which collected only patterns of use, perceived acceptability, and the occurrence of hospitalizations and pregnancies, or to no interview.

A total of 9,965 (96.9 percent of 10,282) patients was assigned to receive two computer-assisted telephone-administered interviews: the first was scheduled five weeks following enrollment and the second was scheduled to be administered eight weeks later. The interviews were scheduled to cover the interval during which antibiotic-associated diarrhea or colitis would be most likely to occur if the patient was using the drug for the first time.[11]

If contact could not be made with the patient, a certified letter was sent to his/her last known address. The individual was asked to call the study collect or return a postcard indicating whether he/she still wished to participate. Patients who did not respond to the certified card were designated lost to follow-up and a questionnaire was sent to the enrolling institution or the prescribing physician. The questionnaire was used to determine last known vital status and whether the patient had been hospitalized during the follow-up period. No deaths were reported in this study. Of the 893 (9.0 percent of 9,965) patients lost to follow-up, 15 hospitalizations for 13 (1.5 percent) patients were reported. The mean age of those lost to follow-up was 22.7 years (range: 13 to 62 years); 60.3 percent were female.

A total of 8,304 (83.3 percent) patients completed the first interview (any module) (Table 1). Only data from the 6,453 patients who completed the full set of questions will be presented in this report. Patients were not paid for their participation.

The mean age of the participants was 22.9 years (range: <1 year to 80.6 years); 63.7 percent were female. The mean age of those not signing a consent form authorizing further follow-up was 23.4 years (range: 2.9 years to 78.1 years); 50.7 percent were female. Of all females registered, 80.2 percent

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TABLE 1—Interview Status of Study Participants

	Frequency	% of Total
Signed Consent	10282	(100.0)
Assigned to interview	9965	(96.9)
First interview completed	8304	(80.8)
First interview not completed	1661	(16.2)
Participation terminated to reduce interviewer backlog	685	(6.7)
Lost-to-followup	521	(5.1)
Registry received later than protocol requirements	199	(1.9)
No telephone	71	(0.7)
Withdrew consent	58	(0.6)
Did not use acne medication	48	(0.5)
Moved outside United States	34	(0.3)
Hostile/abusive respondent	26	(0.3)
Data processing error	19	(0.2)
Second interview completed	7424	(72.2)
Second interview not completed	880	(8.6)
Lost-to-followup	372	(3.6)
Data processing error	247	(2.4)
Moved outside United States	129	(1.3)
Withdrew consent	104	(1.0)
Hostile/abusive respondent	28	(0.3)

NOTE: This table includes only those registries in which patients agreed to participate in the study (76.4% of total registries).

agreed to participate compared to 72.8 percent of the registered males.

Investigation of Health Events

Hospitalizations, pregnancy outcome, and diarrhea requiring a physician contact were investigated further. A letter of inquiry was sent to the physician or institution requesting confirmation of dates, reasons for contact or hospitalization, and the final diagnosis. If insufficient information was returned, the report was referred to field investigation for resolution. The determination of the appropriateness of each investigated diagnosis was conducted by the medical monitor in a blinded fashion.

The criterion to define episodes of diarrhea was the patient report. No attempt was made to characterize the frequency or consistency of the stool. Clinically significant diarrhea was defined as that requiring contact with a physician and was investigated through the patient's physician. Patients who reported having the diarrhea at the time of the interview were contacted two weeks later and queried about the episode.

Results

Acne Medication Use

Multiple drug use was common, with individuals reporting an average of 2.6 acne drugs during the follow-up period. Of the 6,453 patients, 5,832 (90.4 percent) reported using at least one antibiotic during the follow-up period: 1,361 (23.3 percent) reported using only an oral antibiotic; 2,021 (34.7 percent) reported using only a topical antibiotic; and 2,450 (42.0 percent) reported using both an oral and a topical antibiotic.

Reports of Diarrhea

A total of 276 (4.3 percent) patients reported the occurrence of an episode of diarrhea during the follow-up period. The mean age of those reporting diarrhea was 23.8 years (range: 13.2 years to 53.4 years); 73.9 percent were female. A total of 4.8 percent of the female patients reported an occurrence of diarrhea compared to 3.3 percent of the male patients.

Of these 276 patients, 252 (91.3 percent) reported exposure to one or more antibiotics during the follow-up period. Table 2 presents the frequency of reported diarrhea by antibiotic exposure. Due to the large number of different types of antibiotics prescribed and the relatively small number of diarrhea cases, the exposure categories were collapsed to topical antibiotics and oral antibiotics. The rate of reported

TABLE 2—Frequency and Characteristics of Episodes of Diarrhea Reported by Acne Study Patients by Antibiotic Exposure

	Oral and Topical Antibiotics	Topical Antibiotics Only	Oral Antibiotics Only	No Antibiotic Exposure	Total
Total number of patients exposed	2450	2021	1361	621	6453
Number reporting diarrhea (%)	110	98	44	24	276
	(4.5)	(4.8)	(3.2)	(3.9)	(4.3)
Associated Symptoms* (%)					
Blood/Mucus in stools	17	19	6	3	45
	(15.5)	(19.4)	(13.6)	(12.5)	(16.3)
Abdominal cramps	63	68	28	14	173
	(57.3)	(69.4)	(63.6)	(58.3)	(62.7)
Nausea	59	54	23	14	150
	(53.6)	(55.1)	(52.3)	(58.3)	(54.3)
Fever	32	28	9	7	76
	(29.1)	(28.6)	(20.5)	(29.2)	(27.5)
Associated Factors*					
Shared quarters with someone with diarrhea	26	30	4	3	63
	(23.6)	(30.6)	(9.1)	(12.5)	(22.8)
Changed diet prior to onset	29	29	18	7	83
	(26.4)	(29.6)	(40.9)	(29.2)	(30.1)
Trip prior to onset	23	29	11	4	67
	(20.9)	(29.6)	(25.0)	(16.7)	(24.3)
Similar diarrhea previously	65	68	22	14	169
	(59.1)	(69.4)	(50.0)	(58.3)	(61.2)

*Patient may report multiple symptoms/factors
( ) % of number reporting diarrhea

diarrhea was slightly higher among users of topical antibiotic preparations than among users of oral antibiotics (RR = 1.5; 95% CI = 1.06, 2.13).

Further information was obtained about each reported episode. Table 2 shows the distribution of reported symptoms associated with the diarrhea by drug exposure. Blood and mucus in the stools were not asked separately. There were no significant differences in the frequency of reported symptoms associated with the diarrhea episode by antibiotic exposure. Patients were also asked whether they ever had diarrhea like this before, whether any individuals with whom they share living quarters had diarrhea around the same time, whether there had been any significant changes in diet prior to onset, and whether they had taken any trips prior to onset. The frequency of diarrhea reported by the season of the year did not vary significantly.

Twelve (0.2 percent) of 6,453 patients reported discontinuing a drug due to diarrhea; four patients discontinued oral tetracycline, two patients each discontinued topical clindamycin phosphate, oral erythromycin, and oral minocycline, and one patient each discontinued topical meclocycline and topical clindamycin HCL. Of the 3,811 patients exposed to at least one oral antibiotic, eight (0.2 percent) reported discontinuing an oral antibiotic due to diarrhea. Of the 4,471 patients exposed to at least one topical antibiotic, four (<0.1 percent) reported discontinuing a topical antibiotic due to diarrhea.

Eighty-two (1.3 percent) of 6,453 patients reported 85 contacts (visits or telephone contact) with a physician about the diarrhea episode. Confirmation of a diagnosis of diarrhea was obtained for 46 (54.1 percent) of the contacts. Three physician-confirmed cases of antibiotic-associated diarrhea included one case associated with tetracycline (oral), one case associated with erythromycin (oral), and one case associated with minocycline (oral). The causal association was based solely on the medical monitor's review of the physician's diagnosis obtained from the patient's records. No further confirmation of the diagnosis was obtained.

Colitis

Of nine (0.1 percent) patients reporting colitis, four of them reported no contact with a physician. Follow-up of the remaining five cases revealed only two cases of confirmed colitis, neither drug-related. Two other patients reported diarrhea, and follow-up with the treating physicians revealed oral tetracycline-associated colitis and stress-related colitis. Neither patient was hospitalized for the condition.

Hospitalizations

A total of 92 (1.4 percent) patients reported 93 hospitalizations during the follow-up period. The mean age of those hospitalized was 23.9 years (range: 11.1 years to 60.8 years); 63 (68.5 percent) were female. Table 3 presents a summary of the discharge diagnoses for 75 of the hospitalizations. Eighteen (19.4 percent) of the reported hospitalizations could not be confirmed. Two patients reported being hospitalized for diarrhea. One of the reports could not be confirmed and the discharge diagnosis for the other hospitalization was acute viral gastroenteritis.

Pregnancy

A total of 41 (1.0 percent) women reported being pregnant during the follow-up period. Their mean age was 24.8 years (range: 17.5 years to 34.3 years). Twenty-six (63.4 percent) of the deliveries were confirmed and four women volunteered that they had an abortion. Of the 26 confirmed births, 20 infants were normal, one infant was premature, one infant had hyperbilirubinemia, and the status of the remaining four cases was unknown. In all confirmed cases, the women reported discontinuing their acne medications during the pregnancy.

TABLE 3—Distribution of Confirmed Discharge Diagnoses for Reported Hospitalizations among Acne Study Group

Discharge Diagnosis*	Frequency	Column %
Musculoskeletal Disorders	21	18.4
Infections	18	15.8
Trauma	17	14.9
Gastrointestinal Disorders	17	14.9
Gynecologic Disorders	12	10.5
Respiratory Disorders	5	4.4
Neuropsychiatric Disorders	5	4.4
Urinary Tract Disorders	4	3.5
Other, NEC	15	13.2
Total	114	100.0

*Diagnoses categorized with SNOMED[14]

Discussion

This approach relies on the pharmacist or other pharmacy personnel to recruit patients. Probably not all patients who presented a prescription for an acne medication were invited to participate in the study; possibly invitation to participate was not random. For example, pharmacy staff may have invited patients only when they were not hurried or busy, or invited only patients who appeared friendly. We oriented the pharmacy personnel in person and frequently monitored their progress for significant variations. We did not audit the prescription records within each pharmacy, although a study in progress will do so. The impact of incomplete ascertainment or selection of only particular types of patients is unknown, but if the likelihood of invitation was not related to the medication prescribed, the comparisons of side effects among medications would not be affected.

We found that the patients used many other acne drugs that did not appear on the study registry. Since we designed the interviews to collect extensive information only about the registry drugs, we lacked important data about first time vs repeat use, length of prior use, dose and regimen.

The accurate determination of the presence of diarrhea is critical to the interpretation of the results. Stool collections or observations to accurately characterize the presence of diarrhea are generally not achievable in a general population-based study.[12] Daily diaries were thought not to be feasible due to the nature and the setting of the patient recruitment. We were left with the patient recalling the occurrence of diarrhea over a five- to eight-week period of time. We felt that clinically significant diarrhea would result in a contact with a physician and thus be available to the study by confirmation through medical records. Diarrhea serious enough to prompt the patient to seek a physician was confirmed in 46 (0.7 percent of 6,453) patients.

Despite some limitations imposed by the study design, our results are not substantially different from published data. Stergachis reported four cases of antibiotic-associated colitis that required hospitalization for treatment in a population of approximately 280,000 individuals.[13] We identified no cases of antibiotic-associated colitis that required hospitalization for treatment and one case of antibiotic-associated colitis that did not require hospitalization.

We conclude that while the occurrence of antibiotic-associated colitis and diarrhea appears to be rare in this patient population, appropriate education of the patient by the physician and the pharmacist to the potential side effects of treatment appears to be warranted.

Although epidemiologic postmarketing studies have been recommended to get further information about the safety of a drug once it is in customary use, few have been completed. We found pharmacist and patient cooperation to be high. Of the 10,282 patients who agreed to participate, we obtained complete data from 6,453 (62.8 percent). We initially assigned participants to long or short interviews to minimize the costs, but found the difference in the costs of obtaining complete information compared to partial information was inconsequential, once the patient was enrolled.

This methodology requires a sophisticated organization made up of epidemiologists, physicians, computer programmers, biostatisticians, and field personnel. Once the organization is in place, the cost of conducting a study such as this is very reasonable. Borden and Lee estimate that the cost of a study with this design is about 1 percent of that of the standard clinical trial.[10]

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AAAS Meeting to Feature Wide Range of Science, Medicine and Technology

A broad look at science—from the developing brain to exploring Mars—will be the focus of the upcoming 156th annual meeting of the American Association for the Advancement of Science (AAAS) when it convenes in New Orleans, Feb. 15–20, 1990.

Over 250 symposia will showcase what is happening in science and how that affects everyone. Among the topics featured will be neurotransplants, oil contamination in Prudhoe Bay, reducing the toll of natural hazards in the 1990s, exploration of Mars, progress in AIDS treatment, psychology of jury decision-making, climate change, chaos, effects of substance abuse on women, misconduct in science, physiology of exercise, computer viruses, and emotions and the developing brain.

A special feature of the meeting will be the inclusion of two separate three-day seminars on protein folding and the biology of parasitism; these symposia require an additional registration fee. Another feature of the meeting—"Public Science Day"—takes place on the opening day, February 15. The event will include workshops, demonstrations, and conversations with scientists. Public Science Day activities will feature programs to involve local students and their families in math and science. Also on Thursday, February 15, several thousand New Orleans area students will participate in a day-long "Science Encounters" program of lectures, demonstrations, and opportunities for young people to meet and talk with individual scientists and engineers. The AAAS Science and Technology Exhibition, February 16-19, will be at nearby Rivergate Exhibition Center. Poster sessions will also be featured adjacent to the exhibits.

The AAAS, formed in 1848, is the nation's leading general scientific organization, with more than 132,000 individual members and nearly 300 affiliated scientific and engineering societies and academies of science. For more information, contact AAAS, 1333 H Street, NW, Washington, DC 20005. Tel: (202) 326-6440.

Dependency on Soup Kitchens in Urban Areas of New York State

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Abstract: We studied the dependency of persons on soup kitchens in Albany, Buffalo, Rochester, Syracuse, and Westchester County, New York. Seventeen percent of the meal recipients were homeless, 62 percent lived in apartments or houses, 20 percent were working, 40 percent were women, and 17 percent had a child in their household. Fifty-nine percent started eating at the soup kitchen more than a year ago, and 51 percent ate five or more meals at soup kitchens in the last week. Most reported they came to the soup kitchen because of economic problems or lack of food; 93 percent had incomes below the poverty threshold. Most used some government food program; 48 percent received food stamps. Utilization of soup kitchens and other programs differed between men and women and between households with and without children. (*Am J Public Health* 1990; 80:57–60.)

Introduction

The number of meals served by a national sample of private emergency food programs increased by 21 percent from 1983 to 1984 and by 17 percent from 1984 to 1985.[1] In New York State, other than New York City, the estimated number of meals served monthly at soup kitchens was 204,334 in 1987;* this represents an increase from estimates for 1985 and 1986.[2] Other reports have documented the increased use of emergency food programs, discussed possible causes for it, and recommended policies to address the food needs of the users of emergency food programs.[3–6] However, there is little information about the people who are dependent on these programs.

This paper seeks to answer the following questions:

- What were the characteristics of persons dependent on soup kitchens in five urban areas of New York State outside of New York City?
- What was the extent of and reasons for their dependency on soup kitchens?
- What other food programs did they use?
- What were their perceived needs for health and other services?
- Were there differences in the patterns of dependency between men and women and between meal recipients who lived in households with and without a child?

Methods

Selection of Sample

In 1987 a survey was conducted of soup kitchens that served meals to the public during April in Albany, Buffalo, and Westchester County, and during October in Rochester and Syracuse. Of the 34 soup kitchens in these urban areas, 28 participated in the study.

It was not possible to take a random selection of guests from the sites because the total number of unduplicated guests who ate meals at the sites was not available. However, since the total number of meals served by the sites was available, meals were sampled, and the soup kitchen guests served those meals were interviewed. Because meals were sampled, guests who ate more meals at soup kitchens than others had a greater likelihood of being interviewed.

The goal was to select 20 meals at each site. An average of 17.9 meals were selected per site. Because an earlier study[7] found that soup kitchens served a greater number of meals at the end of the month, more meals (70 percent) were selected from the last half of the month.

A variety of mealtimes for interviewing guests was selected, and at each selected mealtime, the recipients of two or three randomly selected meals were interviewed. If the recipient was a child, the accompanying adult was interviewed. If a recipient refused to be interviewed, or if someone in his or her household had already been interviewed, the person next to that individual or the recipient of the next randomly selected meal was interviewed. Of the 501 soup kitchen guests interviewed, 87 percent were the recipients of the original randomly chosen meals.

Data Collection and Analysis

The guests were asked a series of primarily closed-ended questions about their demographic characteristics, household composition, sources and amounts of household income, use of health services, and use of other food programs. Each interview took about 25 minutes.

Unweighted frequency tables were created from the sample data and the chi-square statistic applied.[8] For the frequencies and means of the distribution of single variables, combined urban estimates were calculated from the sample data by combining site estimates so as to weight for the number of meals served in each site and in each urban area, assuming that the guests at nonparticipating soup kitchens were similar to those at participating soup kitchens. The combined urban estimates represent the monthly total of soup kitchen meals (82,449) served in the five urban areas.

To estimate variances and standard errors, and to study the implications of varying sampling designs, the betabinomial distribution[9,10] was used to model percentage estimates. The method of moments[9] was applied to 37 dichotomous variables and adjusted for the finite population of sites.[11] Analysis of variance was used to determine what proportion of the among-site variance was due to within-city and among-city variances.

To determine to what degree the results based on a sample of meals could be generalized to the population of soup kitchen guests, estimates were recalculated for those variables related to the probability of a guest being selected into the sample, i.e., each response was multiplied by the

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reciprocal of the number of times each guest had eaten at soup kitchens in the last week. Responses for those who ate more than 10 meals were weighted by 1/10 so as not to underweight the responses of frequent users.

Results

For 34 of the 37 variables analyzed using the betabinomial distribution, more than half of the total variance was due to the among-site variance. The standard errors estimated from the percentages in the total sample ranged from 1.1 percent to 2.9 percent. Therefore, 3 percent can be considered as a general (conservative) estimate of the precision of the percentages reported in this paper. The variation within cities was found to be greater than the variation among cities. For 26 of the 37 variables examined, the among-city variance was zero. For the remaining 11 variables, the ratios of the among-city variance to the within-city variance ranged from .07 to .45, indicating that the among-city variance was at most less than one-half the size of the within-city variance.

When the estimates based on meals were compared to the new estimates based on people, only five variables had estimates that differed by 5 percent or more. The estimates based on meals were larger than those based on people for eating at another soup kitchen (21 percent vs 12 percent) and homelessness (17 percent vs 12 percent), and smaller for women (40 percent vs 45 percent), children in the household (17 percent vs 22 percent), and another adult in the household (12 percent vs 25 percent). Therefore, except for these few variables, the reported estimates for the population of soup kitchen meals can also be interpreted as estimates for the population of soup kitchen guests. Throughout the rest of the paper, the results based on the sample of meals are reported for meal recipients.

- *Who was dependent on soup kitchens?*

Demographic Characteristics

The majority of the meal recipients were men (60 percent). The average age of meal recipients was 43.6 years and 20 percent of them were 60 years old or older. Most of them were White (65 percent) although 29 percent were Black. Their educational attainment was generally low; 52 percent did not have a high school diploma. Most of the meal recipients lived alone or in a shelter or group home (70 percent).

Employment

Only 20 percent of the meal recipients were employed. Although 37 percent of the unemployed were looking for work, many of the unemployed were disabled (32 percent), retired (19 percent), raising a family (11 percent), or attending school or a training program (5 percent). Meal recipients who were employed earned on average $5.47 per hour and held labor (45 percent) and service (30 percent) positions (Table 1).

Income

Ninety-three percent of the meal recipients' household incomes in the previous month were less than 100 percent of the poverty level and 55 percent were less than 50 percent of the poverty level (Table 2). The most frequently reported sources of household income were public assistance other than Aid to Families with Dependent Children (AFDC) and work relief (28 percent), Supplementary Security Income (SSI) (22 percent), Social Security (22 percent), and wages (21 percent).

TABLE 1—Types of Employment of Soup Kitchen Meal Recipients*

Type of Employment	Examples	Percentage of Meals for Those Employed
Laborer	Day labor, collecting bottles and cans, odd jobs	45
Service	Household worker, wait person, janitor	30
Operative	Delivery person, cab driver, assembly worker	16
Clerical	Teacher's aide, clerk, secretary	7
Crafts person	Carpenter, mechanic	7
Professional/Manager	Substitute teacher	3

*These percentages do not add to 100 because more than one response was possible. Ninety-six sampled meal recipients responded to this question.

TABLE 2—Household Income of Soup Kitchen Meal Recipients in Previous Month

	Percentage of Meals
Monthly Household Income (does not include food stamps)	
$0–150	27
$151–300	22
$301–600	41
$601–$1000	7
$1001 +	4
Average (mean) per person household income $275	
Sources of Household Income	
Other public assistance	28
Social Security	22
SSI	22
Wages	21
AFDC	9
Work Relief	9
Retirement	4
Veterans' disability	2
Workers' compensation	2
Unemployment	2
Child support	1

*These percentages do not add to 100 because households received income from more than one source.

Housing

The problems that bring people to soup kitchens affect a broader segment of the population than just the homeless. Sixty-two percent of the meal recipients lived in apartments or houses. Only 17 percent were homeless—persons who nad lived in a shelter (11 percent), or on the streets or in no one place (6 percent) during the previous month,** and 20 percent lived in a room or group home. However, other indications of housing instability were that 15 percent of the meal recipients had moved since the month before, and 7 percent were living with unrelated people.

Thirty-one percent of the meal recipients who lived in apartments, houses, or rooms had housing that was partially subsidized or paid for by the government. It was not clear what portion of the rent of guests with subsidized housing was subsidized. However, for those without subsidized

**This percentage would have been greater if soup kitchen sites that served meals only to residents of their shelters had been included in the sample.

housing, the average monthly household rent was $193 and comprised 57 percent of the average household income.

- *What was the extent of and reasons for dependency on soup kitchens?*

Soup kitchen meal recipients tended to use soup kitchens often and over a long period of time. Fifty-one percent of the meal recipients had eaten five or more meals at soup kitchens during the previous week, and 59 percent had started eating at the soup kitchen more than a year ago.

The most common reasons that meal recipients gave for eating at the soup kitchen were the need for food (71 percent), including such reasons as no place to cook and nowhere else to eat, and economic problems (48 percent), such as no money (Table 3). Eighty-nine percent gave at least one of these reasons. However, 41 percent of the meal recipients also gave social reasons such as the need to be among people and the support of the soup kitchen staff.

- *What other food programs were used by soup kitchen meal recipients?*

Public Programs

Government food programs were used by almost two-thirds (64 percent) of the meal recipients' households, and many of these households received food from more than one program. Forty-eight percent of the soup kitchen meal recipients' households received food stamps in the previous month, 54 percent of the meal recipients whose households did not receive food stamps had received food stamps sometime in the past, 8 percent had applied for food stamps but did not receive them, and 38 percent had never applied for them.

Meal recipients' households that received food stamps received on the average $80 in food stamps in the previous month, and 96 percent of them had household incomes that were below poverty level even when the value of their food stamps was included as income.

Forty-two percent of the meal recipients' households had received food from a free food, cheese, or other government surplus distribution in the previous month. Of those meal recipients' households who may have been eligible for the program, 29 percent received food from a senior center, 57 percent received food from a free or reduced price school breakfast, and 66 percent from a free or reduced price school lunch program, and 37 percent received the Special Supplemental Food Program for Women, Infants, and Children (WIC) benefits.

TABLE 3—Meal Recipients' Reasons for Using Soup Kitchens[a]

Reasons	Example	Percentage of Meals
Food	No food, no place to cook, don't know how to cook, special diet, nowhere else to eat/go	71
Economic reasons	No job, waiting for public assistance, no money	48
Social reasons	Love the people here, get out, lonely	41
Program	Shelter, religious services, referred to program, to get help, volunteer	12
Convenience	Close by, kill time, break monotony, other site closed, save time on cooking	11
Miscellaneous	Family difficulty, medical problems, for exercise, old	4

[a]These percentages do not add to 100 because meal recipients gave more than one reason.

Private Programs

Many meal recipients had received food from other private food sources in the previous month: food pantries (33 percent), shelters that served meals only to residents (9 percent), and credit from a grocery store (3 percent).

- *What were the meal recipients' perceived need for other services?*

Housing

Many of the meal recipients lived in housing that lacked basic facilities. Sixty-one percent of the meal recipients did not have access to a phone, 29 percent did not have access to a working refrigerator, and 28 percent did not have access to a working stove or range.

Health Services

The most frequently mentioned forms of health insurance were Medicaid (56 percent) and Medicare (21 percent). Twenty-three percent of the meal recipients had no health insurance, and 19 percent reported their families had medical expenses in the previous year that were not covered by insurance. More of the meal recipients reported a need for dental services that their family could not afford (18 percent) than a need for medical services (6 percent).

Access to Food

Fifteen percent of the meal recipients had difficulty getting to a grocery store; 11 percent had problems with transportation, and 7 percent had health or mobility problems that made it difficult for them to get to a store.

- *What differences were there in the dependency of subgroups of guests?*

Reports that have noted an increase in soup kitchen use have also noted an increase in the number of families with children eating meals at soup kitchens.[1,3,4] In the cities included in this study, 40 percent of the meal recipients were women and 17 percent of the meal recipients' households included children.

Men vs Women

The men and women eating meals at soup kitchens differed in that women were more likely to have health insurance (86 vs 75 percent), to have received AFDC (17 vs 1 percent), or SSI (31 vs 20 percent), and to have received food stamps (64 vs 39 percent), surplus foods (58 vs 38 percent), and food from a food pantry (44 vs 32 percent). Men had an average per person household income of $328 compared to $255 for women. Men were more likely to have received food from a shelter in the previous month (15 vs 6 percent), and ate more frequently at soup kitchens in the previous week (4.7 vs 3.2 meals).

Guests Living with and without Children

Soup kitchen meal recipients who lived with children were on average younger than meal recipients who did not live with children (35.7 vs 46.5 years), had lower average per person household incomes ($146 vs $330), were less likely to receive Social Security (12 vs 29 percent) and were more likely to receive AFDC (37 vs 1 percent), other public assistance (36 vs 25 percent) and food stamps (74 vs 43 percent), and were less likely to eat at shelters (2 vs 14

percent). They had on average eaten fewer meals at the soup kitchen in the last week (2.8 vs 4.4) and had been eating at the soup kitchen for a shorter period of time (16.7 vs 23.0 months).

Discussion

For future studies of soup kitchen meal recipients, the large among-site variance suggests that sampling from more sites could reduce the variance of estimates. For example, given a population with 100 sites, the standard error of an estimated percentage based on a sample of 40 meal recipients from five sites would be about 11 percent compared to only 6 percent for a sample of 10 meal recipients from 20 sites.

A variety of people other than single, homeless, males were found to eat at soup kitchens. Subgroups differed in their use of soup kitchens, their use of other programs, and their resources. Men and guests who do not have children in their households may be less likely than others to be, or perceive themselves to be, eligible for government benefits and other programs or more likely to prefer to eat at soup kitchens. Whether the goal is to reduce the number of people having to use soup kitchens or to serve the unmet needs of soup kitchen guests, differences in the utilization of programs among these subgroups should be kept in mind.

We also found that a large number of soup kitchen meal recipients ate often at soup kitchens and had been eating at them for a long period of time. To the degree that guests do not use soup kitchens as a source of "emergency food" but eat at them often and over a long period of time, more attention needs to be given to the nutritional quality of soup kitchen meals.[12]

Over one-half of the meal recipients had no access to a telephone and 18 percent stated their families needed dental services which they could not afford. The use of a telephone is an important resource for obtaining employment as well as interacting with social services to apply for and receive benefits. Neglected dental problems may interfere with soup kitchen guests' abilities to eat adequate diets.

Many soup kitchen meal recipients were participating in government food programs, such as food stamps, and still eating at soup kitchens. This suggests that government food programs may not provide all persons with sufficient resources to obtain enough food. Most of those receiving food stamps reported incomes below the poverty threshold even when their food stamp benefits were considered as income. Many of the meal recipients gave a social reason as one of their reasons for coming to the soup kitchens. Participation in a government food program, such as food stamps, may not substitute for the feeling of belonging and the social support that many soup kitchen guests receive at soup kitchens.

More research is needed on soup kitchen meal recipients to determine: why more than one-half of them were not using food stamps; how housing problems other than homelessness, such as the cost of housing and the lack of basic facilities, e.g., a refrigerator, limit their access to food; and what other services are needed besides meals to improve their situations.

The findings from this study of soup kitchen guests indicate that any policies or programs to address the needs of soup kitchen guests must look past oversimplified views of soup kitchen guests, e.g., the homeless, singles, males, persons who have not accessed public programs, or persons who are eating at soup kitchens because of a temporary emergency. Soup kitchen guests include a variety of persons with a variety of patterns of and reasons for dependency on these programs.

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Predictors of the Initiation of Psychotherapeutic Medicine Use

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Abstract: This study examined the hypothesized predictive association between illicit drug use and the onset of prescribed psychotherapeutic medicine use. The effect of psychiatric symptoms on this relationship was also explored.

Data were gathered retrospectively, through standardized household interviews conducted in 1981 for the Baltimore site of the National Institute of Mental Health Epidemiologic Catchment Area Program. For this analysis, the sample was restricted to 1,716 respondents ages 18–44 at the time of interview, drawn by probability sampling area household residents of all adult ages. Using Cox proportional hazards models, the onset of psychotherapeutic medicine use was modeled as a function of illicit drug use and psychiatric symptoms, both defined as time-dependent covariates. Newly observed in this analysis was an independent association of illicit drug use with psychotherapeutic medicine use, which was unexplained by psychiatric symptoms (relative hazard = 2.61, 95% confidence interval (CI) = 2.15, 3.18). Adjustment for age of onset of alcohol intoxication did not alter the impression that illicit drug users were more likely to initiate use of prescribed psychotherapeutic medicines (relative hazard = 2.52, 95% CI = 2.02, 3.11). (*Am J Public Health* 1990;80:61–65.)

Introduction

Studies of drug sequencing have focused on detecting patterns in the age of onset of drug and medicine use among adolescents and young adults.[1–3] This research has identified a modal sequence of onset beginning with alcohol and cigarette use, followed by marijuana use, and then use of other illicit drugs. In one of the latest studies on this issue,[4] initiation of marijuana use was found to precede initiation of prescribed psychotherapeutic medicine use.

Based on these results, researchers hypothesized[5,6] that there might be a relationship between illicit drug use and psychotherapeutic medicine use. Yamaguchi and Kandel reported that illicit drug use increased the risk of initiating psychotherapeutic medicine use, using retrospective reports of the age of onset of different types of drug use. They then showed that the presence of depressive symptoms reduced this relation between the substances among women but not among men. Other studies lending support to the potential for symptoms to act as an explanatory link are those which found that relief of symptoms was a major reason given for use of illicit drugs [7,8] and those that show a direct relation between psychotherapeutic medicine use and psychiatric distress measured cross-sectionally.[9–12]

In addition to the role that psychiatric symptoms or distress may play in influencing the use of illicit drugs and psychotherapeutic medicines, other possible etiologic mechanisms have been suggested. Raveis and Kandel [13] proposed that a change in substances used over the lifespan may be part of a maturation process that coincides with the assumption of adult roles, such as establishing a residence, beginning a job, and marriage. In a study of young adults during the three years after high school graduation, Bachman, *et al*,[14] found that illicit drug use decreased from high school levels for those who were married, increased for those who were single and living away from home, and was unchanged for those who remained with their parents.

Drug use also may be understood as a learned behavior, in which reinforcing consequences of illicit drug use later influence the taking of prescribed psychotherapeutic medication.[15] Further, in a study by Newcomb, *et al*,[16] illicit drug use was correlated with deviance and with a sensation-seeking trait in adolescents, both of which could account for a relation between illicit drug use and prescribed psychotherapeutic medicine use.

In this study we hypothesized that if the data were to support such a relation, this would be due to the effect of psychiatric symptoms, such as anxiety or depression, on both forms of drug use. We also explored the effect of the sequence of onset of illicit drug use and psychiatric symptoms upon the initiation of psychotherapeutic medication. This allowed us to compare individuals who experienced symptoms prior to illicit drug use with individuals who used illicit drugs and then developed symptoms, in terms of their likelihood of subsequent psychotherapeutic medicine use. Sociodemographic variables that may influence the occurrence of psychotherapeutic medicine use were treated as potentially confounding or modifying covariates.

Our study expands upon prior research in this area by its use of a more heterogeneous sample with a wider age range, and by broadening coverage of psychiatric symptom categories as opposed to earlier research in which only one symptom category, such as depressive symptoms, was measured. Furthermore, incorporation of age of onset data into the analyses, for the first time allows us to examine the effect of sequence of onset of symptoms, illicit drug use, and prescribed psychotherapeutic medicine use.

Methods

Study Sample

The study sample consisted of subjects selected for the Baltimore site of the National Institute of Mental Health Epidemiologic Catchment Area Program. The details of the survey design have been reported elsewhere.[17] Briefly, the population from which the sample was drawn was comprised of adult household residents of the Eastern Baltimore Epidemiologic Catchment Area (ECA), approximately the eastern one-third of Baltimore City. Respondents were chosen for inclusion in the sample using area probability sampling. Household residents eligible for selection were all persons ages 18 or over at the time of contact in 1981, with the ECA defined as their usual place of residence. The survey completion rate for designated respondents ages 18–44 ranged

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from 88–93 percent for race other than White and 80–89 percent for Whites.[17]

Of the 3,481 people interviewed, all subjects ages 18–44 at the time of interview were included in the present analysis (n = 1,716). The upper age limit for inclusion in the study was 44 years, since onset of illicit drug use after this age was quite rare.[18] The sample consisted of 1,048 females (61 percent) and 668 males, with 916 Whites (53 percent), 746 Blacks (44 percent), and 54 (3 percent) of other races. Seventy-six percent of the sample was in the married or single categories at the time of interview, and 45 percent had annual household incomes under $15,000. Twenty-seven percent of the respondents reported that they had sought help for a nervous or emotional problem at least once in their lifetime.

Study Variables

Information on the study variables was obtained from standardized interview questions covering demographics, health services utilization, the Diagnostic Interview Schedule, and a detailed survey of medicine use. Detailed information on the survey interview has been published in a monograph.[17]

Psychotherapeutic medicines were defined as those used primarily to treat, control, or prevent mental and behavioral disturbances, or to alleviate symptoms of psychopathology. Included in this definition were prescribed anti-psychotic agents, anti-anxiety agents, anti-depressants, stimulants, daytime sedatives, and hypnotics. Excluded were medicines with some psychotherapeutic action that are primarily prescribed for other purposes, such as analgesics, and over-the-counter medicines. Respondents were considered to be positive for psychotherapeutic medicine use if they stated that they had taken any medicines included in the above definition one-or-more times. The age of onset of psychotherapeutic medicine use was defined as the age at which a respondent first used a psychotherapeutic medicine.

Illicit drug use was defined as any use of drugs to get high, or without a prescription, or in greater quantity than was prescribed. Drug categories included marijuana, amphetamines, barbiturates, tranquilizers, cocaine, heroin, methadone, opiates, and psychedelics, with age of onset of illicit drug use defined as the reported age at which a respondent first engaged in illicit drug use. Age of onset of illicit drug use was distinguished from age of onset of psychotherapeutic medicine use by means of specific decision rules (see Data Analysis).

Data on psychiatric symptoms were obtained using the Diagnostic Interview Schedule. The term symptom was used to denote either the presence of psychiatric symptoms or a discrete spell of psychiatric disturbance occurring at any time in the respondent's life. Symptoms included were nervousness, delusional thoughts, hallucinations, pathologic worry, compulsions, and phobias. Spells included depressive spells, manic spells, and anxiety and panic attacks. The age of onset of a psychiatric symptom or spell was defined as the reported age at which a symptom or spell was first experienced. For individuals experiencing more than one category of symptoms, age of onset was defined as the youngest age at which the respondent reported onset of a symptom.

Data Analysis

Analyses were conducted using Cox proportional hazards models for censored survival data, with illicit drug use and psychiatric symptoms defined as time-dependent covariates.[19–21] Since time (t) in this analysis was defined as chronological age, all models were carefully age-adjusted as suggested by Breslow.[20] Illicit drug use occurring prior to the onset of psychotherapeutic medicine use was coded as zero until the age of onset of such use, at which point it was coded as 1. Thus, for the covariate illicit drug use, a subject would have a value of zero for every age until an illicit drug was used. If an illicit drug was first used at age 18, the subject would have a value of 1 for illicit drug use beginning at age 18 and continuing from that age onward. Psychiatric symptoms were treated in the same way. Subjects who had not used a psychotherapeutic medicine by the time of interview were treated as censored observations.

Forty-five respondents reported the same age of onset for illicit drug use and psychotherapeutic medicine use. This led us to be uncertain about whether prescribed psychotherapeutic medicines had been used illicitly by these respondents. To address this uncertainty we conducted analyses in two ways: first, these individuals were counted as positive for illicit drug use and second, as negative for illicit drug use. There was little difference in the results of these two approaches. In this paper we present the results of the second approach.

In addition to adjustment for age, models were also adjusted for other fixed covariates selected as a result of the heterogeneity with which they were distributed over psychotherapeutic medicine use in the study data, and from evidence from prior research on psychotherapeutic medicine prevalence.[22–28] Fixed covariates were defined as follows, with reference categories in parentheses: race = (Black)/other; sex = (male)/female; marital status = (married)/never married/widowed/separated-divorced; annual household income = <$15,000/$15–19,999/$20–24,999/($25,000+); and help-seeking = yes/(no). Help-seeking was defined as ever having sought help from a professional or a provider for a mental health, nervous or emotional problem, or for problems with alcohol and drugs.

For the purposes of analysis four models were fitted. Model 1 tested for the existence of a predictive association between illicit drug use and psychotherapeutic medicine use in the presence of the fixed covariates. Model 2 tested the hypothesis that the association between illicit drug use and psychotherapeutic medicine use, if present, could be accounted for by the existence of psychiatric symptoms. This was accomplished by entering the term for symptoms into the model, along with illicit drug use and the fixed covariates. Model 3 added an interaction term to Model 2, to test for a possible interaction between psychiatric symptoms and illicit drug use in relation to psychotherapeutic medicine use. Model 4 examined the effect of the sequence of onset of symptoms and illicit drug use on psychotherapeutic medicine use. In particular, Model 4 took the form:

$$h(t) = h_o(t)\exp[b_1x_1(t) + b_2x_2(t) + \gamma x_1(t)x_2(t) + b_3x_3(t)[x_1(t)x_2(t)] + b_4x_4 + \ldots b_px_p],$$

with $h_o(t)$ representing the underlying hazard of psychotherapeutic medicine use at age t for the reference category (i.e., all covariates = 0). The time-dependent covariates were represented by $x_1(t)$ = illicit drug use, $x_2(t)$ = psychiatric symptoms, $x_1(t)x_2(t)$ = interaction term for (symptoms) (illicit drug use), and $x_3(t)[x_1(t)x_2(t)]$ representing those for whom symptom onset preceded illicit drug use, with the term $x_3(t) = 1$ if symptom onset preceded illicit onset, and the fixed covariates were represented by $[x_4 \ldots x_p]$. The program BMDP 2L was used for fitting the models.[29]

Models 1–4 were generated for all respondents with

complete information on the fixed covariates (n = 1,537). The 25 widowed individuals were excluded from the analyses, since there were too few to generate stable estimates; most exclusions (142 of 179) were due to missing income data.

Since marital status, income, and help-seeking all were measured at the time of interview, the values for these variables prior to the onset of psychotherapeutic medicine use were not known. Therefore, no conclusions about the direction of their association with psychotherapeutic medicine use, if any, can be made. Despite this potential misspecification, these models offer a conservative test of the effect of illicit drug use on psychotherapeutic medicine use, since the covariates in question may be accounting inappropriately for variation due to illicit drug use.

Results

For the entire sample (n = 1,716), 554 (32 percent) of the respondents reported that they had used a psychotherapeutic medicine, 668 (39 percent) were positive for illicit drug use, and 785 (45.7 percent) had experienced psychiatric symptoms. Respondents were defined as positive for the predictor variables if they had used illicit drugs or experienced symptoms prior to the age of onset of psychotherapeutic medicine use or prior to the age at interview.

The age of onset of illicit drug use had a fairly limited range, with onset rarely occurring before age 10 or after age 29. The modal ages for initiation of illicit drug use were 16–19 years of age. Overall, the onset of psychotherapeutic medicine use was concentrated in the older ages of this sample. Instead of a relatively narrow period of initiation as was seen for illicit drug use, the onset of psychotherapeutic medicine use occurred in fairly steady proportions from ages 18–34.[30] The mean age of onset for illicit drug use was 19.35 years, and for psychotherapeutic medicine use 23.88 years.

Table 1 presents the results of the proportional hazards models which included illicit drug use and psychiatric symptoms, with adjustment for age, and for the fixed covariates race, sex, marital status, household income, and help-seeking. Model 1 showed that illicit drug use was associated with an increased risk of psychotherapeutic medicine use (rel. hazard = 2.65; 95% CI = 2.17, 3.21). That is, antecedent illicit drug use was associated with a risk of psychotherapeutic medicine use that was 2.65 times higher than the risk for individuals with no antecedent illicit drug use. In Model 2, the strength of illicit drug use as a predictor of psychotherapeutic medicine use was unchanged by adjustment for psychiatric symptoms. Thus, the presence in the model of a term for symptoms did not alter the relationship between illicit drug use and psychotherapeutic medicine use.

According to Model 3, illicit drug use was associated with an increased risk of psychotherapeutic medicine use whether symptoms had occurred (rel. hazard = 3.60) or had not occurred (rel. hazard = 3.10), with no significant interaction effect. Once again, illicit drug use was a predictor of psychotherapeutic medicine use and symptoms did little to alter this relationship.

Model 4 indicated that when both symptoms and illicit drug use occurred, respondents for whom symptoms preceded illicit drug use were more likely to initiate prescribed psychotherapeutic medicine use than persons for whom illicit drug use preceded symptoms (rel. hazards = 3.86 and 2.64, respectively). However, the confidence intervals were large and overlapped considerably (p = .063). In addition, those individuals with illicit drug use and no symptoms had a risk of psychotherapeutic medicine use similar to those experiencing either sequence of onset of symptoms and illicit drug use, suggesting that the presence of symptoms conferred little, if any, additional risk of psychotherapeutic medicine use over and above illicit drug use.

An additional version of Models 1 and 2 was generated that included onset of alcohol intoxication as a control variable. Onset of alcohol intoxication was defined as the age at which a respondent first reported becoming drunk, and was divided into three dummy variables that were not time-dependent: 1) onset from ages 3–15 years; 2) 16–20 years; and 3) >20 years, with the reference category defined as no onset of alcohol intoxication.

In Model 1, which was adjusted for onset of alcohol intoxication, there was little change in the strength of illicit drug use as a predictor (Table 2), as compared to the unadjusted Model 1 (Table 1). Similar findings were present for the alcohol-adjusted Model 2 (Table 2) as compared to the unadjusted Model 2 (Table 1).

Discussion

All of the models tested in this study showed an

TABLE 1—Cox Proportional Hazards Models of the Association between Psychotherapeutic Medicine Use (1981) and Time-dependent Covariates Illicit Drug Use and Psychiatric Symptoms in Baltimore, with Time Defined as Chronological Age (n = 1,537)

Variables	Model 1 (unadjusted for symptoms)	Model 2 (adjusted for symptoms)	Model 3 (interaction)	Model 4 (sequence of onset)
Illicit Drug Use				
Yes	2.65* (2.17, 3.21)	2.61 (2.15, 3.18)		
No	1.00	1.00		
Symptoms				
Yes		1.43 (1.18, 1.74)		
No		1.00		
Illicit Drug Use/Symptoms				
No/No			1.00	1.00
Yes/No			3.10 (2.33, 4.03)	3.07 (2.33, 4.03)
No/Yes			1.60 (1.26, 2.02)	1.60 (1.26, 2.02)
Yes/Yes			3.60 (2.69, 4.80)	
illicit first				2.64 (0.91, 7.72)
symptoms first				3.86 (1.79, 8.34)

*Relative hazards are adjusted for age and for fixed covariates sex, race, marital status, household income, and help-seeking. 95% confidence intervals in parentheses.

TABLE 2—Alcohol Intoxication-adjusted Cox Proportional Hazards Models of the Association between Psychotherapeutic Medicine Use (1981) and Time-dependent Covariates Illicit Drug Use and Psychiatric Symptoms in Baltimore (n = 1,528), with Time Defined as Chronological Age*

Variables	Relative hazards Model 1 (unadjusted for symptoms)	Relative hazards Model 2 (adjusted for symptoms)
Illicit Drug Use		
Yes	2.52 (2.04, 3.14)	2.52 (2.02, 3.11)
No	1.00	1.00
Symptoms		
Yes		1.41 (1.15, 1.71)
No		1.00
Alcohol Intoxication Age of Onset		
3–15 Years	1.28 (0.92, 1.79)	1.24 (0.88, 1.72)
16–20 Years	1.08 (0.80, 1.44)	1.05 (0.78, 1.41)
> 20 Years	1.10 (0.79, 1.54)	1.09 (0.78, 1.53)
Never Used	1.00	1.00

*Relative hazards are adjusted for age and for fixed covariates sex, race, marital status, household income, and help-seeking. 95% confidence intervals in parentheses.

association between illicit drug use and the later initiation of prescribed psychotherapeutic medication, even when controlling for initial occurrence of psychiatric symptoms. Symptoms did not change this relation. Model 4 showed that the risk of prescribed psychotherapeutic medicine use was elevated, whether illicit drug use preceded or followed onset of symptoms. Finaliy, adjustment of the models for the onset of alcohol intoxication did not alter these results.

Learned behavior[15] and sensation-seeking trait explanations[16] are both consistent with the finding that the association between illicit drug use and psychotherapeutic medicine use occurred independent of psychiatric symptoms, as drug use motivated by these explanations would take place without regard to distress state.[31] In terms of maturation and deviance,[13,14] the change in substances used, which may occur from adolescence to young adulthood, could be viewed as a move from behavior that is more deviant to a behavior that is more consistent with adult social roles.[32]

The results of this study are congruent with those of Yamaguchi and Kandel[6] in terms of the association between illicit drug use and psychotherapeutic medicine use, even though our study included a more diverse sample ranging in age from 18–44 years. Whether these results would hold elsewhere or at other times is a question of generalizability best addressed by replication and by greater attention to members of the population not typically interviewed in field surveys. As an example of how results might not generalize to other times, data were gathered in 1981, midway through the cocaine epidemic in the US. The possible impact of the epidemic on our findings is uncertain.

The key limitations to these findings relate to the retrospective character of the study data. For example, validity of self-reports of onset of psychological symptoms may be affected by many factors,[33,34] including severity of the symptoms, length of time since the symptoms were first experienced, and the number of episodes experienced over the lifetime. Similarly, the recency of use of the medicines and the amount of use,[35] as well as the indications for use (i.e., if nonpsychiatric) also could affect the accuracy of the findings.

In addition, the validity of the order in which drugs were used also is relevant. Evidence[36] supporting validity of self-reported order of drugs used comes from a series of repeated measures design surveys of drug use among high school seniors. The findings of these surveys consistently show that marijuana use precedes the use of other illicit substances. These surveys also corroborate findings of two studies[3,4] of sequencing of drug use which used retrospective lifetime reports. Nevertheless, some errors in this regard are possible, as are errors in ordering of onset of symptoms relative to drug use.

In conclusion, this study has shown a predictive association between illicit drug use and psychotherapeutic medicine use, independent of psychiatric symptoms. Future study of these relationships should include: 1) a design which would minimize memory/reporting errors, and 2) a more detailed examination of the etiologic mechanisms that might be responsible for this association.

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Independence Foundation Grant to Establish Nursing Chairs at Nine Universities

Nine of the nation's nursing schools will benefit from a $9 million grant to endow an Independence Chair for Nursing at each institution. The award—made by the Independence Foundation of Philadelphia—is the largest foundation grant in the history of nursing education.

Robert A. Maes, president of the Independence Foundation, said that the interest from the grant should allow nursing programs to engage in long-term planning that will ensure the future of nursing education. "Unlike medical programs," he said, "nursing programs have not been richly endowed and consequently funds for planning have been scarce." The grant from the Independence Foundation will increase the number of nursing chairs in the United States from 38 to 47. Recipients of the grants are nursing schools at the following institutions: Case Western University, Emory University, Johns Hopkins University, New York University, Rush University, University of Pennsylvania, University of Rochester, Vanderbilt University, and Yale University.

The Independence Foundation, established in 1961 in Philadelphia, originated from the Donner Foundation of New York and, over the years, has largely given to secondary education. Since 1985, the Foundation has focused on the national nursing shortage and, two years later, began extensive scholarship funding of each of the nine schools involved in the new grant. In a further effort to draw attention to the national nursing shortage, the Foundation recently announced an increase in those scholarships, as well as the generous endowment of nine chairs for nursing in universities across the nation.

Increasing Rubella Seronegativity Despite a Compulsory School Law

TIMOTHY R. SCHUM, MD, DAVID B. NELSON, MD, MSc, MATTHEW A. DUMA, MD, AND GERALD V. SEDMAK, PhD

Abstract: To determine if lower rubella susceptibility persisted five to seven years after immunization legislation, we retrospectively reviewed the serologic status of 341 outpatients from 1985 to 1987 in an inner-city school age population. Seronegative rates increased significantly during the two-year study period from 4.2 to 24.5 percent (17 percent overall). (β = 6.8%, 95% CI = 3.3, 10.3).

Charts were reviewed for 57 of 58 seronegative and 114 seropositive controls. Estimates were then made to the population of 341 subjects. Those with documented rubella immunization had a seronegative rate of 13 percent compared to 19 percent if the immunization status was unknown. For patients who received care in our clinic for < two years, 32 percent were seronegative compared to 10 percent for those treated ≥2 years (odds ratio = 0.31, 95% CI = 0.16, 0.60). Among patients immunized in 1977 or 1978, 33 percent were seronegative compared to 7 percent immunized at other times (OR = 0.21, 95% CI = 0.07, 0.63).

Factors associated with increased rubella seronegativity include immunization in 1977 or 1978 and lack of continuity of care in our clinic. Much of the increase remains unexplained. (*Am J Public Health* 1990; 80:66–69.)

Introduction

In an attempt to prevent congenital rubella syndrome (CRS), rubella vaccination of all 15-month-olds is recommended.[1] This strategy has interrupted the six-nine year cycle of rubella epidemics and cases of rubella have decreased by 98 percent since the prevaccine era of 1969.[2] Outbreaks of rubella still occur in the adolescent/young adult age group, however,[3–5] leaving a serious potential for CRS.

The reported prevalence of rubella susceptible adolescents varies from 7 to 24 percent.[6–9] Nelson, *et al*, showed a dramatic decrease in rubella susceptibility in inner-city adolescent and preadolescent females from 22 percent to 5 percent when a mandatory school immunization law went into effect.[10]

The purpose of the present study was to determine whether this improved serologic status persisted, and what factors affect rubella immune status.

Methods

The Downtown Health Center is an inner-city clinic in Milwaukee, Wisconsin providing general pediatric care. The patient population is 75 percent Black and 85 percent Medicaid. All girls at or near the age of 10 years presenting for well child care, or for school or sports physicals, are routinely screened for rubella susceptibility. We reviewed the results of these tests from August 1985 through July 1987 and compared them with tests done in 1980 and 1981 (the same population with clinic name changed).

On the initial screen, we collected each patient's name, age, date, and result of her rubella titer. All rubella hemagglutination inhibition (HI) titers were measured by the City of Milwaukee virology laboratory using kaolin treatment of serums and one-to-three-day-old chick red blood cells. Titers of <1:10 were considered seronegative.[11]

In an effort to identify factors associated with increased seronegativity, we performed a case control study. From the population of 341 patients with known rubella titers, we did a detailed chart review on a subset of 171 patients—57 of 58 seronegative patients (one chart was unavailable) and 114 seropositive patients. Two seropositive controls were randomly selected for each seronegative case. Information collected from these charts included history and date of rubella immunization, type of immunization, clinic at which immunization was administered, type of health insurance, length of residence in Milwaukee, and current zip code. Seronegative rates for the population of 341 based on time periods could be calculated directly. However, population seronegative rates based on other parameters (history of immunization, duration of care in our institution, year immunized, and clinic of immunization) could not be calculated directly. Estimates of these rates were calculated using the overall incidence rates, odds ratios, and relative frequency of risk factors in the seropositive and seronegative groups.[12] This estimate takes into account the proportion of the subset exposed to a particular variable, e.g., year immunized, and then provides an estimate for the population of 341.

Patients were considered to have a positive history of immunization if a specific immunization date was recorded in their medical record. Immunizations given in our clinic also had the day and site of injection documented.

Differences in rates were measured using the Mantel-Haenszel estimate of the relative risk (odds ratio) with test-based confidence intervals.[13] To control for possible confounding, logistic regression was used.[14] Time trends were determined using a test for linear trend in proportions, a method similar to linear regression except each proportion is weighted by the sample size on which it is based.[15]

Results

Three hundred forty-one patients had their rubella serologic status determined during the two-year study period. Overall, 58 (17 percent) were seronegative. The seronegative rate was 5.8 percent in 1981[10] and 4.2 percent in 1985. However, from 1985 to 1987 there was a consistent increase in rubella susceptible girls to 24.5 percent (Table 1), β = 6.8% (95% CI = 3.3, 10.3).

Subsequent analyses refer to the estimates made on the population of 341 after chart review of the 171 cases. Seventy of 171 cases had documented immunization; 37 of these were immunized at another clinic; 27 were immunized in 1977 or 1978 (Table 2). Children without documented vaccination history were no more likely to be seronegative than children with a documented vaccination history, i.e., 19 percent vs 13

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Editor's Note: See also related letter p 85 this issue.

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TABLE 1—Rubella Immune Status of Females by Six-Month Periods, AUGUST 1985–JULY 1987

Time	Rubella Status				
	Seronegative*		Seropositive		Total Patients
	Patients	%	Patients	%	
AUG 85–JAN 86	3	4.2	69	95.8	72
FEB 86–JUL 86	9	12.7	62	87.3	71
AUG 86–JAN 87	21	21.9	75	78.1	96
FEB 87–JUL 87	25	24.5	77	75.5	102
Totals	58	17.0	283	83.0	341
JUL 80–MAR 81**	15	5.8	242	94.2	257

*β = 6.8% [95% CI = 3.3, 10.3] 1985–87 data.
**Data from Nelson, *et al.*[10]

TABLE 2—Univariate Odds Ratios and 95% Confidence Intervals for Selected Variables and Rubella Antibody Status

Variable	N**	Rubella Antibody Status*	Odds Ratio	95% Confidence Intervals
		% Seronegative		
History of prior immunization				
Yes	70	13	.69	0.36,1.34
Unknown	101	19		
Duration of care in our institution				
≥2 yrs	117	10		
<2 yrs	54	32	0.31	0.16,0.60
Year immunized				
1977–78	27	33	0.21	0.07,0.63
All other years	43	7		
Clinic of immunization				
Other	37	18	0.89	0.31,2.51
DHC***	33	16		

*Estimated

$$\text{Incidence (not exposed)} = \frac{\text{Incidence (total population)}}{[(\text{Proportion exposed}) \times \text{Odds ratio}] + (\text{proportion unexposed})}$$

$$\text{Incidence (exposed)} = \text{Odds Ratio} \times \text{Incidence (not exposed)}$$

**N = 171 cases with secondary chart review
***Downtown Health Center, Milwaukee, Wisconsin

percent, respectively (Table 2). Patients immunized elsewhere had seronegative rates similar to those immunized at our clinic, 18 percent vs 16 percent, respectively. Children immunized at times other than 1977 or 1978 had lower seronegative rates than children immunized during 1977 and 1978, 7 percent vs 33 percent. Of the 171 patients, 117 were treated at our institution for greater than two years and showed a seronegative rate of 10 percent compared to a 32 percent rate for those treated here less than two years.

Of the 20 seronegative girls with documented immunization histories, 12 were immunized at 15 months or later, four between 12–14 months, and four at or near their first birthday.

Results from a logistic regression analysis are shown in Table 3. Only the following variables were simultaneously entered into the regression model: duration of treatment in our institution, clinic of vaccination, year vaccinated, and history of vaccination. Positive associations with rubella seropositivity were found for treatment in our clinic ≥2 years and for vaccination in years other than 1977–78.

The mean age for serosusceptible girls (11.9) was not significantly different from seroimmune girls (12.4). Type of immunization, e.g., combined measles-mumps-rubella, geographic location, insurance type, and length of time from immunization were not associated with rubella serologic status.

TABLE 3—Multiple Logistic Regression Analysis on Rubella Status

Variables	Odds Ratio	95% Confidence Intervals
Year vaccinated	0.17	0.06, 0.53
Clinic	0.75	0.24, 2.37
Time treated in our institution	0.27	0.15, 0.48
History of prior immunization	0.94	0.20, 7.80

Discussion

The sudden increase between 1985 and 1987 in seronegativity remains largely unexplained. But it is clear that improved rubella seropositivity associated with the school immunization law was not sustained in our population. The 17 percent prevalence of seronegativity in our 1985–87

population is similar to other reports,[7,8] in particular the 22 percent Nelson described prior to the institution of a statewide school immunization law.[10] Since July of 1987, the seronegative rate in our clinic has leveled off at 18 percent (54 of 300 from August 1, 1987 through January 31, 1989). The Milwaukee Health Department also noted an increase in rubella susceptibility in 1987, the highest level since 1979 (Table 4).

Several factors might explain increased seronegativity. One possible explanation would be failure to enforce the school immunization law. The school system, however, has reported no change in enforcement practices. Documentation of actual dates of immunization are and have been required for school entry. Another possible explanation is patient mobility and lack of continuity of care. The increased seronegativity in patients who have been attending our clinic for less than two years suggests this might have occurred. However, strict enforcement of the law should have identified these children.

Perhaps our 17 percent seroprevalence is biased or not generalizable because we could document immunization history in only 70 of 171 charts reviewed (41 percent). Seronegativity was 5.7 percent in Nelson's study even though they could document immunization history in only 46 percent of their patients,[10] making this an unlikely explanation.

Primary or secondary vaccine failure could explain the increased seronegativity, but our rate of 17 percent is higher than reported rates of 0–12 percent.[16–18,24,25] Secondary vaccine failure is unlikely as we found no association between seronegativity and time elapsed since immunization.

There was no evidence that the HI testing methodology explained the increased seronegativity. The HI test method, laboratory personnel, and titer cutoffs (1:8–1:10) have not changed. Newer testing methods are available and are more sensitive at detecting low antibody titers[21–23] but the standard for comparison still remains the HI test.

The current vaccine was licensed in 1979. We found significantly increased seronegativity for those immunized in 1977–78 but not before or after that time period. Perhaps there was a problem with particular lots of vaccine in those two years. Although problems with improper handling have been reported,[24,25] this is an unlikely explanation because seroprevalence rate was the same regardless of the clinic site.

Finally, the differences found might relate to selectively screening girls who are likely to be seronegative. This, however, is unlikely. The clinic clientele has not changed over the last 10 years. We see mostly Medicaid HMO (health maintenance organization) patients living in the inner-city and our referral patterns have not changed. All females age 8 or over presenting for well child care are eligible for screening. The vast majority are screened, although a few, known to be unvaccinated, will be immunized and not screened. Most patients are unaware of their rubella status and we do not require immunization records prior to screening. There is no reason to suspect this population was at increased risk for rubella seronegativity as most came to the clinic just for routine care rather than being referred for a specific problem.

Despite the increased seronegativity, we have not had sustained rubella transmission in Milwaukee since 1977 when a university-associated outbreak occurred. However, there was a mumps outbreak in the general population in 1987[26] and the possibility of a rubella outbreak exists. If the rate of serosusceptibility to rubella remains as it is, then a large number of women from this population may be entering their childbearing years, susceptible to rubella.

In summary, the dramatic impact of a mandatory school immunization law was not sustained seven years later. Rubella seroprevalence is now the same as it was prior to mandatory immunization laws although reported school enforcement has remained the same. Patients without a documented history of rubella immunization should be immunized as per current recommendations.[1] Females with prior rubella immunization should have rubella serology performed to assure immunity as documented history of rubella immunization did not guarantee seropositivity in our study.* The lack of continuity of care and rubella immunization during 1977–78 were significantly associated with increased seronegativity in our population. The sudden increase in seronegativity in just two years from 4 to 24 percent still remains largely unexplained but has leveled off at 17–18 percent. School enforcement of mandatory immunization laws is important but in Milwaukee is not sufficient to assure that girls approaching adolescence are protected against rubella.

TABLE 4—Rubella Serology from Specimens Submitted to the Milwaukee Health Department 1979–87

	Rubella Status		
	Seronegative	Seropositive	Total
Year	No. (%)	No. (%)	No.
1979	666 (13.7)	4190 (86.3)	4856
1980	472 (13.4)	3043 (86.6)	3515
1981	304 (12.8)	2071 (87.2)	2375
1982	114 (10.4)	985 (89.6)	1099
1983	140 (10.5)	1194 (89.5)	1334
1984	117 (11.6)	890 (88.4)	1007
1985	123 (12.9)	832 (87.1)	955
1986	107 (12.2)	768 (87.8)	875
1987	189 (22.0)	671 (78.0)	860
Totals	2232 (13.2)	14644 (86.8)	16876

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*See Letters section for recommendations of the Centers for Disease Control on this point.

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Noise and Hearing Loss is Focus of Upcoming NIH Consensus Development Conference

Noise and hearing loss is the subject of an upcoming consensus development conference, to be held January 22-24, 1990 at the Masur Auditorium of the National Institutes of Health Clinical Center, Bethesda, Maryland. In the background material provided by NIH, it was noted that:

- Approximately 28 million individuals in the United States have impaired hearing;
- Exposure to occupational and recreational noise has, in many instances, caused irreversible damage to the inner ear;
- Sources of potentially damaging noise include high-volume music, recreational vehicles, airports, heavy traffic, lawn mowers, household appliances, chain saws, and power tools, to name a few;
- Hazardous levels of noise on the job are experienced daily by firefighters, military personnel, disc jockeys, construction workers, musicians, farmers, factory workers, taxicab/bus/truck drivers, and others.

The NIH conference will bring together specialists in hearing, physiology, and pathophysiology and other relevant fields. The purpose of the conference is to reach agreement on noise and hearing loss. Experts will present current scientific thinking about the diagnosis, management, and prevention of hearing loss, and concerned voluntary organizations will be invited to make statements. On the basis of the scientific evidence presented, the consensus panel will prepare a draft report and invite comments.

Key questions to be addressed include: 1) What is noise-related hearing loss? 2) What sounds can damage hearing? 3) What factors, including age, determine an individual's susceptibility to noise-related hearing loss? 4) What can be done to prevent noise-related hearing loss? 5) What are the directions for future research.

To register for the noise and hearing consensus conference or to obtain further details, contact: Dina Rice, Prospect Associates, Suite 500, 1801 Rockville Pike, Rockville, MD 20852. Telephone: (301) 468-6555.

Public Health Briefs

Foreign-Born and US-Born Black Women: Differences in Health Behaviors and Birth Outcomes

HOWARD CABRAL, MPH, LISE E. FRIED, MSPH, SUZETTE LEVENSON, MPH, HORTENSIA AMARO, PHD, AND BARRY ZUCKERMAN, MD

Abstract: We studied health behaviors and birth outcome among 201 foreign-born and 616 US-born Black women receiving prenatal care at Boston City Hospital. Foreign-born women had better pre-pregnancy nutritional status and prenatal health behaviors, and their infants had greater intrauterine growth. Black women are not a homogeneous group; culture and ethnicity, in addition to other variables, must be considered in the study of their birth outcomes. (*Am J Public Health* 1990; 80:70–72.)

Introduction

In the United States, Black neonates are at high risk of low birthweight and increased perinatal mortality.[1–3] The rate of low birthweight among foreign-born Blacks is, however, two-thirds that of US-born Blacks.[4] The percentage of Black births in the US to mothers of foreign birth has increased in recent years. By 1986, this percentage had risen to 8 percent, an increase of 64 percent from 1976 and 39 percent from 1980, largely in urban areas of the northeast US.[5–7]

Differences in the rates of low birthweight among Blacks by foreign birth status may be related to lifestyle, since the health behaviors of those born outside of the US are different from those born in the US. For example, among Hispanics, those born outside of the US are less likely to smoke cigarettes, use illicit drugs, or receive late or no prenatal care than Hispanics born in the US, whose health behaviors more closely resemble those of US natives of other ethnic groups.[8–12,*]

The goal of this paper is to ascertain whether similar differences in health behaviors, and consequently birth outcomes, are present in a sample of 201 foreign-born and 816 US-born Black women receiving prenatal care at Boston City Hospital.

From the School of Public Health, Boston University (all authors) and the Division of Developmental and Behavioral Pediatrics, Boston City Hospital/Boston University School of Medicine (Zuckerman). Address reprint requests to Howard Cabral, MPH, Boston University School of Public Health, Data Coordinating Center, 85 East Newton Street, M-806, Boston, MA 02218-2389. This paper, submitted to the Journal February 1, 1989, was revised and accepted for publication July 3, 1989.

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*Amaro H, Whitaker R, Coffman G, Heeren T: Acculturation and Drug Use: Findings from the Hispanic HANES. Paper presented at the First National Conference of the National Hispanic Psychological Association, New York, August 1987.

Methods

As part of a study of maternal health, psychosocial characteristics, health behaviors, and pregnancy outcome initiated in 1984, subjects were consecutively recruited in the prenatal clinics of Boston City Hospital. Interviews were conducted in English or Spanish at the time of prenatal registration and in the immediate postpartum period. Information on each participant's race and country of birth was obtained from closed-ended items on the initial interview. Substance use was assessed by self-report, except for marijuana and cocaine, which were also identified through urine assays. Infant assessments were conducted by pediatricians blind to the hypothesis of the analysis.

The 817 Black women presented here are a subsample of the total study sample of 1,226 women who delivered a liveborn, singelton infant and on whom complete data, including two interviews, a maternal medical record review, and an infant assessment, were available. A more detailed description of the study protocol and population has previously been published.[13]

Results

The demographic characteristics of the 817 Black women presented here are comparable to those of the low-income (47 percent with a monthly income of less than $500, 51 percent currently receiving Medicaid), inner-city prenatal population served by Boston City Hospital. Eighty-two percent of these women were unmarried, 15 percent were 18 years of age or younger, and 36 percent had received less than 12 years of education. Of the 616 US-born women, 7 percent were first generation Americans. Of the 201 foreign-born Black women, 72 percent were from Caribbean countries (40 percent from Haiti, 11 percent from Jamaica, 21 percent from other Caribbean countries), 17 percent from Africa, 8 percent from Cape Verde, and 3 percent from England. Country of origin was not significantly associated statistically with the dependent variables used in this analysis among women of foreign birth.

Foreign-born women were more likely to be older, married, better educated, to have better pre-pregnancy weight-for-height, and to have had nine or more visits for prenatal care than those born in the US (Table 1). The two groups of women were similar on parity, trimester of first prenatal visit, and pregnancy weight gain of less than 16 lbs. They were also less likely to smoke cigarettes, drink alcohol, or use marijuana, cocaine, or opiates during pregnancy (Table 2); in addition, they used fewer cigarettes and consumed less alcohol (data not shown).

TABLE 1—Comparison of Foreign-Born US-Born Black Women on Demographic and Selected Obstetric Factors

Factors	Foreign-Born (n = 201)	US-Born (n = 616)	% Difference	95% CI of Difference
	%	%		
Maternal Age				
18 years or younger	7	18	−11	(−6, −16)
Marital Status				
Married	33	12	21	(14, 28)
Maternal Education				
11th grade or less	22	40	−18	(−11, −25)
Monthly Household Income*				
$500 or less	44	48	−4	(−13, 5)
Pre-Pregnancy Weight-for-Height				
Less Than 90% of Ideal	6	13	−7	(−3, −11)
Primiparity	47	47	0	(−8, 8)
Trimester of First Prenatal Visit				
First or Second	87	85	2	(−3, 7)
Prenatal Care				
Less than nine visits	33	47	−14	(−6, −22)
Pregnancy Weight Gain				
Less than 16 lbs	12	16	−4	(−9, 1)

* of the 81% reporting an income

TABLE 2—Comparison of Foreign-Born US-Born Black Women on Substance Use Anytime During Pregnancy

Substance Used	Foreign-Born (n = 201)	US-Born (n = 616)	% Difference	95% CI of Difference
Cigarettes	12%	53%	−41	(−35, −47)
Alcohol	45%	64%	−19	(−11, −27)
Marijuana*	6%	39%	−33	(−28, −38)
Cocaine*	3%	24%	−21	(−17, −25)
Opiates**	1%	4%	−3	(−1, −5)
Other Drugs	1%	2%	−1	(−3, 1)

*Determined by positive self-report and or urine assay
**Heroin, Methadone

As seen in Table 3 infants of the foreign-born women were, at birth, heavier, longer, had larger head circumferences, and had longer durations of gestation than did infants of the US-born women. Foreign-born Black women in this sample were also less likely to have given birth to a low birthweight (≤2500 grams) infant (odds ratio = 0.59, 95% CI = 0.33, 1.03) or a premature (<37 weeks gestation) infant (odds ratio = 0.46, 95% CI = 0.22, 0.94) than those born in the US.

After controlling for the effects of gestational age, weight gain during pregnancy, pre-pregnancy weight-for-height, marital status, maternal age, level of education, the number of visits made for prenatal care, and the use of cigarettes, alcohol, marijuana and cocaine during pregnancy, infants born to foreign-born Black women remained heavier, longer, and larger in head circumference at birth than infants born to US-born Black women (Table 3). Duration of gestation was not different between infants of foreign-born Black women and infants of mothers born in the US. The adjusted odds of having a low birthweight infant or a premature infant remained decreased for foreign-born Black women as opposed to Black women born in the US (odds ratio, low birthweight = 0.81, 95% CI = 0.42, 1.53; odds ratio, prematurity = 0.54, 95% CI = 0.24, 1.18).

TABLE 3—Differences in Fetal Growth Measured at Birth between Infants of Foreign (N = 201) and US-Born (N = 616) Black Women

	Foreign-Born vs US-Born		Foreign-Born vs US-Born	
	Unadjusted Difference	95% CI of Difference	Adjusted Difference	95% CI of Difference
Birthweight	272 g	(174, 369)	135 g	(51, 218)
Length	1.3 cm	(0.9, 1.8)	0.7 cm	(0.2, 1.1)
Head Circumference	0.8 cm	(0.5, 1.2)	0.4 cm	(0.1, 0.7)
Gestational Age	0.4 wk	(0.01, 0.7)	0.3 wk	(−0.1, 0.7)

Discussion

The infants of foreign-born Black women experienced greater intrauterine growth than those of Black women born in the US, even after accounting for many of the factors known or suspected to influence fetal growth. Why such a difference remained deserves more in-depth study than these data can provide.

The following limitations should also be considered when interpreting these findings:

- First, although the percentage of births to foreign-born Black mothers in these data virtually matches that of the entire northeast US (26 percent) in 1986,[7] it is possible that the characteristics of the women studied here are unique to the Black population who use Boston City Hospital as their primary source of prenatal care.
- Second, foreign birth status is an approximation of the cultural characteristics of these women and does not necessarily reflect the heterogeneity of either the US or foreign-born Black women in this regard.
- Third, the exclusion of non-English or non-Spanish-speaking women, who may have been less acculturated to US norms than those who participated, may have affected these findings.

These data illustrate some important differences in prenatal risk and birth outcome by foreign birth status within the prenatal population of Black women at Boston City Hospital. They suggest that low-income Black women are not a homogeneous group in terms of prenatal risk and birth outcome, and highlight the importance of considering ethnicity as indicated by place of birth, in addition to race, as a demographic factor in the study of the relationship of maternal prenatal health behaviors and birth outcomes.

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Some Sources of Error in the Coding of Birth Weight

ANDREW J. BRUNSKILL

Abstract: Three types of error in coding birth weight to computer tapes are described: 1) confusion of ounces with pounds, 2) mistaken reading of one pound as eleven pounds, and 3) errors in placement of the decimal. All will allocate low birth weight infants to high birth weight categories. Examination of the reported gestational age of the infant or of the reported cause of death may allow these errors to be detected. (*Am J Public Health* 1990; 80:72–73.)

Introduction

There is increasing use of computerized birth certificate records in pediatric epidemiology. Such records may contain both systematic and random errors. David described sources of systematic error in the recording of birth weight and gestational age in North Carolina birth certificates.[1] In that study, it was suggested that when birth weight was abnormally large for gestational age, gestational age was most likely to be inaccurate. However, original birth certificates were not actually compared to the birth tapes for accuracy of transcription. Although the need for careful editing of data is emphasized by experienced researchers, the nature and the effects of errors which occur in coding seem to have received less emphasis.

Methods

This analysis is based on a partial review of the cases present in a state birth tape of 1984–85 data. For those infants who had died within the first year of life, linked death certificates were also available. All White infants with birth weights of 4.5 kgs or greater ("macrosomic") were sampled and a corresponding random sample in an approximately 2:1 ratio of infants with birth weights 2.5–4.0 kgs. This selection, which related to the primary aims of a project on macrosomia, yielded 5,399 macrosomic and 10,250 control infants. Within these samples, the records of all those infants who had died in the first year of life were reviewed and the birth weight reported on the birth certificate compared with the birth weight reported on the computer tape. Additionally, 34 birth certificates of those infants with the highest birth weights who had not died were reviewed to bring the total number of birth certificates reviewed to 120.

Results

Within the high birth weight group, 39 infants were reported to have died within the first year of life. However, 18 cases of miscoded birth weight were detected in this group, leaving only 21 infants whose birth weights were confirmed as being high. Within the normal control group, 47 infants were reported to have died within the first year of life. The birth weights of all these infants were confirmed as correctly coded. All of the additional high birth weight infants who had survived the first year of life and whose certificates were checked had correctly coded birth certificates.

Three types of errors appeared to be responsible for the miscoding:

- One type of error is the confusion of ounces with pounds. Examples include 12 oz being coded as 5,443 grams (12 lbs), and 14 oz being coded as 14 lbs. This error occurred in nine of the 18 cases of detected coding error.
- The second type of error consisted of mistaken reading of birth weights of one pound or more as eleven pounds. The ease with which one pound can be misread as eleven pounds can be understood by considering the abbreviation used for one pound (1 lb) where the lower case letter "l" is mistaken for a second digit one "1." This is illustrated in Figure 1 which consists of a photocopy of the relevant part of the six birth certificates in which all six birth certificate weights were coded as eleven pounds. In fact, only the infant whose birth certificate is coded as "e" had a true weight of eleven pounds. This error occurred six times in the series.
- The third set of errors occurs when a decimal is wrongly placed in metric values. This includes cases such as 510 grams being mistakenly coded as 5100

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Infant Formula Promotion and the Health Sector in the Philippines

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Abstract: We use data collected in 1984, 1986, and 1988 from public and private health facilities in the Metropolitan Cebu area of the Philippines to assess effects of World Health Organization and Government of the Philippines Codes of Marketing of Breast Milk Substitutes on distribution of formula supplies. Distribution of free and low-cost infant formula declined drastically between 1986 and 1988. Industry compliance was almost complete. (*Am J Public Health* 1990; 80:74–75.)

Introduction

After nearly a decade of controversy on the role of infant formula promotion in changes in Third World breast-feeding behavior, the World Health Assembly adopted the International Code of Marketing of Breast Milk Substitutes on May 21, 1981. Most troublesome among these provisions was Article 6, paragraph 6, stating that "[d]onations or low-price sales to institutions or organizations of supplies of infant formula or other products within the scope of this Code, whether for use in the institutions or for distribution outside them, may be made. Such supplies should only be used for, or distributed to, infants who have to be fed on breast milk substitutes."[1]

The World Health Organization (WHO) Guidelines issued on April 10, 1986, defined "infants who have to be fed on breast milk substitutes" as those who have rare metabolic disorders, cannot suck, or are motherless through death or abandonment as well as those whose mothers have decided not to breast-feed, fully or partially, for whatever reason.[2] The World Health Assembly passed a resolution on May 16, 1986 urging governments to take action "to ensure that the small amounts of breast-milk substitutes needed for the minority of infants who require them in maternity wards and hospitals are made available through the normal procurement channels and not through free or subsidized supplies."[3]

On December 22, 1986, the Philippine Government passed its own code of infant food marketing (the GOP code),[4] banning the distribution of free and subsidized formula supplies to hospitals.

WHO in 1988 affirmed that the International Code, which permits free or subsidized supplies of infant formula for infants who have to be fed on breast milk substitutes, had not been modified by the 1986 World Health Assembly Resolution.[5,6]

While the International Code acknowledges a legitimate market for infant formula that includes mothers who cannot breast-feed or choose to bottle-feed, the National Code for Marketing of Breast Milk Substitutes of the Government of the Philippines is silent on the issue of procurement and forbids infant food manufacturers and distributors from giving free supplies to the general public, hospitals, and other health facilities and medical personnel except on request by, or with the approval of, the Department of Health (DOH). Approval by the DOH is given only for catastrophic occurrences such as floods or famine. Violators of the GOP Code face fines or imprisonment. Omission of any reference to procurement in the GOP code raises questions on how to obtain formula for infants unable to breast-feed and/or whose mothers are unwilling or unable to suckle them. We analyzed existing data to assess the effect of these different codes on hospital practices.

Methods

Between 1984 and 1988, as part of a larger study, we surveyed health facilities serving the Philippines' second largest metropolitan area (Cebu City) three times. During this period, the major infant food companies serving this market (American Home Products, Bristol-Myers, Nestlé, and Abbott) had accepted the WHO Code.

We collected data from 73 (1984), 78 (1986), and 72 (1988) Metropolitan Cebu health facilities, all providing prenatal and obstetrical care. These facilities include all public and private hospitals and a representative sample of clinics and health centers. Approximately 70 percent of these facilities are public (government) institutions; about one-fifth of the public facilities and three-fourths of the private facilities have on-site obstetrical services; the remainder provide home-delivery assistance.

We obtained information on whether facilities had received any infant formula, other milk, or food supplies in the three months preceding each survey from manufacturers of those products; the brand name and source of each sample received; how the facilities had used infant formula supplies; and their policies toward industry representatives. The 1988 survey asked additional questions on procurement practices particularly for higher risk infants such as premature babies confined in special care nurseries.

Results

The proportions of facilities receiving as manufacturers' gifts any kind of milk remained basically unchanged between 1984 and 1986 but fell in the following two years (Table 1): infant formula distribution declined by 95 percent, while the proportion of facilities with formula for older infants increased by 80 percent in the same period. These foods intended for use as the liquid part of the weaning diet are not covered by the WHO or GOP codes.* The same applies to any weaning food.

Before passage of the GOP code, public facilities with on-site delivery were most likely to receive free formula (Table 2). All on-site delivery facilities show an increase in

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*Editor's Note: "Follow-up" formulas have not been marketed successfully in the United States. A policy statement of the American Academy of Pediatrics, "Follow-up or Weaning Formulas," appears in the February 1989 issue of AAP News.

TABLE 1—Percentage of Facilities Receiving Any Infant Food as Gifts by Type

Type of Food	1984	1986	1988
Infant formula	57.5	52.6	2.8
Follow-up formula	11.0	15.4	27.8

distribution of formula supplies between 1984 and 1986 by all manufacturers. In the 1988 survey we find only one manufacturer still distributing free formula supplies. Recipients were two private health facilities with on-site delivery which had received a soy-based preparation.

In 1984 and 1986, over half the total sample of facilities had distributed free samples to mothers; in 1988, only 3 percent still did so (data not shown).

The proportions of facilities allowing representatives to visit their patients rose from 30 percent to 40 percent from 1984 to 1986. In 1988, only 2.6 percent of the facilities allowed them to do so. Moreover, only seven of 72 facilities in Metropolitan Cebu still supplied clients' names to industry representatives—a marked decrease from previous years.

The 1988 survey found that seven private facilities sold infant formula in their own hospital pharmacies, an increase from two in 1984 and five in 1986. Bottle-feeding mothers who deliver in these private facilities have a choice of purchasing formula from these pharmacies or from outside sources. Mothers delivering in public hospitals have to buy formula from outside retail outlets if they need or want to bottle-feed their newborns.

Discussion

The trends shown over these three surveys suggest that the WHO International Code of 1981 had little direct influence in reducing the level of infant formula donations to hospitals which the Code did not prohibit.

TABLE 2—Percentage of Facilities Receiving Free Infant Formula Supplies from Specific Industry Sources by Facility Type and Year of Survey[a]

	1984		1986		1988	
Facility Type	%	(N)	%	(N)	%	(N)
Public facility						
With on-site delivery	85.7	(7)	100.0	(7)	0.0	(6)
Without on-site delivery	54.4	(46)	38.0	(50)	0.0	(46)
Private facility						
With on-site delivery	78.6	(14)	93.3	(15)	13.3[b]	(14)
Without on-site delivery	0.0	(6)	16.7	(6)	0.0	(6)

[a]Formula supplies were received within a three-month period preceding each survey.
[b]Soy-based formula only.

While committing itself to eliminating indiscriminate distribution of infant formula supplies, industry took the position that the responsibility of providing directives to health care facilities and manufacturers on infant formula promotion rests with national governments. We observed significant decreases in such activities in 1988 which may be attributable to restrictions and penalties of the National Code passed by the Philippine Government late in 1986.

Whether the changes in the distribution of free supplies observed in the 1988 survey will lead to an increase in breast-feeding among Filipino mothers and an improvement in infant health and development is still unknown. What is apparent is that present policies and practices are designed to make it inconvenient and difficult for mothers to bottle-feed in hospitals.

Finally, it is important to note that this study does not imply that the WHO Code has been of no value. The WHO Code has served as the basis for the development of country codes and has centered extensive publicity on the need to use legislation as one means of promoting breast-feeding. The GOP Code is noteworthy in being one of the first to implement the purpose of the 1986 WHO resolution.

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A Randomized Evaluation of Smoking Cessation Interventions for Pregnant Women at a WIC Clinic

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Abstract: Pregnant smokers attending a local health department WIC clinic were randomly assigned to one of two self-help smoking cessation programs or usual care. The multiple component program resulted in larger quit rates than usual care during the last month of pregnancy (11 percent vs 3 percent) and postpartum (7 percent vs 0 percent). Achieving quit rates in WIC similar to those in studies conducted at prenatal care settings, suggests that smoking cessation programs for low-income pregnant WIC clients are feasible. (*Am J Public Health* 1990; 80:76–78.)

Introduction

Estimates suggest that 32 percent of all women of child-bearing age smoke with only 21 percent quitting during pregnancy.[1] Evaluations of self-help cessation programs have demonstrated some success with pregnant women receiving prenatal care at an HMO (health maintenance organization),[2] private practices,[3] hospital clinics,[4*] and local health departments.[5]

WIC (the Special Supplemental Food Program for Women, Infants, and Children) is an unexplored setting for smoking cessation research. Analyses of matched WIC-birth certificate data for Missouri in 1982 indicated that 45 percent of pregnant women WIC participants were smokers,[**] 50 percent higher than the 30 percent rate observed among all Missouri women that year. Data on 127,512 pregnant women enrolled in several state WIC programs from 1979 through 1985 suggested that smokers were 2.5 times more likely to deliver a low birthweight infant, and that very few participants stopped smoking following their enrollment in WIC.[***]

The present investigation is a randomized trial of a self-help smoking cessation program implemented at a local health department WIC clinic

Method

The study was conducted from August 1985 to August 1986 at the Kent County Health Department in Grand Rapids, Michigan. A brief structured interview was used to characterize an incoming pregnant women as a current smoker (at least one cigarette per day), a nonsmoker, or a former smoker. Current smokers were asked to participate in the study and to complete an informed consent statement and a pretest. Following informed consent, participants were randomly assigned to one of three groups.

The usual care (UC) group received printed information about the risks of smoking during pregnancy and completed the clinic in the traditional manner.

The multiple component (MC) group received a 20-minute one-to-one counseling session which included both risk information and behavior change components. The risk information component employed the *Because I Love My Baby* materials developed by the American Lung Association. These materials included a "flip chart" used by the health educator in presenting the information and a printed brochure given to clients to take home.

The behavior change component of the MC intervention employed a self-help manual adapted from Windsor, *et al*,[5] and from the American Lung Association's *Freedom From Smoking* program. Behavioral contracting and self-monitoring were the primary strategies. An individual behavioral contract was developed during the session which specified a quit date and selection of some significant other as a co-signer. Self-monitoring included charts for recording daily smoking behavior, and the development of an individualized plan of action for breaking recorded behavioral chains.

The risk information (RI) intervention was a face-to-face session of about 10 minutes duration. The health educator used the same "flip chart" as the MC group and provided the factual brochures, but did not present behavior change counseling, or furnish the self-help manual.

During the postpartum WIC visit, the posttest was completed, which included questions about smoking during the final month of pregnancy as well as postpartum. The postpartum self-reports, on average, occurred 4.7 weeks following birth of the infant, a relatively short period. Saliva samples (N = 66) were collected from the last one-third of the participants. The correlation between the saliva thiocyanate and self-report measures (r = .25) was similar to correlations found in more extensive analyses with larger samples that showed excellent sensitivity and specificity.[6,7] Saliva samples were equally distributed across groups (28 percent of MC participants, 30 percent of RI participants, and 32 percent of UC participants). Resource constraints did not allow collection of more complete data.

Demographic and health service data were abstracted from WIC program records for study participants and for the women who refused to participate. Agreement rates between different coders, and between the WIC record and prenatal care medical record, for a 30 percent random sample, were 99.2 percent and 87.9 percent, respectively.

Results

Two hundred and seventy-one of 641 pregnant women entering the clinic were identified as current smokers (42 percent); 219 volunteered to participate in the study (81 percent). Of these 219, posttest data were collected from 186 (15 percent attrition rate) (about one-fourth actually refused to complete the posttest; three-fourths either moved from the community, left the

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WIC program, or miscarried). Those lost to follow-up were treated as continuing smokers in the following analyses.

Differences between study participants and refusals on variables available from the WIC record were relatively minor for important variables as were study group differences (Table 1).

Table 2 presents the quit proportions and differences by treatment group for both follow-up periods. The MC group quit rate was higher than the UC group at both post time periods. While all UC participants had returned to smoking postpartum, some proportion of the participants in the MC and RI groups remained smoke free.

Discussion

The 42 percent smoking rate among pregnant women entering the WIC clinic was similar to the estimates of Land and Stockbauer,** and was indicative of the important need for smoking intervention at WIC clinics. The quit rate for the MC group (11 percent) was just under that found at prenatal care settings serving low-income women. Both Windsor, *et al*,[5] and Secker-Walker *et al*,* in studies conducted at low-income public maternity clinics, reported quit rates of 14 percent.

Although limitations of the present formative evaluation study include small sample size, and lack of complete biochemical verification, the findings have preliminary implications for extending health education services to pregnant smokers visiting WIC clinics. The high level of program acceptance among our WIC participants (81 percent) is impressive. Although prenatal care settings see pregnant women more regularly, offering the possibility of multiple health education contacts (and potentially improved quit rates), the WIC program, with broader eligibility than typically found at public prenatal care clinics, would seem to offer greater opportunity to reach a larger share of low-income women. Additionally, WIC clinics offer the advantage of a centralized site where health educators and public health nurses can be made available to provide one-to-one counselings.

TABLE 1—Comparison of Multiple Component, Risk Information, Usual Care, and Refusal Groups on Selected Demographic and Health Care Variables

Variables	Multiple Component (n = 72)	Risk Information (n = 70)	Usual Care (n = 77)	Total (n = 219)	Refusals (n = 52)
Mean Age	22.3	23.4	22.4	22.7	23.8
Parity					
% Primiparous	33.8	27.9	23.4	28.2	21.2
% Multiparous	66.2	72.1	76.6	71.8	78.8
Race					
% Black	18.3	25.0	19.5	20.8	18.9
% White	78.9	75.0	71.4	75.0	75.5
% Other	2.8	0.0	9.1	4.2	5.7
Trimester Prenatal Care Began					
% First	88.7	80.3	82.7	84.0	NA
% Second	11.3	18.2	16.0	15.1	NA
% Third	0.0	1.5	1.3	0.9	NA
Trimester of First WIC Clinic Visit					
% First	22.5	27.5	19.5	23.0	23.1
% Second	54.9	44.9	48.1	49.3	50.0
% Third	22.5	27.5	32.5	27.6	26.9
Source of Payment					
% Insurance or HMO	14.5	9.1	21.0	15.1	8.5
% Medicaid	79.0	80.0	71.0	76.5	80.9
% Self-pay or other	6.5	10.9	8.1	8.4	10.6
Received Advice from Physician to Quit					
% Yes	68.6	67.1	68.9	68.2	NA
% No	31.4	32.9	31.1	31.8	NA
Mean Number of Years Smoking	7.2	7.5	7.3	7.4	NA
Mean Age Smoking Began	15.1	15.8	15.1	15.3	NA
Number of Quitting Attempts Prior to Pregnancy					
% None	26.4	34.3	28.9	29.8	NA
% One or more	73.6	65.7	71.1	70.2	NA
Number of Quitting Attempts Following Pregnancy					
% None	69.4	64.3	66.2	66.7	NA
% One or more	30.6	35.7	33.8	33.3	NA
Mean Number of Cigarettes per Day Prior to Pregnancy	19.9	19.7	20.3	19.9	NA

NOTE: All differences between MC, RI and UC groups are not statistically significant at the .05 level using chi-square tests for categorical variables and ANOVA for differences between means. Differences between the total intervention group and the refusal group are also not significant. Sample size may vary slightly due to missing data. NA = not available from WIC record.

TABLE 2—Quit Proportions and Differences at the Ninth Month of Pregnancy and Postpartum

	At Ninth Month	Postpartum
Multiple Component (N = 72)	.111 (.039, .183)	.069 (.010, .128)
Risk Information (N = 70)	.071 (.011, .131)	.071 (.011, .131)
Usual Care (N = 77)	.026 (−.009, .062)	.000 (0.00, 0.00)
Total (N = 219)	.068 (.035, .101)	.046 (.018, .074)

Note: 95% CI in parentheses

	Difference	95% CI
At Ninth Month		
MC vs UC	.085	(.004, .166)
RI vs UC	.045	(−.025, .115)
MC vs RI	.040	(−.054, .134)
Postpartum		
MC vs UC	.069	(.011, .128)
RI vs UC	.071	(.012, .130)
MC vs RI	.002	(−.082, .086)

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Eight-Year Follow-Up Results of an Adolescent Smoking Prevention Program: The North Karelia Youth Project

ERKKI VARTIAINEN, MD, PHD, UNTO FALLONEN, PHD, ALFRED L. MCALISTER, PHD, AND PEKKA PUSKA, MD, MPOLSCI

Abstract: In the North Karelia Youth Program five to 10 classroom sessions over two years taught skills to resist pressures to start smoking to 13 to 15 year old students. Compared to students from comparison schools, the treatment groups reported less smoking immediately after the intervention and in a four-year follow-up survey. At the eight-year follow-up, there was consistent evidence of possible preventive effects only among those who had been nonsmokers when the program began. (*Am J Public Health* 1990; 80:78–79.)

Introduction

From 1978 to 1980, a study to demonstrate methods for reducing cardiovascular risk factors in 13-year-old students was carried out in the province of North Karelia, Finland. One of the main aims was to prevent the onset of smoking.[1] Although many studies of the effectiveness of persuasive and behavioral approaches to smoking prevention have been carried out,[2] long-term follow-up is rare.[3,4] Details of the methods and the outcome of the intervention immediately after the program[1], after half a year[5], and two years after the program[6] are reported elsewhere. This brief report describes the follow-up results eight years after the program ended.

Methods

In brief, three pairs of matched schools were chosen for the program. In two "direct" program schools, 10 sessions were carried out during a two-year period from 1978 to 1980 by the project health educator and trained peer leaders. In two "county-wide" program schools, five behavioral sessions were to be carried out by health education teachers at the schools who were trained as part of an effort to disseminate new curricula throughout North Karelia. Two schools were chosen for controls from another province in eastern Finland. In each matched pair, one school was chosen from the capital of the county and the other from a rural village. The program began in 1978 among the seventh graders and finished with the ninth grade in autumn 1980. During the persuasive-behavioral sessions, students were taught about

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social pressures that may lead to smoking (peer, adults, and mass media) and were trained to deal with such pressures through role playing and other active learning techniques. Older peer leaders assisted in implementing the program at school. The short-term and long-term health hazards were also discussed during the program. At the pretest, there were 897 students in the seventh grade in the three pairs of matched schools. At the posttest, the participation rate varied from 92–97 percent, in the eight-year follow-up from 72–79 percent. In all the surveys, the smoking habits of students were asked by the following question: Do you smoke now? Multiple choice responses: 1) Not at all; 2) Less than once a month; 3) One to two times a month; 4) One–two times a week; 5) Daily.

Results

When the youth were in the seventh grade, the proportion who reported smoking at least once a month varied in schools from 4–9 percent. Immediately after the program, there were one-third fewer smokers in both types of intervention schools compared to control schools. One half year and two years later, the difference was about the same, although smoking increased during the follow-up. In 1986, six years after the program had terminated, an increase in smoking between ages 16 and 21 was seen (Figure 1). The differences between the direct program (38 percent smokers) and reference schools (42 percent smokers) were minimal, while the differences between the county-wide program and control schools were more impressive (31 vs 42 percent). When only those students who reported they were nonsmokers in the pretest were included in the analyses, the proportion of nonsmokers was lower in both the direct and teacher-led schools than in the controls at the eight-year follow-up (Table 1). If attrition is taken into account by assuming all dropouts to be smokers, smoking rates in the two treatment groups are more similar (48–49 percent) but they remain

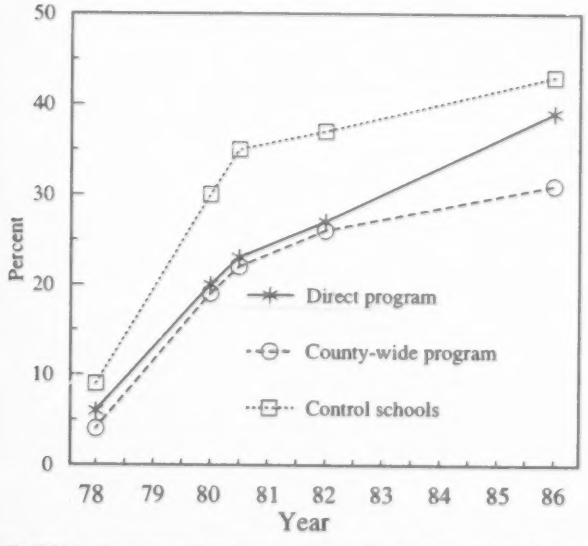

FIGURE 1—Proportion of Students Who Reported Smoking at least Once a Month in Different Surveys

TABLE 1—Proportion of Smokers (any smoking) by School Group in the Surveys among Nonsmokers in the Beginning of the Program

	Direct Program (%) (N)	County-wide Program % (N)	Control Schools % (N)	Difference (95% CI) Direct vs Control %	Difference (95% CI) County-wide vs Control %
Pretest	0 (249)	0 (243)	0 (251)		
Posttest	19 (248)	19 (242)	29 (250)	10 (3,17)	10 (3,17)
Two-and-one-half-year follow-up	23 (213)	22 (216)	38 (231)	15 (7,23)	16 (8,24)
Four-year follow-up	28 (230)	26 (225)	41 (238)	13 (4,21)	15 (7,23)
Eight-year follow-up	37 (200)	31 (182)	47 (209)	10 (1,19)	16 (6,25)

CI = confidence interval

lower than the rate of smoking in the campaign group (59 percent). This adjustment probably overestimates smoking prevalence.

Discussion

The interpretation of these results is complicated by several factors. Implementation evaluation of the project indicated that students at the urban school in the county-wide condition received only one or two anti-smoking sessions as part of the regular health education curriculum. The urban junior high school in the control condition shared its school yard with a senior high school whose students were allowed to smoke in the school yard. The primary difference that was observed, lower smoking rates, could be attributed to the overall community program in North Karelia rather than the specific programs that were carried out in the study schools. Given these qualifications, we cannot conclude from this study that preventive programs at school have long-term effects on smoking. On the other hand, our findings are consistent with the hypothesis that community and school programs can influence youthful nonsmokers to avoid tobacco.

ACKNOWLEDGMENTS

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The Effects of Detoxification of Domestic Gas on Suicide in the United States

DAVID LESTER, PHD

Abstract: As domestic gas was detoxified in the United States, the rate of suicide by domestic gas decreased. During this time period (1950–60), there was a parallel increase in the per capita ownership of cars and an accompanying increase in the rate of suicide by motor vehicle exhaust. However, displacement of suicide method from domestic gas to car exhaust occurred only for males and not for females. (*Am J Public Health* 1990; 80:80–81.)

Introduction

It has been argued that the detoxification of domestic gas in England and Wales in the 1960s and early 1970s was responsible not only for a drop in the rate of suicide by domestic gas, but also for a drop of about one-third in the overall suicide rate.[1,2]

However, it has also been noted that detoxification of domestic gas in Scotland and The Netherlands was not accompanied by a decrease in the overall suicide rate. Clarke and Mayhew[3] have suggested that this reflected a rising suicide rate in Scotland and The Netherlands at the time of detoxification which masked any reduction made possible by the detoxification of domestic gas.

Lester[4] has argued that, in the states of the USA where the availability of firearms is reduced by strict gun control laws, there is an accompanying reduction not only in the rate of suicide by firearms, but also in the overall suicide rate. Rich and Young[5] have shown that in Toronto people switched from using firearms to jumping in front of subway trains, leaving the suicide rate unchanged.

Domestic gas in the United States contained carbon monoxide in the early part of this century. The carbon monoxide content was reduced gradually during the 1940s and 1950s as gas companies switched to natural gas. The present paper examines the relation of the detoxification of domestic gas with rate of suicide.

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During this same period, the per capita ownership of cars increased in the USA. The per capita ownership of cars in the states of America is positively associated with the use of car exhaust for suicide.[6] The present paper, therefore, also explored whether people used car exhaust rather than domestic gas as a suicide method as the relative availability of these two methods for suicide changed.

Method

Data on the rates of suicide by domestic gas and motor vehicle exhaust were obtained from the annual volumes, *Vital Statistics of the United States*, published by the National Center for Health Statistics. Data on the percentage of customers receiving natural gas were obtained from the annual volume *Statistical Abstract of the United States*, published by the Bureau of the Census. Data on the number of cars per capita each year were obtained from *Historical Statistics of the United States from Colonial Times to 1970* published by the Bureau of the Census.

Data were obtained each year from 1946, the first year for which data on domestic gas customers could be obtained using a consistent set of categories, to 1970, by which time almost all domestic gas customers were receiving natural gas. Suicide by natural gas is possible, but since natural gas is less toxic, suicide is by the exclusion of air containing oxygen and is less apt to occur.

Results

Table 1 shows that the percentage of domestic gas customers receiving natural gas increased from 1950 to 1970 (regression coefficient = 22.75, standard error = 1.86), as did the number of cars per capita (regression coefficient = 8.90, standard error = 0.22). The accidental death rate from domestic gas was strongly and positively related to the percentage of customers receiving the toxic manufactured or mixed gas and with the rate of suicide by domestic gas.*

The rate of suicide by domestic gas decreased over the years (regression coefficient = −34.70, standard error = 4.12), while the rate of suicide by motor vehicle exhaust increased (regression coefficient = 29.45, standard error = 2.52). The sum of these two suicide rates remained constant

*Complete tables of data are available on request to author.

TABLE 1—Suicide Rates 1950–70, using Domestic Gas and Motor Vehicle Exhaust and the Extent of Use of Natural Gas by Consumers

			Suicide Rates per 100,000 per Year by:				
	Cars per Capita	% of Customers Using Natural Gas	Domestic Gas	Motor Vehicle Exhaust	Total Gas/ Exhaust	All Other Methods	Overall
1950	0.26	60.8	0.73	0.60	1.33	9.93	11.26
1955	0.31	81.6	0.19	0.74	0.93	9.17	10.10
1960	0.34	92.8	0.10	0.91	1.01	9.53	10.54
1965	0.39	98.0	0.08	1.09	1.17	9.90	11.07
1970	0.43	98.6	0.02	1.02	1.05	10.40	11.45

TABLE 2—Changes in the Suicide Rates 1950–70, by Method for Men and Women

	Suicide Rates by Men				Suicide Rates by Women			
	Motor Vehicle Exhaust	Domestic Gas	Total Gas	Other Methods	Motor Vehicle Exhaust	Domestic Gas	Total Gas	Other Methods
1950	1.09	0.85	1.94	15.75	0.12	0.61	0.73	4.32
1955	1.29	0.23	1.52	14.33	0.21	0.16	0.37	4.18
1960	1.55	0.13	1.68	14.78	0.29	0.07	0.36	4.59
1965	1.74	0.11	1.85	14.69	0.51	0.06	0.57	6.04
1970	1.48	0.03	1.51	15.30	0.61	0.01	0.62	5.95

during this period (regression coefficient = −3.81, standard error 6.67). The rates of suicide by domestic and motor vehicle gas were negatively related (r = −0.94). The total suicide rate during this period did not change systematically (regression coefficient = 1.84, standard error = 15.56).

The data were examined separately for men and women (Table 2). The use of domestic gas for suicide by men declined from 1950 to 1970 while the use of car exhaust increased. The use of both methods varied greatly from year to year, but the data suggest a slight decrease over the period. For women, the results are similar, except that the data suggest a drop in the use of domestic gas and car exhaust from 1950 to 1960 followed by a rise from 1960 to 1970.

Discussion

It has often been argued that removal of one lethal method for suicide will simply cause would-be suicides to turn to alternative methods.[7] The present results indicate that as domestic gas was detoxified and as per capita ownership of cars increased, fewer people used domestic gas for suicide while more used motor vehicle gas for suicide.

The situation is considerably more complicated, of course. During this period (1950 to 1970), the availability and use of firearms for suicide was increasing,[8] as perhaps was the availability of lethal medications such as antidepressants. Thus, as domestic gas became less toxic, several other methods for suicide became more available. This, together with the lesser use initially of domestic gas for suicide in the US as compared to England and Wales, is perhaps why detoxification of domestic gas in the US had little impact on the overall suicide rate, while in England and Wales detoxification of domestic gas was accompanied by a one-third reduction in the overall suicide rate.[7]

The data are not clear on whether switching occurred from domestic gas to car exhaust as the relative availabilities of these methods changed. A case could be made for or against switching, depending upon which time period (1950–60 or 1960–70) and which sex is examined. Two important questions for future research are: "Which people will switch methods for suicide if one is made less available?" and "To which methods will people switch if one method is made less available for suicide?"

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NIA Funds Three New Alzheimer's Disease Research Centers

The National Institute of Aging (NIA) has announced funding of three new Alzheimer's Disease Research Centers (ADRCs), with $2.3 million for the first year of operation and a total of $11 million over the next five years. This brings to 15 the number of Alzheimer's centers funded by the NIA. The purpose of the ADRC program is to enhance research on Alzheimer's disease by providing the resources and environment for collaborative, multidisciplinary studies. Each center will serve as the site for new and expanded studies of the basic, clinical and behavioral aspects of Alzheimer's disease. In addition to their research mandate, the centers train scientists and health care professionals and offer consultation and education to Alzheimer patients and their families.

The three new centers are located at Baylor College of Medicine, Houston, Texas; Columbia University, New York City; and the University of Michigan, Ann Arbor.

The other 12 NIA-funded centers are located at: Duke University, Durham, NC; Harvard Medical School/Massachusetts General Hospital, Boston, MA; Johns Hopkins Medical Institutions, Baltimore, MD; Mt. Sinai School of Medicine/Bronx VA Medical Center, New York; University of California-San Diego, La Jolla, CA; University of Kentucky, Lexington, KY; University of Pittsburgh, Pittsburgh, PA; University of Southern California, Los Angeles; Case Western Reserve University, Cleveland, OH; University of Washington, Seattle, WA; Washington University, St. Louis, MO; and University of Texas, Southwestern Medical Center, Dallas, TX.

Variables Influencing Condom Use among Intravenous Drug Users

STEPHEN MAGURA, PHD, JANET L. SHAPIRO, MA, QUDSIA SIDDIQI, PHD, AND DOUGLAS S. LIPTON, PHD

Abstract: Correlates of condom use were identified using cross-sectional data from a convenience sample of 211 sexually active intravenous drug users enrolled in methadone maintenance in New York City. Sixty-eight percent did not use condoms at all in the previous month and only 11 percent used condoms every time. Nineteen percent were planning on conception, only 20 percent of whom had been tested for human immunodeficiency virus (HIV) antibody (all seronegative). Multiple logistic regression analysis indicated that condom use was independently associated with greater personal acceptance of condoms, greater partner receptivity to sexual protection, and recent entry to methadone treatment. *Am J Public Health* 1990; 80:82–84.)

Introduction

Thirty percent of adult AIDS (acquired immunodeficiency syndrome) cases diagnosed in the United States during 1988 involved intravenous drug users (IVDUs).[1] In New York City, at least 50 percent of IVDUs are already HIV (human immunodeficiency virus) seropositive.[2,3] There has been extensive public health focus on reducing needle-sharing and other drug-use-related HIV transmission risks within the IVDU population.[4] However, sexual contact is an important secondary means of HIV transmission among IVDUs and is the main route of HIV transmission to the general heterosexual population.[5,6] Heterosexual contact accounted for 5 percent of US adult AIDS cases diagnosed during 1988.[1] Infected IVDU women are also the primary source of perinatal HIV transmission.[7]

Condoms are an accepted measure to prevent the sexual transmission of HIV; they also can be a useful although not fully effective birth control method for seropositive women.[8–10] One recent study found that only 13 percent of IVDUs used condoms (usually inconsistently) and that 65 percent of their sexual contacts were with non-IVDUs; the sample was drawn from a treatment population with 57 percent HIV seroprevalence.[11] Overall there has been little or no change in sexual practices in response to the AIDS threat either among IVDUs[12–14] or the general heterosexual population.[6] We present a quantitative analysis of the factors influencing IVDUs' decisions about condom use.

Methods

The study sample is 289 IVDUs enrolled in methadone maintenance who had volunteered for an AIDS prevention demonstration/research project at three New York City clinics. Because participants were self-selected, they may not be entirely representative of the clinic populations. This analysis involves the 211 subjects who had reported sexual activity within the previous month. The data are derived from baseline (pre-intervention) questionnaires completed between January 1987 and February 1988. The self-administered questionnaire consisted of fixed-response, Likert-type items and was available in both English and Spanish. Research staff were present on site to assist subjects who needed help in completing the instrument. Confidentiality was guaranteed; the results were not shared with methadone clinic staff. Subjects were paid $20 in two of the clinics and $10 in the third for completing the questionnaire. The questionnaire included measures of knowledge about AIDS and its transmission; drug use and needle sharing; sexual activities and condom use; and attitudes and beliefs related to condom use.

The dependent variable was condom use, measured by the item: "In the last month, how often did you (or your partner) use a rubber (condom) for sex?" ("Never," "sometimes," "most times," or "every time"). Responses to this question were reasonably internally consistent; subjects reporting at least some use usually disagreed with a different, negatively phrased item on condom use: "I almost never have sex using rubbers (condoms)" ($r = -.70$). For the analysis condom use was dichotomized as no use vs some use in the last month. The independent variables were drawn from theory and research on the determinants of condom use in the general population[6] and among gay/bisexual men.[15]

Results

Sixty-eight percent of the subjects did not use condoms at all in the previous month and only 11 percent used condoms every time for sex. Table 1 presents a multiple logistic regression on condom use. Variables independently related to condom use were: greater personal acceptance of condoms, greater partner receptivity to sexual protection, and recent entry to methadone treatment.

Discussion

The low rate of condom use among these subjects is consistent with findings from other surveys of addicts,[11,16] gay/bisexual men,[15] and the general population.[6]

The multivariate results indicate that norms and expectations within sexual partnerships exert the primary influences on condom use for these IVDUs. Condom acceptability (believing that condoms do not impair enjoyment of sexual relations and being willing to use condoms when requested by a sexual partner) was the variable most strongly associated with condom use. Perceived partner receptivity to suggestions of condom use and other sexual risk reduction was also directly related to condom use. That is, when individuals are amenable to condom use, and when sexual partners effectively communicate reciprocal willingness, there is greater likelihood of condoms being used in that relationship.

Beliefs, attitudes, and communication skills involving condoms can be addressed by methods such as cognitive-behavioral group work with addicts and, when possible, with their sexual partners.[17] In groups participants who already know that condoms do not interfere with sexual pleasure can encourage or challenge others to verify this for themselves; experience with condoms is likely to decrease resistance to

From Narcotic and Drug Research, Inc. Address reprint requests to Stephen Magura, PhD, Deputy Director of Research, Narcotic and Drug Research, Inc., 11 Beach Street, New York, NY 10013. Ms. Shapiro is a Research Associate, Dr. Siddiqi is an Assistant Project Director, and Dr. Lipton is Director of Research at NDRI. This paper, submitted to the Journal February 27, 1989, was revised and accepted for publication June 26, 1989.

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TABLE 1—Multiple Logistic Regression on Condom Use

Variables	(N)	Percent Condom Use	Odds Ratio (95% Confidence Intervals)
Gender			
Male	(132)	31	1.00
Female	(79)	33	1.16 (.74, 1.84)
Ethnicity			
White[a]	(144)	31	1.00
Black	(31)	39	1.47 (.85, 2.55)
Hispanic	(36)	31	.96 (.54, 1.69)
Age (years)[b]			1.30 (.91, 1.84)
Under 30	(45)	31	
30–34	(76)	33	
35–39	(58)	28	
40 & over	(30)	33	
Marital status			(Not entered)[c]
Single, never married	(85)	39	
Married	(77)	26	
Other	(49)	29	
Living with a Sexual Partner[d]			
No	(74)	43	1.00
Yes	(137)	25	.70 (.44, 1.12)
Sexual Partners, Past Year			
One	(144)	27	1.00
Two or more	(66)	42	1.06 (.65, 1.71)
Sexual Partners Injecting Drugs, Past Year			
None	(140)	29	1.00
One or more	(70)	37	.98 (.62, 1.55)
Planning on Conception			
No	(170)	35	1.00
Yes	(39)	18	.84 (.46, 1.50)
Frequency of Heterosexual Relations, Past Month			1.91 (.83, 4.39)
One or two times	(84)	26	
One day a week	(26)	38	
A couple days a week	(78)	38	
Almost every day	(20)	20	
IV Drug Use Risk, Past Month[e]			
None	(125)	30	1.00
Injects, no needle sharing	(50)	34	1.09 (.67, 1.76)
Injects and shares needles	(36)	36	1.11 (.62, 1.97)
Knows AIDS/ARC Victims[f]			
None	(130)	26	1.00
One or more	(81)	41	1.50 (.98, 2.29)
Knows HIV Serostatus			
No	(174)	30	1.00
Yes, Seronegative	(30)	40	.80 (.29, 2.21)
Yes, Seropositive	(7)	43	1.39 (.81, 2.39)
Fatalism about AIDS[g]			
Agrees will get AIDS	(48)	40	1.00
Disagrees will get AIDS	(160)	29	.79 (.60, 1.62)
Condom Reliability[h]			
Agrees condoms break	(124)	27	1.00
Disagrees condoms break	(82)	40	1.12 (.73, 1.70)
Knowledge of Sexual Risks[i]			.91 (.71, 1.16)
Low	(52)	36	
Medium	(98)	34	
High	(61)	24	
Condom Acceptability[j]			1.75 (1.21, 2.52)
Low	(29)	10	
Medium	(116)	24	
High	(63)	56	
Condom Efficacy against AIDS[k]			1.13 (.76, 1.67)
Low	(91)	24	
Medium	(53)	28	
High	(66)	44	
Partners' Receptivity to Protection[l]			1.47 (1.02, 2.12)
Low	(24)	13	
Medium	(47)	26	
High	(135)	37	
Perceived Past Exposure to HIV[m]			1.07 (.75, 1.52)
Low	(33)	21	
Medium	(75)	35	
High	(103)	33	
Perceived Susceptibility to AIDS[n]			1.27 (.90, 1.79)
Low	(55)	27	
Medium	(88)	26	
High	(67)	42	
Time in Methadone Program[o]			.83 (.72, .96)
1–11 months	(50)	40	
12–23 months	(43)	39	
24–35 months	(27)	26	
36–47 months	(11)	0	
48 months and over	(78)	28	

[a]Includes 5 "others."
[b]Entered in regression as a continuous variable. Odds ratio is for a 5-year age interval.
[c]Not entered in the logistic regression because of high association with next variable, "Living with a Sexual Partner."
[d]"Are you now living with someone with whom you have a sexual relationship?"
[e]"In the last month, how often did you shoot up any drugs?" and "In the last month, how often did you share a needle or works with someone?"
[f]"How many people with AIDS or ARC (AIDS-related complex) do you know personally?"
[g]"My gut feeling is, I'm going to get AIDS sooner or later."
[h]"I don't like using rubbers (condoms) because they sometimes break."
[i]The correct answers were summed for seven items: "You could get AIDS by having unprotected sex with a person who shoot drugs" (T); "only homosexuals can give you AIDS through sexual relations" (F); "pregnant women who carry the AIDS virus can pass it to their newborn children" (T); "anal intercourse is the only way sex can give you AIDS (F); "a positive AIDS antibody test means you could give the AIDS virus to someone through sex" (T); "you can give the AIDS virus to someone else by sex or needle-sharing even if you feel healthy" (T); "you can't get AIDS just by oral sex (F)." Higher knowledge = higher score. Entered in regression as a continuous variable.
[j]Two items were summed: "I don't like using rubbers (condoms) because they cut down on my enjoyment" (agree = 0, not sure = 1, disagree = 2); "I would use rubbers (condoms) if my partner asked me" (agree = 2, not sure = 1, disagree = 0). Higher acceptability = higher score. Entered in regression as a continuous variable.
[k]Two items were summed: "Using a rubber (condom) can help protect you from getting AIDS" (agree = 2, not sure = 1, disagree = 0); "there's no way to have 'safe sex' with someone who's carrying the AIDS virus" (agree = 0, not sure = 1, disagree = 2). Higher perceived efficacy = higher score. Entered in regression as a continuous variable.
[l]Two items were summed: "My sex partner would be insulted if I suggested that we use a condom" (agree = 0, not sure = 1, disagree = 2); "my sex partner would be insulted if I suggested any changes in the way we have sex" (agree = 0, not sure = 1, disagree = 2). Greater receptivity = higher score. Entered in regression as a continuous variable.
[m]Two items were summed: "I've already done plenty that could have exposed me to AIDS" (agree = 2, not sure = 1, disagree = 0); "I never did anything that could give me AIDS" (agree = 0, not sure = 1, disagree = 2). More exposure = higher score. Entered in regression as a continuous variable.
[n]Two items were summed: "I'm worried that some of my sexual partners could give me AIDS" (agree = 2, not sure = 1, disagree = 0); "I'm worried about my chances of getting AIDS" (agree = 2, not sure = 1, disagree = 0). Higher susceptibility = higher score. Entered in regression as a continuous variable.
[o]Odds ratio is for a 6-month interval. Entered in regression as a continuous variable.

continued

their use.[18] Participants can also practice communication and negotiation skills pertinent to sexual relationships. Even if neither partner personally objects to condom use, as our data suggest is often true, one partner must nevertheless take the initiative and broach the subject of use.

Addicts who recently entered methadone treatment were more likely to use condoms. This relationship requires more detailed investigation.

It is of particular concern that, of the 39 subjects (19 percent) planning on conception, only 20 percent reported being tested for HIV antibody (all seronegative). These findings held true equally for men and women. AIDS health education must emphasize the importance of HIV testing for IVDUs who wish to conceive, given the at least 30 percent risk that seropositive women will bear infants who will develop HIV-related illness.[19,20]

The study has several limitations. Causal interpretations should be considered tentative because the data are cross-sectional. The subjects were all in methadone treatment; an estimated 15 percent of IVDUs in New York City are in treatment at any given time.[21] It is possible that a study of street addicts would yield different results, but note that we found no association between current intravenous drug use and condom use for our sample (Table 1). Not all variables hypothesized or found to be associated with condom use in the research literature were measured by the study. These include a possible belief that condoms make sex seem premeditated, possible embarrassment in purchasing condoms, and the cost of condoms for low-income persons. The first two factors, however, have seemed particularly important only for adolescents.[22–24] The cost of condoms has become a less pertinent consideration for addicts since many drug abuse treatment programs (including the study clinics), as well as AIDS prevention outreach projects, have been distributing condoms for free.

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Future APHA Meeting Dates/Site

1990 SEPTEMBER 30–OCTOBER 4 NEW YORK, NEW YORK
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1994 OCTOBER 30–NOVEMBER 3 WASHINGTON, DC
1995 NOVEMBER 12–16 SEATTLE, WASHINGTON

Results of an Intervention to Improve Compliance with Referrals for Evaluation of Suspected Malignancies at Neighborhood Public Health Centers

CLARA MANFREDI, PHD, LORETTA LACEY, RN, DRPH, AND RICHARD WARNECKE, PHD

Abstract: A study was conducted at Neighborhood Health Centers to evaluate procedures to improve compliance with referrals of patient at risk for cancer. Intervention consisted of a standardized communication from the exit nurse, a patient form to be returned after compliance, and one written and one telephone reminder as needed. Compliance was 68.2 percent in a control group and 89.0 percent among patients who received the experimental intervention, a significant increase of 20.8 percent (95% CI: 12.5, 29.1). (*Am J Pubic Health* 1990; 80:85–87.)

Introduction

Excessive cancer mortality among low income and minority populations could be reduced with better screening and earlier detection and treatment.[1–6]

Indigent populations rely upon public clinics for primary care which refer persons with suspected cancer to other medical facilities for further care; compliance with referrals is often problematic, contributing to delayed treatment.[7,8]

Delay in seeking care has been associated with difficulty communicating with physicians,[7,9,10] difficulty obtaining prompt appointments,[7,11] and concern about financial costs.[12–15] In contrast, compliance improved with prompt appointment dates, clear and specific instructions, and reminders.[7,11,16–18] Fear of cancer may be a deterrent to promptly seeking a diagnosis,[19,20] whereas knowledge of cancer symptoms and belief in the possibility of early detection and cure may lead to prompt diagnosis and treatment.[13,14,20–22] This study, however, focused only on intervention designed to improve referral information and remind those patients who may not have kept the referral appointment.

Methods

The study was conducted between October 1984 and January 1986 at four Chicago Department of Health Neighborhood Health Centers (NHCs). Subjects were adult medicine and maternal and family planning patients, at least 18 years old, without prior cancer history and referred for treatment or further diagnostic workup for a suspected cancer.

At the exit interview, nurses identified eligible patients and, in the experimental condition, administered the intervention. Overall, 292 control and 226 experimental group patients were accrued. No patient consent was needed because the intervention was an improvement on normal care and placed the subject at no risk. Monitoring procedures revealed that approximately 50 percent of eligible cases were not included in the study because of direct physician referral, missed opportunity by nurses to see the patient, nurse was too busy. Missed cases were more likely than those accrued to the study to be referrals for non-specific evaluation and to lack an appointment date, but occurred equally in the control and experimental conditions based on NHC assignment. Study results are based only on identified cases.

Unpredictability of case occurrence and need to integrate the study procedures into routine NHC's activities made random assignment to study conditions unfeasible. Instead, each NHC was assigned to experiment or control on alternate months, with two NHCs in either condition in any given month.

The experimental intervention consisted of:

- A verbal nurse-patient communication, structured with a mini-questionnaire designed to facilitate queries about referral procedures (i.e., why needed, where to go, whom to contact, etc.).
- A form, which patients were requested to return in a prestamped, pre-addressed envelope after referral completion.
- A reminder note, mailed to 163 patients (72.1 percent) whose chart had no return form nor other record of compliance two weeks after appointment or six weeks after referrals without appointment.
- A telephone reminder, attempted with 102 patients (45.1 percent) still without record of compliance three weeks later. Only 79 calls were completed, because patients could not be found or nurses could not make numerous attempts. Those not contacted by phone were sent a second written reminder and 13 patients were reminded of the referral as they revisited the NHC.

Control patients received the NHCs' usual referral procedures.

Compliance was ascertained by phone interviews. Interviews were attempted with all patients three weeks after final chart review and completed with 393. The remaining were: 106 not located, 15 with communication barriers, two deceased, and two refused. Completion was 75.3 percent in the control and 76.5 percent in the experimental group. Study results are based only on the 393 cases with final interview.

Results

Compliance was reported by 304 patients (77.5 percent). Of these, 61 (27.7 percent) of the control group and 73 (42.2 percent) of the experimental group also had documentation of compliance in their medical chart and 51 patients (29.5 percent of experimental group) had returned their patient intervention form.

Study patients were predominantly Black (69.5 percent), female (88.5 percent), 48 percent were under age 55, and 23 percent under age 35. Only 18.8 percent of referrals received

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TABLE 1—Percent of Patients Who Complied with Referrals in Each Study Group, Overall and by Patient and Referral Characteristics

Variables	Control Group (N = 220) %	N	Experimental Group (N = 173) %	N	Per cent Difference	(95% CI)
Overall	68.2	(150)	89.0	(154)	20.8	(12.5, 29.1)
Sex						
Male	84.6	(22)	100.0	(19)	15.4	(−1.6, 32.4)
Female	66.0	(128)	87.7	(135)	21.7	(12.7, 30.7)
Race						
Black	64.7	(101)	87.2	(102)	22.5	(12.5, 32.5)
White	61.1	(11)	77.8	(7)	16.7	(−16.3, 49.7)
Hispanic	88.9	(8)	96.0	(24)	7.1	(−23.9, 38.1)
Others	81.5	(22)	94.7	(18)	13.2	(−6.5, 32.9)
Age (years)						
18 to 34	73.7	(28)	90.3	(28)	16.6	(−2.0, 35.2)
35 to 44	65.0	(13)	100.0	(25)	35.0	(13.4, 56.6)
45 to 54	69.6	(16)	79.3	(23)	9.7	(−14.0, 33.4)
55 to 64	63.3	(30)	87.5	(36)	23.7	(5.5, 41.9)
65 to 74	65.4	(34)	96.3	(26)	30.9	(11.0, 50.8)
75 or more	72.5	(29)	81.0	(17)	8.5	(−14.4, 31.4)
Education (years)						
Less than 9	71.6	(58)	92.2	(47)	20.6	(6.5, 34.7)
9 to 11	68.1	(49)	86.2	(150)	18.1	(3.4, 32.8)
12 to 14	67.2	(39)	89.5	(51)	22.3	(7.2, 37.4)
15 or more	44.4	(4)	85.7	(6)	41.3	(−6.5, 89.1)
Clinical Reasons for Referral						
Prior test results	81.6	(40)	97.1	(33)	15.5	(1.3, 29.7)
Symptoms only or missing information	64.3	(110)	87.0	(121)	22.7	(12.9, 32.5)
Procedure Requested in the Referral						
Evaluate, rule out	83.3	(50)	93.0	(53)	9.7	(−2.1, 21.5)
Mammography	54.7	(52)	84.1	(58)	29.4	(14.8, 44.0)
All others	73.8	(48)	91.5	(43)	17.7	(3.0, 32.4)
Appointments						
Within 15 days	92.9	(39)	96.9	(31)	4.0	(−6.4, 14.4)
Within 16 to 147 days	53.5	(38)	82.5	(52)	29.0	(13.1, 44.9)
No appointment	68.2	(73)	91.0	(71)	22.8	(10.7, 34.9)
Insurance Coverage						
Yes	67.5	(77)	89.6	(60)	22.1	(9.2, 35.0)
No	68.9	(73)	88.7	(94)	19.8	(7.9, 31.7)
Referral Hospital						
Cook County Hospital	60.8	(79)	86.8	(105)	26.0	(15.0, 37.0)
Other hospitals	78.9	(71)	94.2	(49)	15.3	(2.9, 27.7)

an appointment date within 15 days and 47.1 percent received no appointment. Almost half of the patients had no insurance and 63.9 percent were referred to the public county hospital.

Experimental group patients were more likely than controls to be Hispanic (14.7 versus 4.1 percent), lack insurance (61.3 versus 48.2 percent) and be referred to the county hospital (69.9 versus 59.1 percent).* Within the experimental group, patients who had received an appointment date were more likely to have returned their intervention form (36.8 percent) than those without an appointment date (20.5 percent).

Compliance with referrals was 68.2 percent in the control and 89.0 percent in the experimental group, an increase of 20.8 percent (Table 1). Characteristics associated with better compliance in both groups included: male gender, referrals following prior test results and for appointments within 15 days. Compliance was less likely for referrals for Blacks, for mammography, and for referrals to the county hospital.

Improved compliance was most evident for conditions associated with non-compliance in the control group. It was least evident for appointments within 15 days, which were associated with high compliance regardless of intervention.

The high appointment-keeping of patients with prompt appointments is striking and may be due partly to the latter being associated with more urgent medical conditions. Appointments within 15 days were more likely to be for follow-up of positive Pap smears (47.4 percent) than for positive guaiac or other laboratory results (28.9 percent) or for all other conditions (13.9 percent) and for referrals for surgical or other procedures (33.7 percent) than for evaluation (20.5 percent) or mammography (9.1 percent) (data not presented in tables).

Because the majority of patients in the study were Black females, results were analyzed separately for this group. Compliance among Black females was 61.9 percent in the control and 85.8 percent in the experimental group. Their patterns of compliance by patient and referral variables within study group were very similar to those reported in Table 1 for the entire sample.

Discussion

The study findings indicate that compliance with referrals can be improved when appointments are within two weeks of referral and when clinic staff give clear instructions and follow-up patients to ensure receipt of prescribed procedures. Even when the situation of the referral would predict poor compliance, careful attention by the clinic staff reduced the noncompliance significantly.

The majority of referrals in this study were for mammography and to follow-up positive Pap smears. Without the intervention, compliance with referrals following prior test results (thus including positive Pap smears), was 81.6 percent, but only about half of the women referred for mammography received the procedure.

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*Exact data available on request to authors.

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APHA Invites Proposed Policy Statements for 1990 Consideration

The American Public Health Association has announced the schedule for the annual process of considering proposed policy statements. Individual members of APHA may submit resolutions. The various sections, affiliates, caucuses, SPIGs (special primary interest groups), and other components defined in the APHA constitution and by-laws may offer position papers.

Resolutions are short, single statements of 500 words or less, focusing on a specific action or series of actions endorsed by the Association, whereas a *position paper* is a major exposition, of up to 3,000 words, of the Association's viewpoint on a broader issue affecting the public's health. Each proposed statement will be considered by the APHA Joint Policy Committee which may recommend revisions to authors before the time of the APHA annual meeting. When the convention opens in New York City, open public hearings are held on all proposed policy, and the Governing Council takes final action by adopting or rejecting the proposed policy statements.

These public policy statements form the basis of APHA's stand on legislative, regulatory, legal, and health matters of concern to the public health community. They may stimulate scientific inquiry or be intended to initiate government action. The statements also provide a record of APHA's position on a variety of health issues, reflecting the diverse interest of the Association's membership, and further serve as a chronicle of changing and evolving public health concerns over the years of American history.

The *deadline* for receipt of proposed resolutions and position papers for consideration this year is *February 26, 1990*. A copy of the schedule and procedures is available from: Joint Policy Committee, APHA, 1015 15th Street, NW, Washington, DC 20005.

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Fecal Contamination of Shanty Town Toddlers in Households with Non-corralled Poultry, Lima, Peru

Grace S. Marquis, MS, Gladys Ventura, BS, Robert H. Gilman, MD, Esperanza Porras, BS, Elba Miranda, MS, Luz Carbajal, MS, and Marta Pentafiel, BS

Abstract: We used direct observer techniques to measure the frequency with which toddler-aged children were contaminated by poultry feces in homes in a peri-urban shanty town in Lima, Peru. The mean number of fowl was 5.4 (SD 3.1), with 10.0 (SD 10.7) poultry defecations per 12 hours. Toddlers' hand contact with poultry feces occurred a mean of 2.9 (SD 3.0) times/12 hours. A mean of 3.9 (SD 4.6) feces-to-mouth episodes per household/12 hours occurred both by direct hand-to-mouth contamination and indirectly by handling soiled objects which were then placed in the mouth. There was a strong correlation between feces-to-hand contamination and feces-to-mouth contamination (r = 0.94). There was also an association between feces-to-mouth contamination and the number of stools deposited in the house (r = 0.66). For each additional chicken stool deposited during the day, there was an average increase of 0.27 in feces-to-mouth episodes.

We collected feces from 68 infected chickens and found viable *Campylobacter jejuni* for up to 48 hours after deposition. Yet, a survey of 108 families demonstrated that free-roaming poultry were often not thought of as a health risk for children. An intervention program to reduce oral-fecal contamination should emphasize that all poultry be corralled and not allowed access into the house. (*Am J Public Health* 1990;80:146–149.)

Introduction

Diarrheal disease is an important factor in the infection-malnutrition cycle of infants and young children in developing countries. In Peru, children under three years of age have a diarrheal incidence of 10 episodes per child-year, which can significantly affect their growth rates.[1] One important cause of diarrhea in Lima children less than one year of age is *Campylobacter jejuni*. This organism was found in 15 percent of diarrhea stool samples[2] and accounted for the majority of dysenteric diarrheas.[3]

In Peru, *C. jejuni* has been isolated from 88 percent of commercial chickens and 50 percent of free-ranging domestic chickens in a pueblo joven (a peri-urban shanty town community).[4] In an aged-matched case-control study of Lima peri-urban households, children in families with chickens had a three-fold relative risk of having *C. jejuni* diarrhea which increased to 12.5 in families with *C. jejuni* infected chickens.[2] These risk factors suggest that direct contact with free-roaming *C. jejuni*-infected chickens that defecate on household surfaces is an important transmission vehicle for young children.

Direct observation of the personal hygiene of small children and mother-child behavioral patterns is the most reliable method for obtaining data.[5] We used directed observer techniques to measure the rate at which toddlers in a shanty town contaminate themselves with domestic poultry feces.

Methods

Study Sites

The study was done in two pueblos jovenes (recent shanty towns): Huascar, Canto Grande[6] and Las Pampas de San Juan, San Juan de Miraflores, in Lima Peru. The majority of the study population are rural immigrants who are daily laborers or street vendors with low, unstable incomes. Housing materials in Huascar and San Juan range from woven cane matting to permanent structures of brick and cement. Although the majority of pueblos jovenes do not have electrical installations, most of our study households had them. Water is scarce in both pueblos. It is provided through tanker trucks, and in half of San Juan through piped communal water spigot. In-house sewage systems are not available.

Direct Observation Data

Ten families from Huascar participated in an in-depth behavioral observation study, with a total of 21 children under five years of age. All the families had at least one child under two years of age, and domestic chickens which were not corralled at least part of the day. They gave informed verbal consent prior to their participation. The study received ethical approval by all involved Peruvian institutions (Universidad Peruana Cayetano Heredia, Instituto de Investigación Nutricional, and PRISMA - Proyectos en Informática, Salud, Medicina y Agricultura).

To gather the direct observation data, a field worker remained in the toddler's home for one 12-hour observation period (7am to 7pm). At the beginning of the visit, a descriptive inventory of the domestic animals was completed. During the 12-hour period the observer noted all activities in the house for each fowl, including the number of times the fowl entered the home, the duration of time spent inside, and how frequently and where the poultry defecated. The observer also tabulated the activities of the toddler, specifically noting the number of times the child's hands were contaminated with poultry feces (defined as feces-to-hand episodes) and the number of times that his/her hands, or other objects contaminated with poultry feces, were introduced into his mouth (defined as feces-to-mouth episodes). Feces-to-mouth episodes were considered to have been interrupted and were not tabulated if the child's contaminated hands had been washed before he placed them in his mouth.

All feces-to-hand and feces-to-mouth episodes were tabulated for each family and analyzed as episodes per child. Correlations and linear regressions for the feces-to-hand and

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feces-to-mouth episodes were performed with the following variables: number of fowl, hours in the house/12 hours, and number poultry stools/12 hours.

Poultry Raising Practices and Beliefs

We surveyed a group of 108 families living in Las Pampas de San Juan to document their practices and beliefs regarding the raising of chickens and other fowl. The survey identified the reasons families gave for raising or not raising poultry, their beliefs about corralling the poultry, and the effect that confinement has on the growth and the health of the animal, the perceived advantages of raising chickens in the home compared to buying them commercially, and the mothers' perception concerning whether poultry roaming within the house presented a health risk to young children.

Socioeconomic Data

As part of a longitudinal study of *Enterobius vermicularis*, 121 randomly selected families from Huascar were included in a socioeconomic survey. The same data were collected for the Pampas families that participated in the poultry raising practices survey. The socioeconomic survey detailed age, sex, level of education, and occupation of all members of the family. Questions on housing quality included the materials used to build the house, number of rooms, ownership of functioning radios, televisions, and refrigerators, and presence or absence of a latrine. Finally the number and types of animals belonging to the home owners were also registered.

The practices and beliefs of mothers who did and did not raise poultry were compared using Chi square analysis. Each socioeconomic data set from the two pueblos jovenes was analyzed individually, and compared for socioeconomic indicators, using Student t test and Chi-Square analysis.

Survivability of *C. jejuni* in Deposited Chicken Feces

Stool samples were taken from *C. jejuni*-positive chickens, raised in a household in Huascar, Canto Grande. A cloacal swab was taken immediately after the chicken defecated and another swab was taken from the stool on the ground. These served as baseline samples. The stool sample, on the ground of a sun-exposed patio, was fenced in with a metallic wire and marked with its respective identification number and code. After 24 and 48 hours, additional samples were taken. All the samples were immediately placed in Cary Blair media containing Skirrow's antibiotic supplement,[7] and processed by direct plating on blood agar with Butzler's supplement.[8] Another sample was inoculated into thioglycolate broth supplemented with iron and the following antibiotics: vancomycin 10 μg/ml, polymyxin B 2.5 I.U./ml and trimethoprim lactate 5 μg/ml. The plates were incubated immediately while the broth was refrigerated for 24 hours and then plated with Butzler's agar. All plates were incubated at 37°C under microaerophilic conditions. Campylobacter species were identified based on colony morphology, oxidase production, and spiral appearance in the Gram stain.

Results

Direct Observation in the Homes

The interaction between toddlers and poultry was observed for a total of 119 hours in 10 Huascar households. The majority of the poultry were chickens (fowl/house = 5.4, chickens/house = 4.4). Three out of 10 families also had ducks and turkeys. Chickens and ducks only were allowed to roam freely through the house for about one-third of the 12-hour observation period (Table 1). The number of poultry and the time they were in the house were correlated (r = 0.64), as were the number of observed stools and the time the animals spent within the house (r = 0.86).

The children had approximately three feces-to-hand episodes per 12 hours (Table 1). The risk of feces-to-hand contamination was from both fresh stool and from that deposited several days previously but not removed from the household surfaces. In three households where no observed defecations occurred, hand and oral contamination still happened with stools that had been deposited prior to the day of observation. The two most important variables were number of droppings and number of minutes chickens were in the house.

There were about four feces-to-mouth episodes per child (Table 1). Children only rarely washed their hands after touching feces, and often put their fingers in their mouths, so feces-to-hand and feces-to-mouth episodes were highly correlated (r = 0.94); feces-to-hand episodes and the number of poultry accounted for 93 percent of the variation in the dependent variable—the number of feces-to-mouth episodes. Neither feces-to-hand nor feces-to-mouth episodes were related to the number of young children in study families. For each additional chicken stool deposited during the day, there was an average increase of 0.27 in feces-to-mouth episodes.

Poultry Raising Practices

One hundred and eight mothers from the Pampas de San Juan participated in the poultry raising practices survey. Eighty-one (75 percent) of the mothers were migrants, usually from the sierra zones. Whereas 88 percent (14/16) of the Lima-born mothers raised multiple types of poultry, only half of the migrant mothers raised other types of poultry in addition to chickens (27/54).

The major reasons given for a raising poultry (Table 2) included their use for home consumption and enjoyment of raising animals. The majority of mothers realized that commercial chickens were cheaper than those raised at home. When mothers were asked whether commercial or domestic chickens cost more, 77/108 (71 percent) thought the commer-

TABLE 1—Number of Domestic Poultry and Their Interactions with Toddler-Aged Children, Direct Observation Households (n=10)

			Feces-to-Hand*		Feces-to-Mouth*	
	Mean	SD	Regr Coef	95 CI	Regr Coef	95 CI
# Foul per house	5.4	3.1	−0.0326	(−0.693+0.628)	0.2831	(−0.735+1.301)
# Min/12 hours fowl in house	14043.0	2099.8	0.0004	(−0.001+0.001)	0.0006	(−0.001+0.002)
# Stools/12 hours	10.0	10.7	0.1664	(−0.025+0.308)	0.2718	(+0.057+0.487)
# Feces-to-hand episodes	2.9	3.0			1.474	(+1.101+1.846)
# Feces-to-mouth episodes	3.9	4.6				

*Dependent variable in simple linear regression model.

TABLE 2—Pampas Family Practices and Beliefs Concerning the Raising of Domestic Poultry

Family Practices, Beliefs	Families with Poultry		Families without Poultry	
	n	% of total	n	% of total
Why they raise poultry:				
To have to eat	41	60		
Enjoy raising animals	17	24		
No response	11	16		
Why they do not raise poultry:				
Are filthy/carry disease			11	29
Not economic (more expensive/ are stolen)			8	21
Without infrastructure/time			12	32
Do not like it/not accustomed			7	18
Where poultry grow best:				
Corralled	27	39	22	58
Roaming freely	37	54	13	34
Either or both places	4	6	2	5
No response	1	1	1	3
Is there a health risk for the family, having poultry loose in the house?				
No	20	29	11	28
Yes, cause illness	16	23	15	38
Yes, dirty or bring flies	13	19	8	21
Don't know	20	29		13

TABLE 3—Huascar and Pampas Family Socioeconomic Status, According to Selected Variables

	Huascar families		Pampas families	
Mean family size	6.5 S.D. 2.5		5.1 S.D. 1.8	
Mean age of mother(yrs)	31.5 S.D. 9.8		27.1 S.D. 5.8	
Mean number of years in Lima	—		15.8 S.D. 7.8	
	n	% of total	n	% of total
% with radio	79/120	66	77/108	71
% with television	82/120	68	73/108	68
% with radio and/or television	108/120	90	93/108	86
% with refrigerator	22/120	18	19/108	18
% with bore hole latrine	80/120	67	73/108	68
Housing quality:				
% with noble materials (bricks)	42/120	35	18/108	17
Education of mother:				
% completing secondary	15/120	13	31/108	29
% completing primary	60/120	50	56/108	52
% not completing primary/none	43/120	36	21/108	19
% no response				
Education of father:	2/120	2		
% completing secondary	31/120	26	56/108	52
% completing primary	66/120	55	42/108	39
% not completing primary/none	14/120	12	3/108	3
% absent	5/120	4	4/108	4
% no response	4/120	3	3/108	3

cial chickens were cheaper while only 22/108 (20 percent) thought the reverse.

In 83 percent (58/70) of the families, the poultry roamed freely for much of the day, with access into the house. This practice was explained in part by the belief of more than one-half of the mothers that the animals grew better when they were allowed to wander freely. Seventy four percent of the mothers not raising poultry and 68 percent of the mothers with poultry thought that there was a health risk or a negative hygienic aspect associated with free-roaming poultry.

Socioeconomic Data

Table 3 shows the socioeconomic indicators studied in both pueblo joven communities. No socioeconomic differences were found in families that did or did not raise chickens and other poultry.

Microbiological Results

Forty percent (27/68) of the stool samples from *C. jejuni*-positive chickens deposited in sun-exposed patios still had viable organisms demonstrable after 24 hours. At 48 hours, only 18 percent (10/57) of the samples were positive.

Discussion

Campylobacter jejuni infection most commonly occurs through human fecal-oral transmission or through contaminated food or water.[9–13] This transmission is difficult to document, unless direct observation of family hygienic practices is used. Stanton, *et al*, demonstrated that responses to 24-hour recall and knowledge-attitude-practices questionnaires do not reliably reflect observed hygienic household practices.[5] The direct observation method is a strength of this study. A limitation is that the sample size is small. These data nonetheless demonstrate that contamination of toddlers' hands with feces deposited in the house by free-roaming birds is extremely common in Peruvian pueblo jovenes. In Bangladesh, play areas of infants and toddlers were also seen to be contaminated with chicken feces in approximately 66 percent of the households.[14] Approximately one-half the Bangladeshi mothers claimed to have seen the child touching or eating animal feces during the previous two weeks.

Unwashed hands of toddlers in a pueblo joven are frequently contaminated with poultry feces. As 50 percent of the chickens in a pueblo joven are carriers of *C. jejuni*,[4] the health risk to these toddlers is evident. We found that few of these feces-to-hand episodes were interrupted by hand washing.

Many pueblo joven mothers do not believe that chicken stools provoke illness. Similar results were found in the Bangladesh study, where over one-half of the mothers also did not recognize animal feces as disease-producing.[14]

The absence of running water in pueblo jovenes and the high cost of the water used may be important factors discouraging hand washing. Even in developed countries where water is easily available, oral-fecal contamination is common. Direct observations in an English nursery school during an epidemic of shigellosis showed that 50 percent of the children had contaminated their hands with *Shigella sonnei* after using the school toilet. Moreover, one-third of the children handled their face or sucked their fingers after using the toilet.[15]

The risk of acquiring viable *C. jejuni* in the study area is augmented because the house floors are usually made of dirt or unfinished concrete, so the complete removal of poultry feces is difficult to achieve, and *C. jejuni* can survive in the ambient environment for several days. Even where water is plentiful, in homes with free-roaming chickens, it is not realistic to assume that mothers will be able to effectively interrupt transmission by handwashing.

In addition to *C. jejuni*, a small percentage (2 percent) of poultry were fecal carriers of *Aeromonas* species.[4] The relationship of this organism to diarrhea has not been established. None of the free-roaming chickens sampled excreted *Salmonella* species. In both Peru and Bangladesh,[1,16] *Sal-*

monella species is an infrequent cause of community epidemics of diarrhea.

The present study suggests similar socioeconomic background and characteristics of families with and without poultry in a pueblo joven population, making it difficult to identify a target population. In addition, an intervention program would have to overcome common beliefs that inherently encourage the transmission of *C. jejuni*. Many families raised uncorralled chickens even though they recognized that purchasing poultry was cheaper than home grown animals, believing that chickens grow better when they are corralled; and appreciated the risk in having chickens roaming freely through the house. This suggests that this behavior will be difficult to modify.

In Peru, as our data show, most individuals living in pueblo jovenes have access to mass media. Most children attend primary school. Both mass media and formal education in the school system could be utilized for intervention programs. These educational programs should emphasize the health risks to toddlers of raising free-roaming poultry within the household and the need to corral poultry and prevent their entrance into the house.

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8th Competition Announced for Health Professions Students

Dr. Louis W. Sullivan, Secretary of Health and Human Services, has announced the 8th annual national competition among health professions students for the "Secretary's Award for Innovations in Health Promotion and Disease Prevention." Winners will receive awards of $3,000 for first place, $2,000 for second, and $1,000 for third; up to 17 semifinalists will receive $250 each.

The competition is open to a broad range of health professions students enrolled in baccalaureate or higher degree programs affiliated with the Federation of Associations of Schools of the Health Professions, cosponsor of the competition. Those eligible to compete include students of medicine, osteopathic medicine, nursing, veterinary medicine, optometry, pharmacy, podiatric medicine, public health, health administration, health education, and allied health.

Papers should present innovative proposals for health promotion or disease prevention in the community, at the worksite, in an educational setting, or for a special population group.

The deadline for submitting entries is March 15, 1990. A paper must be submitted to the school attended by the author or authors. Papers will be judged first by the school and then by the professional associations. A departmental panel will make the final selections, and HHS Secretary Sullivan will announce the winners in July. For further information, contact Blake Crawford, Health Resources and Services Administration (HRSA), US Public Health Service, DHHS, 5600 Fishers Lane, Rockville, MD 20857; tel: (301) 443-3376.

HIV Seropositivity of Needles from Shooting Galleries in South Florida

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Abstract: Needle/syringe combinations were collected from three shooting galleries in South Florida and tested for the presence of antibodies to HIV-1. Fifteen of 148 needles (10.1 percent) tested positive for HIV-1 antibody. Seropositivity rates did not vary by the day of the week of collection, nor by shooting gallery from which they were collected. When the needle appeared to contain blood residue, 20.0 percent were positive versus 5.1 percent with no blood residue. These findings suggest that needles/syringes used in shooting galleries are likely to serve as reservoirs and/or vectors of transmission of the HIV-1 virus, and that although visual inspection of the needle/syringe may be useful in lessening the chance for transmission, even the visually "clean" needles may result in transmission of infection. (*Am J Public Health* 1990; 80:150–152.)

Introduction

The self-reported sharing of needles and syringes, particularly those belonging to shooting galleries, has been consistently associated with increased risk for HIV-1 infection among parenteral drug users.[1–3] A shooting gallery is a place where addicts go to rent syringes and needles ("works") and to inject drugs.

Shared drug injection equipment can transmit HIV-1 by parenteral transfusion when residual, contaminated blood remains in previously used syringes and needles and that equipment is reused by another person. Blood residue is often present in the syringe because of the aspiration of venous blood into the syringe.

No one has assessed the extent to which equipment owned by shooting galleries is positive for HIV-1 antibodies. Recently a study of needle-and-syringe exchange programs in Sydney, Australia determined that antibodies for HIV-1 were present in 3.1 percent of 1,544 needle and syringe combinations exchanged at two exchange centers.[4] Risk of exposure to HIV-1 probably is higher in shooting galleries where a wide variety of people rent the same works and needle and syringe combinations are routinely reconditioned to extend their useful life far beyond the average nine uses reported by intravenous users in Miami who reuse or share personal works.

In order to examine the potential for HIV-1 transmission through the use of injection equipment available in high-risk settings, needle/syringe combinations were collected from shooting galleries frequented by parenteral drug users in Miami, Florida and were tested for antibodies to HIV-1. Although this study measures antibody and not the presence of an infectious dose of virus, these data help explain the association between shooting galleries and HIV-1 infection.

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Editor's Note: See also related editorial p 142 this issue.

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Methods

Selection of Shooting Galleries

Three shooting galleries located in Miami, Florida were selected as the sites from which to collect needle/syringe combinations. These were among the most frequently mentioned galleries that were attended by parenteral drug users enrolled in coordinated investigations of the natural history of HIV-1 and the evaluation of risk behavior reduction programs. Seroprevalence* among users enrolled in these studies is approximately 36.1 percent for Blacks who were not Hispanic, 30.2 percent for Hispanics,** and 8.9% for Whites who were not Hispanic. The majority of persons who frequented these galleries were Blacks who were not Hispanic. Each gallery was located in a different inner-city area known for high rates of drug use.

Field observations at eight additional galleries indicate that the modes of operation at the study sites were typical for area shooting galleries. Each was housed in the residence of the gallery operator. All three rented used works for $2 and had bleach or alcohol available for cleaning works, and none sold drugs. Heroin and/or cocaine, both available for purchase nearby, were predominant. Syringe and needle repair and cleaning occurred in these galleries but no observations were made specifically for these needles and syringes.

Needle/Syringe Collection

Access to the three galleries was gained through the efforts of a staff outreach worker who had established a variety of contacts with the intravenous drug using population. He was a follow-up worker who spent all of his time locating drug using participants of a longitudinal study. Because he was not an outreach worker in an HIV prevention program, the shooting gallery operators were less likely to feel pressure to manipulate the selection of syringes. Needle/syringe units that had been used in the shooting galleries during the previous 24-hour period were collected each morning by the follow-up worker during two one-week periods in 1988. In order to avoid possible bias by day of the week, no more than six units were collected per site per day. Wherever more were available, six were selected by the follow-up worker without regard to appearance or condition.

*Seroprevalence is based upon data from three coordinated studies of HIV infection among intravenous drug users (N = 1364) in Miami, Florida.

**The study population includes Hispanics who are White and Hispanics who are Black; they are Hispanic in culture and do not identify themselves as White or Black.

Each gallery operator was paid a flat fee of $24 per visit and did not participate in the selection of syringe units.

Each morning after collection, the needle and syringe combinations were immediately transported to the University of Miami and visually graded as follows:

1. "Visible Blood"—The needle/syringe appeared to contain liquid or dried blood.
2. "Dirty, No Visible Blood"—The needle/syringe contained visible dirt, or stains, but no blood.
3. "Clean, No Visible Blood"—The needle/syringe contained no visible dirt, stains or blood.

Needles were then delivered to the laboratory where they were tested for the presence of antibodies to HIV-1.

Laboratory Methods

A pilot study had been conducted with 17 needle and syringe combinations under conditions simulated to represent the condition of the needles/syringes which would be collected at the shooting galleries. Using the blood samples from a high-risk group whose blood was sent to the laboratory for routine testing, 200 μl of blood from each sample were drawn into a syringe and expelled. After standing overnight each needle/syringe was rinsed with 200 μl of saline and flushed 10 times with the saline solution. This solution was assayed twice by ELISA (Abbott Enzyme Immunoassay).

Ten samples from the test needle/syringe combinations were found to be reactive for HIV-1 antibodies confirmed by Western Blot and seven were non-reactive. These results were identical to the results of the routine testing for HIV-1 antibodies done on the blood samples.

Needle/syringe combinations collected from shooting galleries likewise were rinsed with 200 μl of saline, flushed 10 times and assayed in the same manner as the test needles. These procedures were similar to those used by Wolk[4] except that needles were tested more quickly and less saline was used in our study.

Needle/Syringes Not Tested

The field worker collected needle/syringe units regardless of condition or potential for testing or reuse. Sixty-two of the units collected could not be tested because of physical damage to the syringe (58 percent) or a clogged/damaged needle (48 percent). Needles and syringes were collected for three days during a subsequent week until 50 testable combinations had been received from each site. In two cases, results of HIV-1 testing were indeterminate on Western Blot, and were excluded from the analysis.

Analytical Procedure

Differences in proportions and 95 percent two-sided confidence intervals around the difference were calculated.

Results

Fifteen (10.1 percent) of the 148 needle/syringe combinations tested were found to be positive for HIV-1 (Site 1 = 10.2%; Site 2 = 12.3%, Site 3 = 8.0%) and 133 were negative (89.9 percent). There were no differences in seropositivity by day of the week on which needles were collected.

The relation between the graded condition of the needle/syringe combinations and the presence of HIV-1 antibodies is presented in Tables 1 and 2. Twenty percent of the equipment combinations which had visible remnants of blood had evidence of HIV-1 antibodies, compared to 5.1 percent in combinations without blood. Thus the risk of having an infected needle/syringe was approximately four times higher if the needle/syringe appeared to be bloody. No differences in seropositivity were observed when syringe and needle combinations which contained no visible blood were partitioned into clean (4.7 percent) and dirty (5.5 percent) categories (Table 2).

TABLE 1—Seropositivity of Needle/Syringe Combinations without and with Visible Blood

Appearance	Total Tested	Proportion HIV Ab+*
	N	%
No Visible Blood	98	5.1
Visible Blood	50	20.0
Total	148	10.1

*Difference in proportions 0.149 (0.06, 0.21)

TABLE 2—Seropositivity and Visual Appearance of Needle/Syringe Combinations without Visible Blood

Appearance	Total Tested	Proportion HIV Ab+*
	N	%
Clean	55	5.5
Dirty	43	4.7
Total	98	5.1

*Difference in proportions 0.008 (−0.06, 0.12)

Discussion

This study confirms epidemiological evidence that the use of needles belonging to shooting galleries can transmit HIV-1. Ten percent of the needle-syringe combinations which were tested from three shooting galleries were positive for antibodies to HIV-1. The use of such needles presents a clear public health hazard.

Shooting galleries provide needle and syringe combinations for "rent" to drug users. The combinations usually are held by the gallery operator or laid out on a table and are selected for use by either the operator or customer depending on house rules. Water and in some cases, alcohol or bleach, are available to rinse the works, i.e., to clean the syringe and needle. The custom in many galleries is to clean the works after injecting—not prior to injecting. However, there are no mechanisms to assure that works are cleaned after use and in many instances we found they were not cleaned. Moreover, cleaning often entailed simply rinsing with water rather than bleach or alcohol.

The data from this study suggest that the choice of clean-appearing injection equipment does not eliminate the possibility of using an HIV-1 infected needle. Needle and syringe combinations which contain visible blood were more likely to be positive for antibodies to HIV-1 than those that do not contain visible blood, but the selection of a needle without visible blood was no assurance of safety. This is important because drug users may mistakenly believe that they are capable of determining which used works are "clean" i.e., safe.

Works rented in most shooting galleries are returned for future use until they are no longer functional. In this study, 29.2 percent of the collected needle/syringe combinations were clogged and could not be tested. No attempt was made to test these non-functional needle/syringe combinations because of the risk of a needle stick or contamination by

laboratory personnel (who would have had to remove needles to test the contents of the clogged needle or syringe). However, 60 percent of the untested combinations contained blood residue while only 33 percent of those tested contained visible blood. Thus, our estimate of seropositive needle-syringe combinations probably is a conservative estimate of seropositivity in usable needle/syringe combinations which at the time they were collected were available for use by shooting gallery customers.

Our choice of shooting galleries was not by random sample and does not purport to represent all shooting galleries in the area. For obvious reasons, it is not possible to draw up an inclusive list of shooting galleries from which to randomly select representative galleries. However, those studied were three of the largest and most frequented galleries in the South Florida area. The operators of each gallery reported they serviced an average of 125 intravenous drug users per week and many of these clients rented works on more than one occasion.

As with other drug use issues, the solution to the problem posed by HIV-1 contaminated needles and syringes is not simple. Intravenous drug users frequent shooting galleries for a variety of reasons.[5,6] In communities such as Miami where new syringes may be purchased by prescription only and possession of drug paraphernalia is illegal, galleries provide the user with a place to inject which is relatively safe from police intervention. Shooting galleries increase the availability of needle and syringe combinations and some also sell drugs. Galleries provide a place where drug users can socialize with other drug users. At some locations prostitution is common. Even faced with evidence that shooting galleries are sources of HIV-1 infection, many users still frequent galleries.

Intervention programs must be developed to reach intravenous users who continue to use shooting galleries. Both the users and gallery operators need to be educated on the necessity of either using new works or cleaning works and need to be taught proper cleaning techniques. Gallery operators exercise control over injection behavior at their locations. If they become convinced that sterile needles and syringes are essential, those central figures could have considerable influence upon a large number of other users. For example, two gallery operators in Miami have been observed requiring all customers to purchase new works.

If risk reduction is to occur among users who continue to inject drugs, techniques to clean works must be incorporated into the value system of the operators of the galleries as well as the individual parenteral drug users and made a part of the shooting behavior of those users who frequent shooting galleries.

ACKNOWLEDGMENTS

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NIH Consensus Statement on Oral Complications of Cancer Therapies

On April 17–19, 1989, the National Institute of Dental Research (NIDR) and the Office of Medical Applications of Research (OMAR) of the National Institutes of Health (NIH) held a consensus development conference on *Oral Complications of Cancer Therapies: Diagnosis, Prevention, and Treatment*. After hearing presentations on the subject by scientific experts, a consensus panel issued its report containing the conclusions and recommendations of the conferees. Free single copies of the consensus statement may be obtained from:

William H. Hall
Directior of Communications
OMAR, NIH, Bldg. 1, Room 259
Bethesda, MD 20892
Tel: (301) 496-1143

OMAR will also provide multiple copies of a two-page summary of the consensus statement to organizations that wish to distribute it to members of their organization. Contact Mr. Hall's office and advise him of the number of copies required.

Occupational and Other Exposures Associated with Male End-Stage Renal Disease: A Case/Control Study

N. Kyle Steenland, PhD, Michael J. Thun, MD, MPH, C. William Ferguson, BA, and Friedrich K. Port, MD

Abstract: We conducted a case-control study of 325 men ages 30–69 who were diagnosed with end-stage renal disease (ESRD) between 1976 and 1984, and resided in four urban areas of Michigan in 1984. Cases were selected from the Michigan Kidney Registry and excluded men with diabetic, congenital, and obstructive nephropathies. Controls were selected by random-digit dialing and were pair-matched to cases for age, race, and area of residence. Telephone interviews were conducted with 69 percent of eligible cases and 79 percent of eligible controls. Risk of ESRD was significantly related to phenacetin or acetaminophen consumption (odds ratio(OR) = 2.66), moonshine consumption (OR = 2.43), a family history of renal disease (OR = 9.30); and regular occupational exposures to solvents (OR = 1.51) or silica (OR = 1.67). Particular occupational exposures with elevated risk were solvents used as cleaning agents and degreasers (OR = 2.50) silica exposure in foundries or brick factories (OR = 1.92), and silica exposure during sandblasting (OR = 3.83). Little or no trend of increased risk with duration of exposure was found for these occupational exposures, with the exception of silica in sandblasting. Limitations of these data include representativeness of cases, possible overreporting by cases, and misclassification of exposures inherent in self-reports. (*Am J Public Health* 1990; 80:153–159.)

Introduction

End-stage renal disease (ESRD) is a major public health problem in the United States. In 1985, over 120,000 Americans suffered from ESRD, and each year approximately 30,000 new cases arise. Cases require dialysis or transplant, and the current annual cost exceeds $2 billion a year, almost entirely paid for by the federal government.[1] Yet very little is known about the causes of ESRD. A minority of ESRD cases are characterized etiologically (e.g., lead nephropathy, analgesic nephropathy, diabetic nephropathy).[2] Many cases are described histologically (e.g., glomerulonephritis, interstitial nephritis). Others are described as "hypertensive nephrosclerosis," with no determination of whether the kidney damage preceded or followed the hypertension.

Acute exposures to heavy metals can cause acute renal disease or dysfunction. There is also some indication that occupational exposures to metals, solvents, and silica may play a role in chronic renal disease.[2] Evidence indicates excess renal disease among workers exposed chronically to uranium,[3] cadmium,[4] and lead.[5–8] In addition, animal studies have shown that high exposures to carbon tetrachloride or perchloroethylene can also cause acute kidney damage.[9] In recent years, case-control studies have indicated that chronic glomerulonephritis is associated with occupational exposure to hydrocarbons.[10–15] Evidence of silica-related renal disease is limited to case reports[16,17] of acute renal failure following high exposures to silica.

To further investigate the hypothesis that occupational exposures might be associated with end-stage renal disease, we have conducted a case-control study of males diagnosed with ESRD between 1976–1984 in four urban areas of Michigan.

Methods

Cases

Patients were identified from the Michigan Kidney Registry,[18,19] which registers all new cases of ESRD in Michigan, collects demographic data, and records the diagnosis provided by the attending physician at the time of the ESRD diagnosis. Eligible cases were all men diagnosed between 1976 and 1984 with selected diagnoses of ESRD, ages 30–69 at diagnosis, and living in four urban areas in southeastern Michigan in 1984. Cases were restricted to those still living because of the unreliability of next-of-kin for reporting detailed exposure history. Diagnoses excluded were diabetic nephropathy, polycystic kidney disease, heroin nephropathy, lupus nephropathy, nephropathy due to malignancy, Alport's syndrome, unspecified chronic renal failure, nephropathy due to obstruction, and a variety of less common nephropathies grouped as "other." These diagnoses were excluded because most have known nonoccupational causes. The diagnoses included were glomerulonephritis, hypertensive kidney disease (nephrosclerosis), and interstitial kidney disease (including pyelonephritis, lead nephropathy and other unspecified interstitial disease). These diagnosis included represented about 65 percent of the ESRD patients in Michigan.

The summary diagnoses in the Michigan Kidney Registry may not be accurate, since many of ESRD patients present with advanced disease, and only an estimated 5 percent or less of ESRD patients have been biopsied. For example, some physicians may have classified a case as nephrosclerotic on the basis of high blood pressure at the time of diagnosis, without further data indicating that the high blood pressure preceded the kidney disease. While the diagnoses available to the Registry were sufficient to exclude certain well-defined entities such as diabetic nephropathy, the diagnoses of the cases which were included (nephrosclerosis, glomerulonephritis, and interstitial nephritis) were not clearly differentiated one from another, and were therefore analyzed together.

The Michigan Kidney Registry identified 612 eligible cases. Contact was initially made by mail, with phone calls to nonrespondents. Eighty-seven of these men had died prior to any attempt to contact them, and 14 died after consenting to be interviewed but prior to being interviewed; another 26 men were found upon interview to be ineligible because they had kidney disease subsequent to diabetes or prolonged heroin use; and 14 men could not participate either because they were in prison or were mentally incompetent. Of the remaining 471 men, we interviewed 325 (69 percent). More nonre-

From the National Institute for Occupational Safety and Health (Steenland, Thun) and the Michigan Kidney Registry (Ferguson, Port). Address reprint requests to N. Kyle Steenland, PhD, NIOSH, R13, Robert A. Taft Laboratories, 4676 Columbia Parkway, Cincinnati, OH 45226–1998. This paper, submitted to the Journal February 6, 1989, was revised and accepted for publication June 26, 1989.

spondents than respondents were Black (67 percent vs 44 percent) and were living in inner Detroit (61 percent vs 40 percent).

Controls

Controls were chosen from the general population in the four urban areas of the study, via random-digit dialing. Controls were pair-matched to cases on age (within five years), race (White versus other), sex, and residential area based on telephone prefix, grouped in 15 geographical areas covering the entire study area. Men who had chronic kidney disease were excluded.

Random digit-dialing was conducted according to a modified Waksberg method[20] from February to October 1985. A potential control identified in calling the randomized number was allowed to match any case of the same age and race in the same geographical area. If numbers did not answer, repeated phone calls were made varying the time of day and day of week prior to abandoning the phone number.

A total of 3,962 numbers were called (with an average 1.95 calls per number). Of these, 61 percent (2,430) were working residential numbers. Twenty-three percent (567) of those who answered at a residential phone refused to provide initial information about males in the household. Once a household did provide such initial information and proved to have an eligible male, 79 percent (325/410) agreed to be interviewed. We compared the 325 controls (respondents) with the 85 individuals who were eligible but refused the interview (refusals). Potential controls who refused differed from respondents regarding race (55 percent Black versus 44 percent), and residence (60 percent from inner Detroit, versus 40 percent).

Interviews

Telephone interviews lasting 30–45 elicited demographic, medical history, and occupational histories. Cases were interviewed from November 1983 through December 1985, while controls were interviewed from March 1985 to January 1987. Interviewers knew which respondents were cases from the answers to a variety of questions. Neither cases nor controls were aware of the hypotheses of the study.

Respondents were asked about regular moonshine use, regular pain pill use, family history of serious kidney disease, years of education, smoking, lead poisoning, injected antibiotics, and environmental exposure to metals and solvents. Exposures were truncated at the year of diagnosis as end-stage for cases and their matched controls. Phenacetin or acetaminophen use was determined by a review of answers to a question about the use of pain pills on a regular basis (more than one pill per week for two years or more). Brands of analgesics were classified by whether or not they contained phenacetin or acetaminophen in the early 1970s, based on data provided by Dr. Dale Sandler at the National Institute of Environmental Health (personal communication). Acetaminophen was grouped with phenacetin because it is a metabolite of phenacetin and may also be associated with renal disease.[21]

Family history of renal disease was considered positive if kidney disease (excluding stones and cancer) occurred among parents, children, or siblings, and was not known to have occurred subsequent to diabetes or trauma.

Each job held for more than six months since age 18 was classified for occupation and industry using 1980 US Census codes.[22] For each job, questions were asked regarding regular exposure to solvents, metal particles, metal fumes, mercury, oil and gasoline, and ammonia. Ammonia was included as a check, since it was not thought to be related to kidney disease. For each material, details of the exposure (e.g., hours per week, process description) were recorded. Work history for cases and their matched controls was considered only up until the year of the diagnosis as end-stage for the case.

We reviewed the completed interviews without knowledge of case-control status and with the assistance of NIOSH (National Institute for Occupational Safety and Health) industrial hygienists. For positive responses to questions about metal fumes, metal particles, solvents, and silica, we created sub-categories by type of solvent, metal, etc (Table 2). When an individual had been unable to recall the number of hours per week during which he was exposed, we arbitrarily assigned five hours a week for infrequent exposure, 20 hours a week for frequent but not constant exposure, and 40 hours a week for constant exposure. In a few cases, a self-reported exposure to a substance was clearly wrong due to misclassification by the respondent, and we corrected the response (e.g., self-reported exposure to solvents such as "lime" or "pesticides"). In other cases, we called a respondent back for clarification.

Analysis

Odds ratios (OR) were estimated from conditional logistic regression.[23] Odds ratios for exposure were adjusted for confounding by nonoccupational variables and other occupational variables. Dummy variables (missing versus not exposed) were added to the model to account for those instances in which study subjects were missing data for lifestyle variables. When subjects were missing data on exposure, they were called again to resolve the problem. No cases or controls were lost at the analysis stage due to missing data.

We compared the occupational history of cases and controls in several ways:

- We considered a history of having ever been exposed to specific substances (e.g., all solvents, or solvents used as degreasers).
- We estimated cumulative exposure by multiplying the number of hours per week a man was exposed by the number of years in the job for each job, and summing over all jobs.
- Finally, for solvents, we used an exposure matrix developed by Shalat and others[24] to identify jobs likely to have involved high exposure to solvent (see Appendix). Cases and controls were then compared regarding work in these jobs. Analyses were also run which discounted any exposures (lifestyle or work history) which occurred in the five years preceding the date of diagnosis of the case.

Results

Table 1 gives the frequency of a number of lifestyle variables among (unmatched) cases and controls. After controlling for confounding by years of education and date of birth, phenacetin/acetaminophen use and moonshine use were both significant predictors of kidney disease, with odds ratios of 2.66, (95% confidence intervals 1.04, 6.82) and OR 2.42 (95% CI 1.10, 5.36), respectively. Cumulative measures of exposure for these variables showed positive but nonsignificant trends of increased risk with increased duration of use. The findings for family history were the most remarkable, with cases much more likely than controls (OR 9.30, 95% CI 7.99, 10.82) to have a close family member (parent,

TABLE 1—Demographic Characteristics of Study Population

Characteristics	Cases (n = 325)	Controls (n = 325)
Average years of education	11.7	12.5
Number non-White*	143	143
Number living in inner Detroit	130	130
Number living in outer Detroit	114	114
Number living in Flint, Saginaw, or Lansing	81	81
Some college	106	129
Average date of birth	1930.0	1930.5
Family kidney disease	37	7
Regular moonshine use	31	10
Regular use of phenacetin/acetaminophen	22	7
Current cigarette smokers**	143	151
Former cigarette smokers	119	91
Average years smoked, current smokers	35.8	34.4

*93% of non-Whites were Black.
**Includes men who quit within one year prior to their diagnosis.

child, sibling) with kidney disease (excluding kidney stones and kidney disease subsequent to trauma or diabetes). When any exposure within five years of the date of diagnosis of the case and his matched control was ignored, the results for moonshine consumption were unchanged, while the odds ratio for phenacetin/acetaminophen fell slightly from 2.66 to 2.47 (0.86, 7.12).

Controls were more likely ever to have worked in jobs as professionals, managers, or technicians (56 percent vs 46 percent). The most common industry for both cases and controls was the automobile industry, in which 51 percent of cases and controls had worked. The next most common was construction (17 percent), followed by foundries (9 percent), and hospitals (6 percent).

Table 2 presents the results of a logistic regression for occupational variables. Adjusted odds ratios were significantly elevated for exposure to solvents used as degreasers and silica used in foundries or in sandblasting. An elevated odds ratio was also observed for exposure to lead. No interaction terms were significant between any exposure variables and any lifestyle variables or other exposure variables.

Most men exposed to solvents used as degreasers worked in metal manufacturing industries (Census codes 271–370, 47 cases, 22 controls). Within this sector, the principal industry was auto manufacturing (32 cases, 17 controls). About equal numbers of cases and controls were exposed in dry cleaning shops (six cases, five controls). The subcategories of silica with elevated risk were exposure in foundries, brick making, or sandblasting, which were the categories likely to have involved the highest exposures.

There were no strongly positive trends of increased risk with increased duration of exposure for any exposure variable, with the exception of silica used in sandblasting. The odds ratio for exposure to silica in sandblasting for 2.5 years for 40 hours a week (cumulative duration = 100, an average exposure) was 2.74 (.69, 9.97).

We also considered employment in jobs considered to have had high solvent exposure (see Appendix). There were no differences between cases and controls regarding either ever having worked in these jobs (31 cases, 30 controls) or the length of time having worked in these jobs (5.3 years for cases, 6.8 for controls).

The above results for occupational variables were changed little when all exposures in the five years preceding the date of diagnosis of the case (and matched control) were ignored. For example, the odds ratio for ever having been exposed to solvents increased from 1.51 to 1.68 (1.13, 2.48), while the odds ratio for ever having been exposed to silica increased from 1.67 to 1.81 (1.09, 3.02). The coefficients for cumulative duration of exposure also were affected only slightly (for solvents, the coefficient changed from −.0000095 to .00009, while for silica it changed from −.0039 to −.0004).

Discussion

This study suffered from several limitations inherent in an interview study of ESRD patients. First, patients are often

TABLE 2—Adjusted Logistic Regression Results for Occupational Variables

Exposure	Number Cases Exposed	Number Exposed	Odds Ratio* (95% CI) Ever-Never Exposed*
All solvents	124	82	1.51 (1.03, 2.22)
Solvents used in paints and glues	38	34	1.01 (.58, 1.74)
Solvents used as cleaning agents or degreasers	94	40	2.50 (1.56, 3.95)
Solvents used in other processes	17	13	1.05 (.44, 2.48)
Metal fumes	139	94	1.17 (.77, 1.80)
Lead (soldering and other)	32	16	1.73 (.82, 3.65)
Iron/steel	109	72	1.15 (.73, 1.80)
Welding fumes	51	44	0.75 (.44, 1.28)
Metal particles	119	96	0.97 (.58, 1.48)
Oil and gas	135	129	0.74 (.64, 1.51)
Gas and diesel fuel	74	65	0.98 (.49, 1.06)
Motor and fuel oil	68	49	1.13 (.69, 1.84)
Cooling fluids	26	40	0.50 (.27, .92)
Silica	87	54	1.67 (1.02, 2.74)
Cement and sand	17	18	0.78 (.34, 1.78)
Brick and foundry	57	31	1.92 (1.06, 3.46)
Other silica	16	12	1.08 (.42, 2.77)
Sandblasting	9	3	3.83 (.97, 15.19)
Ammonia	33	19	1.31 (.66, 2.60)

*Models included variables for date of birth, years of education, regular use of moonshine, and family history of kidney disease, metal fumes, solvents, and silica.

quite ill and difficult to interview. This may contribute to a high refusal rate. Second, 482 men had been diagnosed between 1976–84 and would have been eligible for our study but had died by 1984, so that our study was of a population of survivors. A demographic analysis of those who died before they could enter the study indicated they differed little on race or residence, but were approximately five years older than men who were studied. A bias in our results could exist if these decedents differed substantially in their occupational exposures with those who survived to be studied. It is possible, for example, that those who died were of lower socioeconomic status and had different exposures than those who survived, although it is not clear that this would either increase or decrease odds ratios. We did not attempt to use of surrogate interviews by next-of-kin because next-of-kin could not report the decedent's detailed occupational histories. Third, it is difficult to study separate diagnostic groups among ESRD patients. We did not have confidence that the summary diagnosis in the Registry was sufficiently accurate to allow us to divide our data into the principal diagnostic groups (glomerulonephritis, nephrosclerosis, pyelonephritis) for diagnosis-specific analyses. Many of the above difficulties could be overcome by studying individuals with less severe kidney disease, prior to end-stage. With little data available to date, it is not clear whether occupational associations are more or less apparent in patients with end-stage versus less severe disease.

As in many case-control studies, we relied on self-reports to determine exposures. Cases may have had a tendency to overreport exposures compared to controls. We included a variable (ammonia exposure) thought not to be related to renal disease to test such overreporting. Cases did report more exposure to ammonia than did controls, although numbers were small and the difference was not statistically significant. Complicating any assessment of overreporting among cases was the fact that cases were more likely to have had less education than controls, and were more likely to have worked in blue-collar jobs than controls. Hence, they were more likely to have truly been exposed to occupational agents. This increased likelihood of "blue collar" status among cases compared to controls may have been an artifact of the random-dial dialing process. An alternative explanation is that our findings may represent a real phenomenon, whereby people with renal disease are of a lower socioeconomic strata than those without renal disease. While the published literature provides little data to support this thesis, one recent report reached similar conclusions.[21] To control for the observed confounding effects of socioeconomic class in our study, we have included a variable in the model for years of education.

Regarding nonoccupational variables, the effect of moonshine on renal disease is presumably due to contamination by lead.[25] Other studies have indicated that analgesics, particularly phenacetin, may be associated with renal disease (see the consensus report[26]).

Regarding occupational variables, our findings of elevated risk associated with self-reported exposure to solvents (used as cleaning agents or degreasers) and silica (used in foundries or brick manufacturing, and in sandblasting) are consistent with reports of renal disease resulting from acute exposure to these substances. An effect of solvents on chronic renal disease has also been seen in case-control studies.[15]

The lack of trend with duration of exposure to solvents can be seen as weakening the observed association between solvents and ESRD. However, duration of exposure may be a poor surrogate for cumulative dose, if those with short-term exposures also have higher exposures. Similarly, the negative findings for solvents using the pre-defined "high exposure" jobs may be due to the insensitivity of the job-exposure matrix which we used. Few attempts have been made to validate job-exposure matrices (none had been attempted for the one we used), and when such attempts have been made results have not always been encouraging.[27]

The covariate with the strongest association with renal disease in our study was a history among close relatives (parents, children, siblings) of renal disease (other than stones, or kidney disease subsequent to cancer or trauma). Due to the extremely strong association (odds ratio 9.3) observed for this variable, it seems unlikely that this positive association was due to overreporting. Familial patterns are known to exist for a number of relatively rare kidney diseases, but many of these would have been excluded from our case series (e.g., Alport's syndrome, polycystic kidney disease). Family history of hypertension, however, was not examined in our study, and may have contributed to the positive association we observed.

In summary, this study found a number of associations with ESRD which, in our opinion, warrant further investigation. These associations were generally supported by other data in the literature. The epidemiology of chronic renal disease is a relatively new area of investigation with many potential benefits, in light of the high prevalence of renal disease, its high mortality rate, and its high cost of treatment.

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APPENDIX

*US Census Codes for Jobs Considered Likely to Have Had High Exposures to Solvents**

Occupation Code/Occupation	Industry Code	Industry
203 Clinical lab tech	any	any
224 Chemical tech	any	any
455 Pest control	any	any
188 Painter, sculptor	any	any
789 Handpainting, coating, decorating	any	any
579 Painters, constr and maint	any	any
736 Typesetters	any	any
734 Printing machine operators	any	any
759 Painting/paint spray machine opr	any	any
885 Garage servs/gas station attnd	any	any
764 Washing, cleaning, pickling	270–301	Metal
725 Misc. metal/plastic processing machine operator	270–301	Metal
756 Mixing/blending machine opr	181,191,192,190	Chemicals, drugs, paints
	180,210,211,212	Rubbers and plastics
757 Separating filtering mach opr	181,191,192,190, 200,201	Chemicals, drugs, paints Fuel
758 Compressing/Compacting mach opr	180,210,211,212	[illegible]ber, plastics
709 Grinding, abrasive, buffing, polishing	180,210,211,212	[illegible]lastics
705 Milling/planning mach opr	180,210,211,212	[illegible]astics
777 Misc. machine opr	180,210,211,212	[illegible]ber, plastics
755 Shaping/forming mach opr	180,210,211,212	Rubber, plastics
753 Cementing/gluing mach opr	180,210,211,212	Rubber, plastics
786 Handcuffing/trimming	180,210,211,212	Rubber, plastics
787 Hand molding/casking/forming	180,210,211,212	Rubber, plastics
796 Production inspectors	180,210,211,212	Rubber, plastics
889 Laborers	220,221,222	Leather
745 Shoe machine opr	220,221,222	Leather

*SOURCE: Shalat 1986. Shalat ranked all possible jobs on a scale of 0–3 for both likelihood and intensity of exposure. Jobs here defined as high solvent jobs were those for whom the product of intensity and exposure was five or more.

Sentinel Health Events (Occupational): Analysis of Death Certificates among Residents of Nassau County, NY between 1980–82 for Occupationally Related Causes of Death

JUDITH P. FELDMAN, MD, MPH, AND LINDA M. GERBER, PHD

Abstract: Death certificates for residents of Nassau County, New York dying between 1980 and 1982 were examined for causes of death defined as Sentinel Health Events (Occupational) {SHE(O)}. Of 16,193 deaths from all causes, 2,286 (14.1 percent) were identified as SHE(O) deaths; 142 required no match because the cause of death was inherently related to the occupation or industry. Malignant neoplasms of the trachea, bronchus, or lung were the most frequently occurring SHE(O), accounting for 60 percent of all SHE(O) deaths and 81 percent of matched SHE(O) deaths. The construction industry was associated with the vast majority of such deaths. (*Am J Public Health* 1990; 80:158–161.)

Introduction

Although accurate numbers of job-related injuries, illnesses, and deaths are difficult to obtain, estimates suggest that two to 10 million injuries, 14,000 accidental deaths, and 100,000 occupationally related illnesses result annually from workplace accidents and exposure.[1,2]

Recognition and surveillance of occupationally related deaths are difficult since only 28 of 50 states routinely code industry and/or occupation (I/O) data on death certificates,[2] and methods of record keeping, sorting and coding are not standardized.

Rutstein, *et al*, have suggested a system to identify preventable occupationally related diseases, or Sentinel Health Events (Occupational), [SHE(O)].[3] Recognition of SHE(O)s may aid in the control of occupational diseases by providing the impetus for epidemiologic or industrial hygiene studies, or serving as a warning signal that material substitution, engineering control, or personal protection of medical care may be required.

New York State is one of several states that routinely codes I/O data on death certificates. The New York State Department of Health had recently implemented coding occupational and industry entries on death certificates according to the US Bureau of the Census coding system,[4] and, at the time this study was undertaken, had completed coding on death certificates between 1980 and 1982.[5]

Nassau County on Long Island has a population of 1.3 million,[6] and a diverse industrial and occupational makeup.[7,8] Because of its large population and varied economic environment, a study was developed to implement Rutstein's SHE(O) system to analyze occupationally related disease patterns and examine whether any particular examples of occupations or industries put workers in Nassau County at increased risk for disease.

Methods

All death certificates recorded by the New York State Department of Health for residents of Nassau County whose age at death was between 18 and 74 years, and whose date of death was between January 1, 1980 and December 31, 1982, were examined. Death certificates had been assigned an ICD(9) code based on the diagnosis most likely to have been the underlying cause of death. In addition, a code was assigned for both the occupation and industry listed on the certificate.

Using Rutstein's list of occupations and industries, death certificates whose cause was on the SHE(O) list were sorted into five categories: 0 = unmatched to occupation and industry; 1 = match on industry only; 2 = match on occupation only; 3 = match on both occupation and industry; and 4 = no match needed since listed cause of death was considered to be inherently related to occupation regardless of listed occupation or industry (e.g., mesothelioma).

Categories 1–4 were grouped as "SHE(O) deaths with I/O match" and compared by sex, age, race, occupation, and industry to the total deaths from all causes and to the total SHE(O) deaths (matched and unmatched).

Results

The 50 SHE(O) categories suggested by Rutstein are listed in the Appendix. Only 26 were identified as the underlying cause of death on one or more death certificates in Nassau County (shown with an asterisk in the Appendix.) There were 16,193 deaths from all causes of which 2,286 (14.1 percent) were SHE(O) deaths and 142 (0.9 percent) were matched SHE(O) deaths.

Despite the fact that males made up only 47.8 percent of the population between ages 18–74, they accounted for 60.3 percent of deaths from all causes, 66.3 percent of all SHE(O) deaths, and 90.1 percent of matched SHE(O) deaths.

Malignant neoplasms of the trachea, bronchus, or lung (respiratory tract neoplasms) were the most frequently occurring SHE(O), accounting for 59.5 percent of all SHE(O) deaths. The other frequently encountered SHE(O)s were cirrhosis of the liver without mention of alcohol [10.4 percent of all SHE(O)s], malignant neoplasms of the bladder [5.2 percent of all SHE(O)s], and malignant neoplasms of the kidney [5.7 percent of all SHE(O)s] (Table 1).

Of the 2,286 SHE(O) deaths, 142 (6.2 percent) were matched; of those, 117 (82.4 percent) matched on industry alone, 13 (9.2 percent) required no match, 11 (7.7 percent) matched on occupation alone, and one (0.7 percent) matched on the combination of industry and occupation.

Although respiratory tract neoplasms accounted for the largest proportion of the SHE(O) deaths, only 8.4 percent of

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TABLE 1—Number and Percent of SHE(O) Deaths, and SHE(O) Deaths as Percent of Deaths from All Causes According to Cause and Sex: 1980–82

Underlying Cause of Death	# SHE(O) Deaths			% SHE(O) Deaths			% Deaths from All Causes		
	M	F	Total	M	F	Total	M	F	Total
Pulmonary TB	6	3	9	0.4	0.4	0.4	0.1	0.1	0.1
Chronic hepatitis	12	4	16	0.8	0.5	0.7	0.1	0.1	0.1
Cirrhosis liver without alcohol	142	97	239	9.4	12.6	10.4	1.5	1.5	1.5
Hepatitis-non-A, non-B	1	6	7	0.1	0.8	0.3	0.0	0.1	0.0
Hemangiosarcoma liver and site unspecified	39	44	83	2.6	5.7	3.6	0.4	0.7	0.5
MN nasal cavity	3	2	5	0.2	0.3	0.2	0.0	0.0	0.0
MN larynx	46	10	56	3.0	1.3	2.4	0.5	0.2	0.3
MN trachea, bronchus, lung	936	425	1361	61.8	55.1	59.5	9.6	6.6	8.4
Mesothelioma (all)	10	2	12	0.7	0.3	0.5	0.1	0.0	0.1
MN bone	9	2	11	0.6	0.3	0.5	0.1	0.0	0.1
MN bladder	94	24	118	6.2	3.1	5.2	1.0	0.4	0.7
MN kidney	88	42	130	5.8	5.4	5.7	0.9	0.7	0.8
Lymphoid leukemia	26	6	32	1.7	0.8	1.4	0.3	0.1	0.2
Myeloid leukemia	42	39	81	2.8	5.1	3.5	0.4	0.6	0.5
Erythroleukemia	2	1	3	0.1	0.1	0.1	0.0	0.0	0.0
Aplastic anemia	1	3	4	0.1	0.4	0.2	0.0	0.1	0.0
Agranulocytosis or neutropenia	3	0	3	0.2	0.0	0.1	0.0	0.0	0.0
Toxic effects of lead or its compounds	5	0	5	0.3	0.0	0.2	0.1	0.0	0.0
Extrinsic asthma	8	14	22	0.5	1.8	1.0	0.1	0.2	0.1
Coalworkers' pneumoconiosis	1	0	1	0.1	0.0	0.0	0.0	0.0	0.0
Toxic hepatitis	4	3	7	0.3	0.4	0.3	0.0	0.1	0.0
Acute renal failure	5	9	14	0.3	1.2	0.6	0.1	0.1	0.1
Chronic renal failure	11	17	28	0.7	2.2	1.2	0.1	0.3	0.2
Renal failure-unspecified	21	18	39	1.4	2.3	1.7	0.2	0.3	0.2
Total*	1515	771	2286	100.1	100.1	99.7	15.6	12.2	14.0

*percentages may not total 100.0 due to rounding

TABLE 2—Number and Percent of SHE(O) Matched Deaths by Underlying Cause

Underlying Cause of Death [SHE(O)'s] only	Total SHE(O) Deaths	SHE(O) Matched # Deaths (%)
Pulmonary TB	9	1 (11.1)
Chronic hepatitis	16	0 (0.0)
Cirrhosis of liver without alcohol	239	8 (3.3)
Hepatitis- non-A, non-B	7	1 (14.3)
Hemangiosarcoma of liver and site unspecified	83	0 (0.0)
MN of nasal cavity	5	0 (0.0)
MN of larynx	56	2 (3.6)
MN of trachea, bronchus or lung	1361	115 (8.4)
Mesothelioma, (all)	12	12(100.0)
MN of bone	11	0 (0.0)
MN of bladder	118	0 (0.0)
MN of kidney and urinary organs	130	0 (0.0)
Lymphoid leukemia	32	0 (0.0)
Myeloid leukemia	81	0 (0.0)
Erythroleukemia	3	0 (0.0)
Aplastic anemia	4	0 (0.0)
Agranulocytosis or neutropenia	3	0 (0.0)
Toxic effects of lead or its compounds	5	0 (0.0)
Extrinsic asthma	22	0 (0.0)
Coalworker's pneumoconiosis	1	1(100.0)
Toxic hepatitis	7	0 (0.0)
Acute renal failure	14	0 (0.0)
Chronic renal failure	28	1 (3.6)
Renal failure-unspecified	39	1 (2.6)
Total	2286	142 (6.2)

them were matched. Although 14.3 percent of non-A, non-B hepatitis, and 11.1 percent of tuberculosis cases were matched, the absolute numbers (one each) that were matched were very small. The 12 mesothelioma and one coal workers' pneumoconiosis were matched in 100 percent of the cases, but these diseases are by definition matched on diagnosis alone and fall into category 4 of "no match needed" (Table 2).

The 114 industry-matched respiratory tract neoplasms accounted for 80.3 percent of all matched deaths and 97.4 percent of all industry-matched deaths. Of the 13 deaths that required no match, 12 (92.3 percent) were due to mesothelioma (Table 3). For all respiratory tract neoplasms, the most commonly listed occupations were "housewives" (16.1 percent) and "managers and administrators not elsewhere classified" (8.4 percent); the most commonly listed industries were "private homes" (housewives) (16.2 percent) and "construction" (7.6 percent). For matched respiratory tract neoplasms, "construction laborers, plumbers, pipefitters and steamfitters, carpenters, construction supervisors not elsewhere classified" (7.8 percent) and "managers and administrators not elsewhere classified" (7.8 percent) were the most frequently reported occupations. "Construction" (87.0 percent), and "miscellaneous fabricated metal products" (5.2 percent) were the most frequently reported industries.*

Discussion

While the categories of SHE(O)s that were identified probably reflect the occupational, industrial, and environmental make-up of Nassau County, they might also reflect a lack of time or interest on the part of physicians in obtaining occupational histories. For example, despite the fact that there were 12 reported deaths from mesothelioma (virtually always caused by asbestos and usually easily recognized), there were no reported deaths from asbestosis (also caused by asbestos but not easily recognized in the absence of a good occupational history).

That men accounted for 60.3 percent and women only 39.7 percent of deaths from all causes can be explained by the fact that only ages 18–74 were analyzed. Women have a life expectancy at birth of 7.5 years greater than men.[9] Similarly, the fact that Whites accounted for 92.0 percent of all deaths

*Additional details available on request to author.

TABLE 3—Underlying Cause of Death by Type of Match, SHE(O)s Only

Underlying Cause of Death	Total with Match	Industry Only	Occupation Only	Both I and O	No Match Needed
Pulmonary TB	1	0	1	0	0
Cirrhosis of liver without alcohol	8	1	7	0	0
Hepatitis-non-A, non-B	1	0	0	1	0
MN larynx	2	2	0	0	0
MN trachea, bronchus, or lung	115	114	1	0	0
Mesothelioma (all)	12	0	0	0	12
Coalworkers' pneumoconiosis	1	0	0	0	1
Chronic renal failure	1	0	1	0	0
Renal failure-unspecified	1	0	1	0	0
Total	142	117	11	1	13

can be explained by the fact that Whites make up 91 percent of the total population of Nassau County.[8]

Women accounted for a disproportionately small number of deaths in the SHE(O) category and in the matched SHE(O) category. Many of the diseases listed as SHE(O)s have a latency period of 10–30 years. Deaths in 1980 from SHE(O)s might well be a result of exposure in the 1950s and 1960s when only 25–30 percent of the women were in the labor force.[10] Moreover, many women are classified as "housewife" on their death certificates. Those who may have died from an occupationally related illness would not be identified through this system.

The most striking findings of this study are that while respiratory tract neoplasms accounted for only 8.4 percent of all deaths, they accounted for 59.5 percent of all SHE(O)s and 81.0 percent of the matched SHE(O)s, and that while the construction industry was associated with only 5.9 percent of all deaths and 6.6 percent of all SHE(O) deaths, it was associated with 72.1 percent of all matched SHE(O) deaths and 87.0 percent of all matched deaths from respiratory tract neoplasms. It is difficult, however, to interpret these findings. Although in the construction industry asbestos is a major risk factor for lung cancer, smoking is the single most important cause of these deaths, and enhances the carcinogenic risk of asbestos.[11] Since laborers tend to be heavy smokers,[12] the apparent high proportion of respiratory tract cancers among construction workers may be due to the increased rate of smoking in this population, as well as to asbestos exposure. Smoking history was not available on the death certificates.

Death certificates have other limitations. The accuracy of occupational data on death certificates when compared to information obtained from medical records, union records, and personal interviews has ranged from 51 to 93 percent.[13–16] Occupational data requested on the death certificate is for "usual occupation' " with space for only one entry. However, few people work in a single job or are exposed to a single agent in a working lifetime. Because of the latency period for many diseases, an early job with toxic exposure may not be the "usual job" listed. Occupational categories are general, and there is no information on actual job description, or working conditions.

Finally, because of the latency period for many diseases on the SHE(O) list, the use of the death certificates to evaluate occupational exposure gives information in many instances on exposure that occurred years ago. Since people move, exposure and death may not necessarily have occurred in the same geographic area, thus limiting the usefulness of death certificates in pinpointing occupational hazards except for substances causing diseases with relatively short latency periods.

As the number of potentially toxic substances added to the workplace grows, it becomes increasingly important to identify occupations and industries that pose a threat to workers' health. Rutstein's scheme is an innovative step toward that goal. However, the limitations of death certificates underscore the need for further work in this area, especially for improved reporting systems and data collection methods.

One promising step in that direction is the implementation by the New York State Department of Health of occupational registries for individuals in certain high-risk jobs, with exposure to known carcinogens, or with traces of certain heavy metals in their blood. All businesses employing 100 or more people are required to report annually to the State the names of those employees who fall into any of the aforementioned categories. Such individuals could then be followed for early disease that might be related to their on-the-job exposures.

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APPENDIX
ICD(9) Codes Searched as Sentinel Health Events (Occupational)[+]

Underlying Cause of Death	ICD(9) Code
* Pulmonary TB	0.11.0–011.9
Silicotuberculosis	502
Plague	020.0–020.9
Tularemia	012.0–012.9
Anthrax	022.0–022.9
Brucellosis	023.0–023.9
Tetanus	037
Rubella	056.0–056.9
Hepatitis A	070.0–070.1
Hepatitis B	070.2–070.3
* Chronic hepatitis	571.4
* Cirrhosis of the liver (non-alcoholic)	571.5, 571.8, 571.9
* Hepatitis- non-A, non-B	070.4–070.9, 573.5
Rabies	071
Ornithosis	073.0–073.9
* Hemangiosarcoma of the liver, and, site unspecified	155, 171.5, 171.9
* MM** of the nasal cavity	160.0, 160.1, 160.3, 160.4, 160.5, 160.8, 160.9
* MN of the larynx	161.0–161.9
* MN of the trachea, bronchus or lung	162.0–162.9
* Mesothelimoa	
* Retroperitoneum and peritoneum	158.0–158.9
* Pleura	163.0–163.9
* MN of the bone	170.0–170.9
MN of the scrotum	187.7–187.9
* MN of the bladder	188.0–188.9
* MN of the kidney and urinary organs	189.0–189.9
* Leukemia	
* Lymphoid	204.0–204.9
* Myeloid	205.0–205.9
* Erythroleukemia	207.0–207.8
* Aplastic anemia	284.8–284.9
Acquired hemolytic anemia	283.1–283.9
* Agranulocytosis or neutropenia	288.0, 288.9
Methemoglobinemia	289.7
Toxic encephalitis	323.7
* Toxic effect of lead and its compounds (including fumes)	984
Parkinson's disease-secondary	332.1
Cerebellar ataxia	334.3
Inflammatory and toxic neuropathy	357.7
Extrinsic allergic alveolitis	495.0–495.9
* Extrinsic asthma	493.0, 493.9
Detergent asthma	507.8
* Coalworkers' pneumoconiosis	500, 505
Asbestosis	501
Silicosis and talcosis	502
Chronic beryllium disease	503
Byssinosis	504
Acute bronchitis, pneumonitis and pulmonary edema due to fumes and vapors	506.0–506.9, 507.1
* Toxic hepatitis	570, 573.3
* Acute renal failure	584.0–584.9
* Chronic renal failure	585
* Renal failure, unspecified	586

[+]adapted from Rutstein *et al.*[4]
*causes of death and accompanying ICD codes that were found on at least one death certificate in Nassau County between 1980–82; SHE(O)s without an asterisk were not found on any death certificate.
**MN signifies malignant neoplasm.

A Statewide Case Registry for Surveillance of Occupational Heavy Metals Absorption

MICHAEL E. BASER, MS, AND DAVID MARION, BA

Abstract: The New York State Heavy Metals Registry is a legislatively mandated program through which clinical laboratories, physicians, and health facilities report state residents 18 years of age and older with elevated levels of lead, mercury, arsenic, or cadmium in blood or urine. From 1982–86, the current employer was determined for 95.9 percent of 3,309 cases. Occupational exposures in 328 companies accounted for 82.8 percent of cases. The majority of companies were reported for lead (247 companies, 75.3 percent of total) or mercury (47 companies, 14.3 percent of total). Of the 247 companies reported to the Registry for lead, the Occupational Safety and Health Administration (OSHA) inspected 18 of 98 companies (18.4 percent) in the manufacturing sector, but only one of 149 companies (0.6 percent) outside the manufacturing sector. We conclude that the Registry effectively detects companies with heavy metals exposures, and is an especially useful adjunct to OSHA inspections outside the manufacturing sector. (*Am J Public Health* 1990; 80:162–164.)

Introduction

Heavy metals are common contaminants of the occupational environment. Absorption of metals can cause abnormalities of biochemical, cellular, and physiological function, as well as disease or death.[1] Occupationally related diseases are under-reported,[2] and surveillance may detect industries and companies with excessive exposures.[3–5] Methods of surveillance for occupational heavy metals exposure include Occupational Safety and Health Administration (OSHA) inspections for violations of the federal lead standard,[5,6] workers' compensation claims,[7] and the National Occupational Hazard Survey.[8,9]

In New York State, the two most useful sources of biological monitoring or industrial hygiene data are: 1) the New York State Heavy Metals Registry, a case registry of persons with elevated heavy metals levels in blood or urine, and 2) results of OSHA inspections for violations of the federal lead standard. The purposes of this paper are to present the methods and results of the Heavy Metals Registry during its first five years of operation (1982–86), and to determine whether companies reported to the Registry for lead have been inspected by OSHA for violations of the federal lead standard.

Methods

Sections 22.6 and 22.7 of the New York State Sanitary Code require that clinical laboratories, hospitals, and physicians report persons with heavy metals absorption to the state health department. The health department provides reporting forms which include name and residence, age, sex, heavy metals concentrations, report source (i.e., analytic laboratory, company, hospital, or physician), and private physician. Confidentiality is protected by a public health law which restricts use of the data to scientific or medical research and renders the information inadmissible as legal evidence.

Cases are defined from the reporting form as state residents 18 years of age or older with elevated levels of lead in blood (Pb-B $\geq$ 40 μg/dL or 1.93 μmol/L) or urine (Pb-U $\geq$ 80 μg/L), mercury in blood (Hg-B $\geq$ 5 μg/L or 0.02 μmol/L) or urine (Hg-U $\geq$ 20 μg/L) cadmium in blood (Cd-B $\geq$ 10 μg/L or 0.09 μmol/L) or urine (Cd-U $\geq$ 5 μg/24 h), or arsenic in urine (As-U $\geq$ 50 μg/L).[6,10–12] In 1987, the lead reporting levels were lowered to 25 μg/dL blood and 50 μg/L urine.

Cases are interviewed when the first report is received, either by telephone or in person. The interview includes questions on name and address of employer, work history (job titles and workplace exposures), industrial hygiene and exposure monitoring programs, hobbies, medical history, and demographic information. The Standard Industrial Classification (SIC) system of categorizing the employer is used.[13]

Occupational exposure is defined from the interview as a positive response to the questions of either working with or being exposed to heavy metals; in addition, if other workers from the same company have been reported to the Registry, the employer is interviewed to determine if the case could be exposed. Nonoccupational exposures include pica in the institutionalized, poisonings, avocational use of firing ranges, and home manufacture of lead shot and sinkers.

Clinical laboratories which analyze lead in state residents must be certified through proficiency testing, whether or not the laboratory is located in the state. Clinical laboratories in addition to those listed on the reporting forms have been found through a state survey of companies which store, use, or discharge toxic chemicals.[14] Employers known to be using heavy metals are interviewed to determine the number of workers using heavy metals, whether workplace air or biological samples are monitored, and which clinical laboratory is used.

Employers of reported cases are offered industrial hygiene consultation. The private physician is contacted when high levels are reported to ensure that information concerning the employer has been received and, if necessary, to assist in contacts with the employer. Companies whose employees have especially high or persistently elevated levels may be investigated by the health department or reported to OSHA.

Results

From 1982–86, 2,862 of the 3,309 cases (86.5 percent) reported to the Registry were interviewed; of these, 2,798 (97.8 percent) provided employer information. The employer could be determined from other sources such as the labora-

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TABLE 1—Number of Cases and Companies by Industrial Sector and Metal, New York State Heavy Metals Registry, 1982–86 (occupational exposures only)

Industrial Sector	Number of Cases				Number of Companies			
	Pb	Hg	As	Cd	Pb	Hg	As	Cd
Manufacturing	1241	873	52	22	98	21	3	5
Non-manufacturing	368	127	34	4	149	28	15	4
Total	1609	1000	86	26	247	47	18	9

Pb = lead; Hg = mercury; As = arsenic; Cd = cadmium. Manufacturing sector = SIC major group 20-39; non-manufacturing sector = all other SIC codes (Standard Industrial Classification).[13]

tory report in 375 of the 448 cases (83.7 percent) who were not interviewed; thus, the current employer was known for 3,173 cases (95.9 percent of total). There were no differences in metals levels between cases whose employer could and could not be determined.

A total of 2,739 occupational cases were reported from 328 companies. The cases were predominantly male (92.0 percent) and less than 40 years of age (57.1 percent); all cases were employed at their current company for at least one month. Clinical laboratories (N = 72) reported 88.9 percent of cases. Thirty-three of the clinical laboratories, including the largest reporter, were located outside the state; out-of-state sources reported 53.3 percent of cases.

Table 1 presents the distributions of reports by industrial sector. The predominant reporting was for lead (58.7 percent of cases, 75.3 percent of companies) and mercury (36.5 percent of cases, 14.3 percent of companies). The manufacturing sector (SIC major group 20–39) accounted for 80.4 percent of cases and 39.9 percent of companies. The largest numbers of non-manufacturing sector companies were reported in the service sector (SIC major group 70–89; 58 companies, 23.5 percent of total) and in the construction sector (SIC major group 15–17; 42 companies, 17.0 percent of total).

The four-digit SIC codes of industries with 20 or more cases are presented in Table 2. This criterion is arbitrary and does not imply that significant hazards do not exist in industries with fewer cases. The largest fraction of lead cases was employed in secondary smelting and refining of nonferrous metals; of mercury cases, in the manufacture of alkalies and chlorine. Industrial instrument manufacture was notable for a large number of cases and a high median urinary mercury.

Companies reported to the Registry for lead were com-

TABLE 2—Median and Maximum Heavy Metals Levels in Industries with 20 or More Cases, by Metal and Four-Digit SIC Code, New York State Heavy Metals Registry, 1982–86 (occupational exposures only)

Four-digit SIC Code and Metal	Industry Description	Reported No. of Cases	Metals levels	
			Median	Maximum
Blood Lead			(μg/dL)	(μg/dL)
1622	Bridge, tunnel, and elevated highway construction	50	56	178
1721	Painting, paper hanging, and decorating	22	50	145
2816	Inorganic pigments	37	60	87
2819	Industrial inorganic chemicals, n.e.c.	21	45	70
2899	Chemicals and chemical preparations, n.e.c.	54	47	78
3231	Glass products, made of purchased glass	110	48	340
3341	Secondary smelting and refining of nonferrous metals	343	50	88
3362	Brass, bronze, copper, copper base alloy foundries (castings)	34	53	78
3432	Plumbing fixture fittings and trim (brass goods)	58	52	89
3494	Valves and pipe fittings, except plumbers' brass goods	101	49	83
3691	Storage batteries	146	45	65
3692	Primary batteries, dry and wet	43	51	74
3714	Motor vehicle parts and accessories	74	48	78
5084	Industrial machinery and equipment	37	52	85
5093	Scrap and waste metals	26	51	94
7539	Automotive repair shops, n.e.c.	50	47	79
Urinary Mercury			(μg/L)	(μg/L)
2812	Alkalies and chlorine	494	76	916
3497	Metal foil and leaf	39	34	363
3823	Industrial instruments for measurement, display, and control of process variables	213	145	889
3829	Measuring and controlling devices, n.e.c.	33	68	309
Urinary Arsenic			(μg/L)	(μg/L)
3861	Photographic equipment and supplies	49	80	524

n.e.c. = not elsewhere classified. SIC = Standard Industrial Classification.[13] Reporting criteria: Blood lead ≥40 μg/dL or 1.93 μmol/L, urinary mercury ≥20 μg/L, urinary arsenic ≥50 μg/L.

pared with those inspected by OSHA from 1980–85 for violations of the federal lead standard. OSHA inspections were focused on the manufacturing sector to a much greater degree than Registry reports: 130 of 157 companies (82.8 percent) inspected by OSHA were in the manufacturing sector, as compared to 98 of 247 companies (39.7 percent) reported to the Registry. OSHA inspected 18 of 98 (18.4 percent) of companies reported to the Registry from the manufacturing sector, but only one of 149 (0.6 percent) companies outside the manufacturing sector.

Discussion

The Registry has several advantages compared to other surveillance methods. It is inexpensive to operate because existing data sources are used. Apart from error in the analysis of samples, there is little uncertainty regarding exposure because cases are defined by increased biological absorption. Clinical laboratory-based reporting eliminates the need to enlist the cooperation and time of individual physicians and health facilities, whose reporting is redundant to the extent that samples are sent to clinical laboratories for analysis.

Perhaps most importantly, surveillance can be carried out in industrial sectors which have a low priority for regulatory agencies and for metals for which no governmental standards exist. OSHA inspections target private companies in the manufacturing sector, larger companies, and industries with previously reported exposures. Of the four heavy metals on which Registry data are collected, a federal standard has been promulgated for lead only.

For most companies, the source of exposure is known. Lead exposure in secondary smelting and refining of nonferrous metals (SIC 3341) and storage battery manufacture (SIC 3691) results from melting of lead alloys and dust exposure during foundry or fabrication work. Alkali and chlorine manufacture workers (SIC 2812) are employed in a chloralkali plant where mercury is used as a cathode in the electrolysis of salt to produce sodium hydroxide and chlorine gas; exposure occurs when the mercury cells are opened for maintenance. Mercury exposure in industrial instrument manufacture (SIC 3823) occurs during manual assembly and fabrication of thermometers. In a small fraction of industries, heavy metals exposures have not been previously documented. The source of exposure is apparent in some, but in others more detailed investigations are necessary.

In summary, the Registry is effective in detecting companies with heavy metals exposures, particularly those outside the manufacturing sector which are not detected by OSHA inspections. Since 1982, heavy metals registries have been established in New Jersey, California, Texas, and Maryland. We recommend adoption of this surveillance method by other state health departments, and evaluation of the feasibility of a national heavy metals absorption registry.

ACKNOWLEDGMENTS

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Call for Papers: Nursing Research

The West Virginia Nurses' Association is planning a national research symposium—"Vision of Excellence: The Decade of the Nineties"—to be held at Greenbrier Hotel, White Sulphur Springs, WV, November 9–11, 1990. This two-day symposium will cover a wide range of health-related topics. The deadline for abstracts is April 15, 1990. Selected papers will be published in the conference proceedings. For further information, contact Dr. Janet F. Wang, School of Nursing, West Virginia University, Morgantown, WV 26506; telephone: (304) 293-4297.

Lead-Induced Anemia: Dose-Response Relationships and Evidence for a Threshold

JOEL SCHWARTZ, PHD, PHILIP J. LANDRIGAN MD, MSC, EDWARD L. BAKER, JR., MD, MPH, WALTER A. ORENSTEIN, MD, AND IAN H. VON LINDERN, PE, PHD

Abstract: We conducted a cross-sectional epidemiologic study to assess the association between blood lead level and hematocrit in 579 one to five year-old children living near a primary lead smelter in 1974. Blood lead levels ranged from 0.53 to 7.91 μmol/L (11 to 164 μg/dl). To predict hematocrit as a function of blood lead level and age, we derived non-linear regression models and fit percentile curves. We used logistic regression to predict the probability of hematocrit values less than 35 per cent. We found a strong non-linear, dose-response relationship between blood lead level and hematocrit. This relationship was influenced by age, but (in this age group) not by sex; the effect was strongest in youngest children. In one year-olds, the age group most severely affected, the risk of an hematocrit value below 35 percent was 2 percent above background at blood lead levels between 0.97 and 1.88 μmol/L (20 and 39 μg/dl), 18 percent above background at lead levels of 1.93 to 2.85 μmol/L (40 to 59 μg/dl), and 40 percent above background at lead levels of 2.9 μmol/L (60 μg/dl) and greater; background was defined as a blood lead level below 1.88 μmol/L (20 μg/dl). This effect appeared independent of iron deficiency. These findings suggest that blood lead levels close to the currently recommended limit value of 1.21 μmol/L (25 μg/dl) are associated with dose-related depression of hematocrit in young children. (*Am J Public Health* 1990; 80:165–168.)

Introduction

Lead-induced anemia is produced principally by two mechanisms: impairment in heme biosynthesis, and increased rate of red blood cell destruction.[1]

Lead-induced anemia has been reported to occur in children at a blood lead level of 1.93 μmol/L (40 μg/dl).[2*] However, no clear dose-response relationships between blood lead level and hematocrit have been established. Further, almost no data are available to assess whether this relationship extends downward to blood lead levels below 1.93 μmol/L (40 μg/dl), to ascertain the fraction of children with lead-induced anemia at various blood lead levels, or to determine whether there exists a threshold, below which the association is no longer evident.

To assess dose-response relationships between blood lead level and hematocrit, we examined data obtained in 1974 during an epidemiologic study of lead exposure in children living near a lead smelter.[3]

Methods

A cross-sectional prevalence study of lead exposure in children living near a lead ore smelter in Kellogg, Idaho, conducted in August 1974, found blood lead levels of 1.93 μmol/L (40 μg/dl) and above in 285 (41.9 percent) of 919 children. Within 1.6 km of the smelter, 170 (98.8 percent) of 172 children had blood lead levels above 1.93 μmol/L (40 μg/dl), the highest being 7.91 μmol/L (164 μg/dl). Blood lead levels were positively associated with exposures to airborne lead (correlation coefficient, $r = 0.72$; $p < 0.0001$) as well as with exposures to lead in soil ($r = 0.5$; $p < 0.0001$).[4]

Blood samples (drawn in lead-free vacuum tubes) were analyzed for hematocrit by centrifugation and for erythrocyte protoporphyrin (EP) by the method of Granick, *et al.*[5] Samples were analyzed for lead by modified Delves' cup atomic absorption spectrophotometry.[6] These methods have previously been described in detail.[3]

A total of 1,058 children, ages 1 to 9 years, were identified for whom data on both blood lead and hematocrit were available. In the present analysis, we focus on the 579 children in the age group 1 to 5 years, since children in this age range appear most sensitive to the hematotoxic effects of lead.

Anemia has been defined variously, with the lower bound of the normal range of hematocrit values in young children varying from 33 to 35 per cent.[7–10] We defined anemia as an hematocrit value less than 35 percent. To assess the relation between blood lead level and hematocrit in children using this definition, we fit logistic regressions, which predict the probability of anemia at different age, sex and blood lead levels.[11]

Initial examination of a raw scatter plot of hematocrit versus blood lead level showed a non-linear dose-response relationship. There was little variation in hematocrit at low blood lead levels and a negative relation with lead at higher levels.

Rather than fitting this relationship directly in a non-linear parametric regression, which imposes an explicit functional form on the data, we employed non-linear smoothing techniques developed for robust graphical analysis.[12,13]

Results

The smoothed plot of the data follows a logistic-shaped curve (Figure 1), which is relatively flat at blood lead levels below 0.97 μmol/L, becomes increasingly steep as blood lead levels rise, and, at very high lead levels, flattens again. This curve can be represented by the logistic function:

$$Hct = A/(1 + EXP(B_o + B_1*lead + B_2*age)) + C \qquad (1)$$

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*1 μmol/L = 20.72 mg/dl blood lead.

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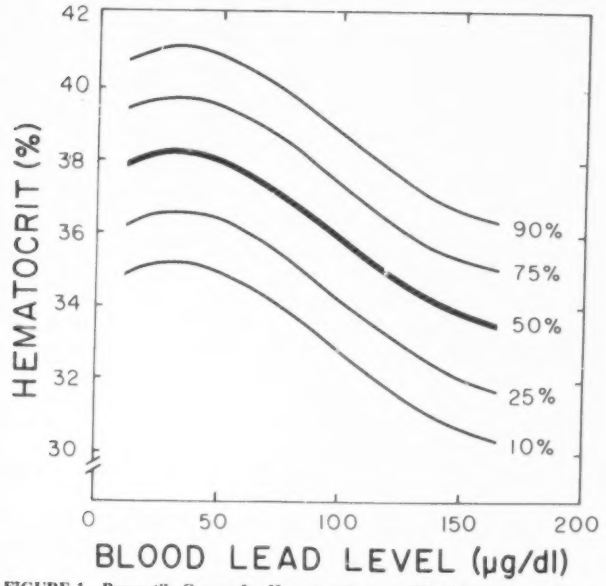

FIGURE 1—Percentile Curves for Hematocrit versus Blood Lead Level in 1–5 Year Old Children, Northern Idaho, 1974

The logistic regression analysis showed a strong association between elevation of blood lead level and the probability of anemia (regression coefficient = 0.3083; (SE = 0.0061). Year of age was also a significant predictor of low hematocrit (coefficient = −0.3831; SE 0.1134). In this age group, sex was not a predictor of anemia. The predicted probability of anemia in relation to blood lead level is plotted graphically for each year of age in Figure 2.

To illustrate the fit of the predicted curve to the actual data, Figure 3 shows both the predicted curve and actual points for the mean proportion of children with anemia for each interval of 0.48 µmol/L (10 µg/dl) in blood lead level after controlling for age. The highest point represents the mean for the five children with blood lead levels greater than 5.79 µmol/L (120 µg/dl).

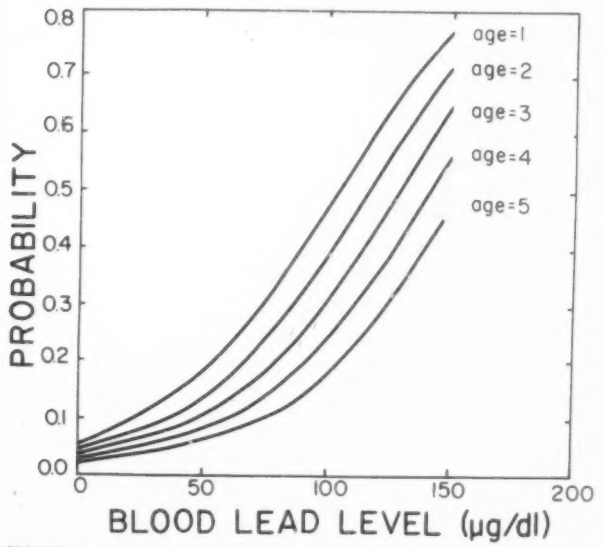

FIGURE 2—Predicted Probability of Hematocrit <35% by Age and Blood Lead Level in 1–5 Year Old Children, Northern Idaho, 1974

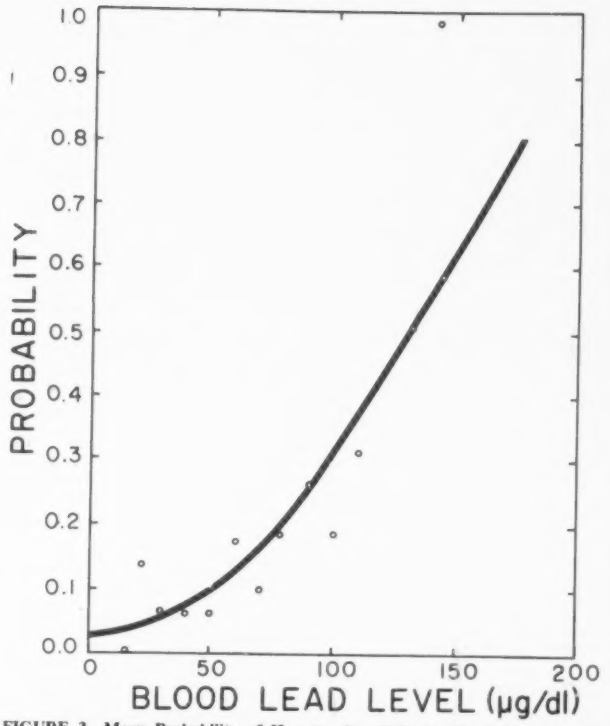

FIGURE 3—Mean Probability of Hematocrit <35% by Blood Lead Level, Northern Idaho, 1974 (This line is the fitted logistic curve for the probability of hematocrit <35% after adjusting, by regression, to the mean age)

Since Figure 2 suggests that the increased risk of depressed hematocrit begins at lower blood lead levels in one year-olds than in five year-olds, we calculated the mean proportion of children in each year of age with an hematocrit below 35 percent at various blood lead levels (Table 1). To compensate for the reduced sample size, we used increments of 0.97 µmol/L (20 µg/dl) in blood lead levels. While the sample size was too small to draw strong conclusions, the results were concordant with the regression analysis (Table 1). In one year-old children the probability of anemia was 0 percent at blood lead levels below 0.97 µmol/L (20 µg/dl), 2 percent at levels between 0.97 and 1.93 µmol/L (20 and 39 µg/dl), 18 percent at levels between 1.93 and 2.90 µmol/L (40 and 60 µg/dl), and 40 percent at blood lead levels above 2.90 µmol/L (60 µg/dl).

We also examined actual hematocrit values rather than proportion anemic. In Figure 1, the 20th, 25th, 75th, and 90th percentile curves reflect the broad variation in hematocrit levels due to age, iron deficiency and other factors. Percentile curves were estimated by confirming the homoscedasticity of the residuals from the smooth curve and then estimating their standard deviation from their interquartile range; percentiles were then calculated from the standard deviation. Table 2 presents the results of nonlinear regression of hematocrit on age and on blood lead level.

Figure 4 plots predicted hematocrit levels by age and blood lead level. Again, it shows the strong effect of age on hematocrit in young children, and demonstrates the interrelationship between age and blood lead level.

TABLE 1—Distribution of Population of Children with Anemia by Year of Age and Blood Lead Level, Northern Idaho—1974

Age (Yrs)	Blood Lead Level μmol/L*									
	0.–0.92		0.93–1.88		1.89–2.85		2.86 +		Total	
	# Anemic	Total	# Anemic	Total	# Anemic	Total	# Anemic	Total	# Anemic	Total
1	0	6	1	47	6	33	6	15	13	101
2	1	3	5	49	6	35	6	18	18	105
3	2	7	3	56	2	33	3	29	10	125
4	1	1	4	60	1	23	4	24	10	108
5	0	3	2	78	1	37	1	22	4	140
Total	4	20	15	290	16	161	20	108	55	579

*1 μmol/L = 20.72 μg dl whole blood lead level.

TABLE 2—Non-Linear Regression Model for Hematocrit versus Blood Lead Level, 1–5 Year-Old Children, Northern Idaho, 1974*

Effect	Parameter	Estimate	Standard Error	p-Value
Constant	A	39.54	0.79	0.0001
Constant	B_0	−3.112	0.446	0.0001
Blood Lead Level	B_1	0.0133	0.0041	0.0005
Age	B_2	−0.2016	0.0905	0.0129

*The parameters A, C, and B_0 all relate to the zero lead intercept and were not simultaneously significant. C was therefore deleted (reducing the error of sum squares) to give the model shown in this table.

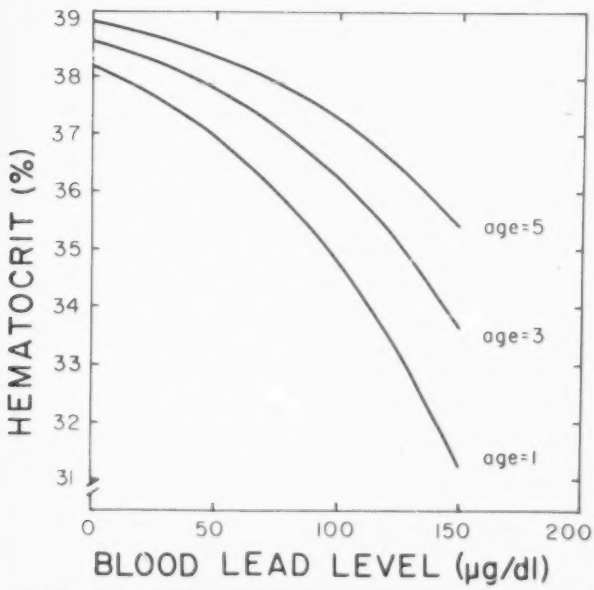

FIGURE 4—Predicted Hematocrit Value versus Blood Lead Level, by Age, in 1–5 Year Old Children, Northern Idaho, 1974

Discussion

The results of this study show a strongly negative, non-linear dose-response relationship between blood lead level and hematocrit in children, strongest in the youngest children. Although there is variability among children in the effect of lead on hematocrit, the data from this study suggest that lead-induced anemia is a biologically and clinically important consequence of lead absorption, even at low levels of exposure.

Iron deficiency may have accounted for some of the observed effect of lead on hematocrit since the effects of lead and iron deficiency on hematocrit have been shown to be strongly interactive.[14] Although data on the iron status of the children in our study were not obtained, the strong correlation between low socioeconomic status, elevated lead levels, and iron deficiency typically seen in urban populations did not exist in our study population. In our population, blood lead levels were determined principally by distance from the smelter and were not correlated with socioeconomic status,[3,4] hence lead exposure and iron deficiency were probably not correlated in these data.

The smoothed dose-response curve of hematocrit versus blood lead level (Figure 1) shows a slight, paradoxical increase in hematocrit at low blood lead levels, followed by a decrease, whose slope steepens until blood lead levels reach 4.83 μmol/L (100 μg/dl) and then subsequently flattens out again. The slight increase in hematocrit observed at the lowest blood lead levels may be artifactual. Since children with lowest blood lead levels came from a rural valley distant from the smelter town, with low family income and socioeconomic status, nutritional deficiency may have been more common there and account for the observed results. Alternatively, the effect may be due to differential partitioning of lead among the soft tissues of the body. At equilibrium, the fraction of lead stored in any body compartment will be proportional to the size of the compartment. At body burdens too low for lead to affect hemoglobin levels, higher hemoglobin levels could increase the fraction of lead stored in red blood cells thus causing the positive correlation seen here.

These data imply that there is no margin of safety between the threshold blood lead level at which lead begins to cause depression of hematocrit in young children and the blood lead level currently considered by the Centers for Disease Control to represent the upper limit of acceptability—1.21 μmol/L (25 μg/dl)[15] These findings complement other data showing deleterious effects of lead on heme synthesis of blood lead levels of 0.48 to 0.97 μmol/L (10–20 μg/dl), on vitamin D metabolism at blood lead levels below 1.21 μmol/L (25 μg/dl), on peripheral nerve conduction velocity at 0.97 to 1.45 μmol/L (20 to 30 μg/dl) and on cognitive function at 0.72 to 0.97 μmol/L (15 to 20 μg/dl).[16–22] Taken together, these findings suggest that the current recommendation on the acceptable upper limit for blood lead level in children requires reexamination.

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Call for Applications: Minority Medical Faculty Development Program 1990

The Robert Wood Johnson Foundation's Minority Medical Faculty Development Program offers four-year, postdoctoral research fellowships to minority physicians who have demonstrated superior academic and clinical skills and who are committed to careers in academic medicine and biomedical research. Each of the Fellows selected (up to eight) will receive an annual stipend of up to $50,000, complemented by a $25,000 annual grant toward support of research activities. Each Fellow will study and conduct research under the supervision of a senior faculty member located at an academic center of biomedical research noted for the training of young faculty and pursuing lines of investigation that are of interest to the Fellow. Priority will be given to basic research. It is expected that Fellows will be spending no less than 70 percent of their time in pursuit of research activities.

The Minority Medical Faculty Development Program seeks to increase the number of minority faculty who can encourage and foster the development of succeeding classes of minority physicians. At present, only 1,281 (3.2 percent) of the nation's 40,136 full-time medical faculty members are minorities.

Foundation support for the four-year fellowships will be provided initially for *two years*, with funding for the third and fourth years dependent upon review and recommendation by the National Advisory Committee. The fourth year of support may be used to assist the Fellow in his or her first year as a full-time medical faculty member.

To be eligible, applicants must be minority physicians who are US citizens, have excelled in their education, are now completing or will have completed formal clinical training (internship, residency, or clinical fellowship), and are committed to academic careers.

April 2, 1990—Deadline for request of application materials.

By April 30, 1990—Receipt of completed applications.

July 1990—Interviews with National Advisory Committee and consultants in biomedical research.

December 1990—Official notification of Fellows.

Requests for applications and inquiries should be addressed to: Harold Amos, PhD, Program Director, Minority Medical Faculty Development Program, P.O. Box 446, Brookline, MA 02147. Tel: 617/732-1947.

An Analysis of Occupational Risks for Brain Cancer

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Abstract: We evaluated the risks of brain cancer in relation to employment history in a case-control study of 312 cases and 1,248 cancer controls. Subjects were identified through the Missouri Cancer Registry for the period 1984 through 1988. Job classification was based on data routinely abstracted from hospital records. Elevated risks were identified for certain white collar occupations: for men employed in engineering, the odds ratio (OR) = 2.1; 95% confidence interval (CI) = 0.4, 10.3; for social science professionals, the OR = 6.1; 95% CI = 1.5, 26.1. Among occupations with potential exposure to occupational carcinogens, increased risks were observed for men employed in agricultural crop production (OR = 1.5; 95% CI = 1.0, 2.4), printing and publishing (OR = 2.8; 95% CI = 1.0, 8.3), and brickmasons and tilesetters (OR = 2.5; 95% CI = 0.5, 11.5). Most of elevated brain cancer risks were due to astrocytic cancers, but the excess among agricultural workers occurred in other cell types. No increase in risk was noted for current cigarette smokers (OR = 0.9; 95% CI = 0.7, 1.5) or ex-smokers (OR = 1.0; 95% CI = 0.7, 1.5). This exploratory study indicates a need for further studies of occupational risks of brain cancer. (*Am J Public Health* 1990; 80:169–172.)

Introduction

Cancer of the brain has been the focus of relatively few epidemiologic studies. Increased risks have been noted for several white collar occupations including accountants,[1] sales managers,[2] engineers,[2,3] teachers,[1] lawyers and judges,[2] and postal clerks.[3] Among occupations with potential carcinogenic exposures on the job, excess brain cancer risk has been suggested for workers employed in the rubber industry,[4,5] polyvinyl chloride production,[6–9] farming,[10–13] electrical industries,[14–16] pharmaceutical production,[17] and production of petroleum and petrochemicals.[18,19] Cigarette smoking has also been linked to brain cancer risk.[20]

Most previous studies of occupation and brain cancer have relied on mortality data, with concomitant potential for misclassification of brain cancer on death certificates.[21] Few previous studies have evaluated differences in occupational risks by histologic type of brain cancer. The current study was based on data from a statewide cancer registry.

Methods

Cases and controls were identified through the Missouri Cancer Registry, maintained by the Missouri Department of Health. The Registry has been collecting data on cancer cases from public and private hospitals since 1972, with reporting mandated by law since 1984.

The case group consisted of White males diagnosed with histologically confirmed brain and other central nervous system cancer (*International Classification of Diseases for Oncology* [ICD-O] codes 191 and 192)[22] between January 1984 and December 1988. Selection was limited to White males due to the small number of non-Whites and the lack of reported occupational diversity among females. The mean age among cases was 54.6 years.

A frequency-matched sample of controls was chosen from all other white male patients diagnosed with cancers in the same time period, excluding cancers of ill-defined or unknown primary sites (ICD-O site codes 195 and 199). For each case, four controls were randomly selected within each of six age strata. The mean age of controls was 55.6 years. The analyses included a total of 312 cases and 1,248 controls. The distribution of cancer sites among controls is shown in Table 1.

Data on occupation and tobacco use are routinely abstracted from the hospital medical record at the time of diagnosis and submitted to the Missouri Cancer Registry as part of cancer incidence reporting. Hospital registrars record information for cancer patients using a standardized protocol and the occupational data are subsequently coded by trained cancer registry specialists using the three-digit 1980 US Census codes.[23] The occupational history reported to the Registry is intended to represent the usual or longest held job of the cancer patient. Smoking history for each patient is characterized according to status (i.e., never, former, or current use) and, among current smokers, according to the number of packs smoked per day.

The present analysis was based on subjects who had codable occupational information (i.e., either occupation or industry or both variables). The remaining subjects had no occupational information or noninformative job titles such as "retired." Duration of employment was only occasionally recorded and not used in these analyses. Of the initially eligible subjects, 34 percent of potential cases and 38 percent of potential controls were excluded due to missing occupational information.

The odds ratio (OR) was the measure of association between occupation and brain cancer. Gart's method[24] was used to calculate maximum likelihood estimates of the OR

TABLE 1—Distribution of Cancer Sites in Control Group, Missouri, 1984–88

Site of Cancer	Number	Percent
Lip, oral cavity, and pharynx	57	5
Digestive organs and peritoneum	276	22
Respiratory system and intrathoracic organs	356	29
Skin	21	2
Bones and connective tissue	24	2
Genitourinary organs	305	24
Leukemia	35	3
Lymphoma and multiple myeloma	63	5
Other sites	111	9
Total	1,248	100

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and corresponding 95 percent confidence intervals (CI). For each occupation and industry with two or more cases, age- and smoking-adjusted ORs were computed. The reference category consisted of all persons not in the occupation of interest.

Separate analyses were conducted for the astrocytic series (ICD-O morphology codes 93803, 93823, 94003-94413) and for "other" cell types of brain cancer. The astrocytic series was comprised primarily of unspecified astrocytomas (31 percent), unspecified glioblastomas (51 percent), and anaplastic astrocytomas (7 percent). "Other" cell types included oligodendrogliomas (31 percent), unspecified cell types (20 percent), and unspecified ependymomas (14 percent). All controls were used in each cell type analysis.

For analyses by smoking history, we excluded controls with cancers of smoking-related sites (lip, oral cavity, esophagus, larynx, lung, and bladder—ICD-O site codes 140-141, 143-145, 150, 161, 162, and 188).

Results

Risk of brain cancer according to industry of employment is shown in Table 2. Men employed in agricultural crop production had a moderately elevated risk (OR = 1.5; 95% CI = 1.0, 2.4). An OR of 2.8 (95% CI = 1.0, 8.3) was observed for workers in printing and publishing. Several other industries were associated with risks of 2.0 or greater, although estimates were based on small numbers. These included men employed in miscellaneous repair services (OR = 2.6; 95% CI = 0.5, 13.0), engineering services (OR = 2.1; 95% CI = 0.4, 10.3), and justice, public order, and safety (OR = 2.1; 95% CI = 0.9, 4.8).

As shown in Table 3, excess risks were found for social science professionals (OR = 6.1; 95% CI = 1.5, 26.1) and for police and fire protection services (OR = 2.2; 95% CI = 1.0, 4.7). The excess among police and fire protection services included an OR of 2.9 (95% CI = 0.8, 11.0) for police and detectives and an OR of 2.0 (95% CI = 0.4, 9.6) for firefighters. An OR of 2.5 (95% CI = 0.5, 11.5) was observed for brickmasons and tilesetters, although there were only three cases in that job category.

Evaluation of risks according to histologic type of brain cancer showed that the elevated risk in agricultural workers was confined to "other" cell types (OR = 5.3; 95% CI = 1.9, 14.3). The elevated overall risk for workers in printing and publishing was equally distributed between astrocytic and "other" cell types. Elevated risks of astrocytomas were observed for automobile repair work (OR = 1.9; 95% CI = 1.0, 4.1) and justice, public order, and safety (OR = 2.4; 95% CI = 1.0, 5.6).

Social science professionals showed elevated risks for both astrocytomas (OR = 4.7; 95% CI = 1.0, 22.9) and "other" cell types (OR = 19.0; 95% CI = 2.1, 145.6). The

TABLE 2—Age- and Smoking-Adjusted Odds Ratios* (OR) and 95% Confidence Intervals (CI) for Brain Cancer by Industry, Missouri, 1984–87

US Census Codes	Industry	Number		OR (95% CI)
		Cases	Controls	
010	Agriculture—crop production	22	62	1.5 (1.0,2.4)
060	Construction	26	135	0.8 (0.5,1.3)
100–122	Food manufacturing	6	36	0.7 (0.3,1.7)
171–177	Printing and publishing	7	10	2.8 (1.0,8.3)
180–192	Chemical manufacturing	4	14	1.1 (0.3,3.6)
270–301	Metal manufacturing	7	22	1.3 (0.5,3.2)
351–370	Transportation equipment manufacturing	20	83	0.9 (0.6,1.6)
391–392	Miscellaneous and unspecified manufacturing	9	46	0.8 (0.4,1.7)
400–432	Transportation	31	121	1.1 (0.7,1.7)
440–442	Communications	5	15	1.4 (0.5,4.1)
460–472	Utilities and sanitary services	3	26	0.5 (0.1,1.7)
591	Department stores	4	12	1.3 (0.4,4.5)
601	Grocery stores	5	12	1.6 (0.5,5.1)
641	Eating and drinking places	2	6	1.6 (0.2,8.9)
700–712	Finance, insurance, real estate	10	38	1.0 (0.5,2.2)
751	Automotive repair shops	11	28	1.7 (0.8,3.7)
760	Miscellaneous repair services	3	5	2.6 (0.5,13.0)
842, 850	Schools	13	37	1.3 (0.6,2.5)
880	Religious organizations	3	9	1.0 (0.2,4.1)
882	Engineering services	3	5	2.1 (0.4,10.3)
910	Justice, public order, safety	10	19	2.1 (0.9,4.8)
999	Industry not reported	21	93	0.9 (0.5,1.5)

*Odds ratios are reported for job categories that contained two or more cases.

TABLE 3—Age- and Smoking-Adjusted Odds Ratios* (OR) and 95% Confidence Intervals (CI) for Brain Cancer by Occupation, Missouri, 1984–87

US Census Codes	Occupation	Number		OR (95% CI)
		Cases	Controls	
003–037	Managers, administrators, executives	34	146	0.9 (0.6,1.3)
043, 063–083	Science professionals	2	11	0.6 (0.1,3.2)
044–059	Engineers	7	20	1.2 (0.5,3.2)
084–089	Physicians and dentists	3	13	0.8 (0.2,3.2)
113–159	Teachers	6	26	0.8 (0.3,2.1)
163–175, 183–184, 195–197	Social science professionals	6	4	6.1 (1.5,26.1)
176–177	Clergy	2	9	0.7 (0.1,3.3)
178–179	Lawyers, judges	3	10	1.1 (0.2,4.5)
185–194, 198–199	Artists, entertainers, athletes	3	9	1.2 (0.3,5.1)
213–235	Technicians, except health	11	27	1.6 (0.8,3.5)
243–285	Sales	25	90	1.1 (0.7,1.8)
303–389	Office supervisors and workers	16	43	1.4 (0.7,2.6)
413–427	Police, fire protection services	12	22	2.2 (1.0,4.7)
448–454	Building services	6	28	0.9 (0.3,2.3)
473–489	Farming	21	80	1.1 (0.6,1.7)
503–549, 864	Mechanics and repairers	22	68	1.4 (0.8,2.4)
553, 563–565	Brickmasons and tilesetters	3	6	2.5 (0.5,11.5)
554, 567–569	Carpenters	6	43	0.6 (0.2,1.5)
557, 585–587	Plumbers and pipefitters	3	12	1.2 (0.3,4.5)
558, 566, 588–589, 599, 863, 865, 866, 869	Construction, not elsewhere classified	11	36	1.3 (0.6,2.7)
594, 843–859	Pavers, surfacers, material movers	4	14	1.2 (0.3,3.9)
633–639, 644–655, 689–693, 796–799, 873	Precision production/tool and die makers, machinists, sheet metal workers	12	43	1.2 (0.6,2.4)
686–688	Food production	3	8	1.6 (0.3,6.8)
753–779	Machine operators, miscellaneous	12	72	0.7 (0.4,1.4)
785	Assemblers	5	15	1.4 (0.4,4.2)
803–814	Motor vehicle operators	22	83	1.2 (0.7,2.0)
889	Laborers, except construction	7	46	0.6 (0.2,1.4)
905–999	Unemployed/last job armed forces/unknown	24	119	0.8 (0.5,1.4)

*Odds ratios are reported for job categories that contained two or more cases.

excess risk among police and fire protection workers was confined to the astrocytic cell series (OR = 2.3; 95% CI = 1.0, 5.1). Farmers had an increased risk for "other" cell types (OR = 3.7; 95% CI = 1.4, 9.8). Machinists had an OR of 1.8 (95% CI = 0.6, 5.2) for astrocytomas.

There were 33.7 percent current smokers and 19.9 percent ex-smokers among cases, compared with 34.4 percent current smokers and 18.5 percent ex-smokers among controls. Current smokers had an OR of 0.9 (95% CI = 0.7, 1.3) and ex-smokers had an OR of 1.0 (95% CI = 0.7, 1.5).

Discussion

The present study confirmed previously reported occupational associations[2,3,25] between brain cancer risk and employment in certain white collar occupations. These increased risks may be related in part to better access and utilization of medical services and, hence, a greater likelihood of brain cancer diagnosis.[26]

An increased risk of brain cancer among agricultural workers has been reported in a number of recent studies,[10–13,27] although several others have failed to identify such a relationship.[21,28,29] In the current study, the elevated risk among farmers was confined to "other" cell types. Mussico, *et al*,[30] recently found an increased risk of brain gliomas in relation to use of insecticides or fungicides. Animal studies[31] and elevated levels of organochlorine compounds in adipose tissue of glioblastoma patients[32] adds biological plausibility to the hypothesis that agricultural chemical exposure may be partially responsible for the excess risk of brain cancer in farmers.

The increased risk seen in this study among workers in the printing and publishing industry was unexpected, although one previous study[33] suggested an elevated brain cancer risk in commercial pressmen. Since printers are exposed to a variety of chemicals, this relationship deserves further scrutiny.

Several *a priori* suspected high-risk occupations showed indications of elevated risks in our study; however, some of these comparisons were limited by small numbers. For example, we found slight elevations in risk for chemical manufacturers, mechanics and repairers, and precision metal workers such as machinists, all of which have been reportedly previously.[1,25,29,34] We identified an elevated risk of astrocytomas for automobile mechanics, who are potentially exposed to solvents, lubricating oils, and hydrocarbons.[25] Rushton, *et al*,[35] found elevated brain cancer mortality among bus mechanics in the United Kingdom. Our data suggested an increase in risk of astrocytomas for brickmasons and tilesetters. This association was also reported in a death certificate study from Massachusetts.[34]

We found no indication of an increased risk among smokers. This contrasts with a recent study[21] but is consistent with two earlier studies.[36,37]

The current study has several limitations. We relied on occupational information collected in conjunction with cancer incidence reporting. Of the initially eligible study subjects, over one-third had missing occupational information. The Registry obtains job information based on the hospital medical record at the time of diagnosis and some degree of misclassification is likely. A recent study that compared risk factor information contained in the Missouri Cancer Registry record with information obtained by interview found an agreement of 70 percent between Registry-reported occupation and usual occupation reported by interview.[38] In addition, occupational misclassification was apparently random and would therefore bias relative risk estimates toward unity.[39]

We selected controls from other cancer patients recorded on the Registry, with the attendant advantages and disadvantages.[40,41] We compensated for the relation between smoking and other cancers in sub-selection of controls and adjustment in the analyses.

The strengths of our study included greater accuracy in hospital-reported tumor diagnosis, compared with death certificates,[21] and the ability to analyze by histologic type.

This exploratory study suggests leads for future investigations with more information on occupational exposures and other possible confounders.

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Work-Related Psychosocial Stress and Risk of Preterm, Low Birthweight Delivery

CHARLES J. HOMER, MD, MPH, SHERMAN A. JAMES, PHD, AND EARL SIEGEL, MD, MPH

Abstract: We investigated whether work-related psychologic stress—defined as work characterized by both high psychologic demands and limited control over the response to these demands—increases a woman's risk of delivering a preterm, low birthweight infant.

We studied 786 employed pregnant women included in the National Longitudinal Survey of Labor Market Experience, Youth Cohort (NLSY), a nationally representative sample of 12,686 young adults. Data concerning work status, job title, and other factors affecting pregnancy outcome were obtained from the NLSY. Assessment of job experience was based on job title, using an established catalogue of occupation characteristics.

After accounting for the physical exertion entailed in a job, occupational psychologic stress as measured by job title was not associated with preterm, low birthweight delivery for the sample as a whole (Relative risk = 1.16, 95% confidence interval .45, 2.95). For those women who did not want to remain in the work force, work-related stress increased their risk of experiencing this outcome (RR = 8.1, 95% CI 1.5, 50.2). Personal motivation toward work, as well as the physical effort of work, should be considered in evaluating the impact of a job's psychologic characteristics on pregnancy outcome. (*Am J Public Health* 1990; 80:173–177.)

Introduction

A greater proportion of pregnant American women now work outside the home for pay than ever before.[1] Whether certain types of work adversely influences the outcome of pregnancy therefore has substantial import for maternal and child health.

Although a number of studies have examined the broader question of the influence of life stress on pregnancy outcome,[2] relatively little research exists in the specific area of work-related stress.[3] Fox, *et al*, compared low birthweight and perinatal mortality rates among active duty military women with a control group matched for race and parity. He found both outcomes significantly more frequent among the active duty group.[4] Mamelle, *et al*, also investigated the combined effects of psychological and physical stressors. She found that women with high levels of occupational fatigue—defined as work with a combination of prolonged standing, excessive routine, exposure to vibrating machinery, exposure to cold, heat or chemicals, and lifting—experienced higher risk of preterm delivery.[5] These studies both exhibit major limitations, including inadequate control for confounding factors and, in Mamelle's study, the possibility of recall bias.[6]

We evaluate here the impact of job related psychologic stress on pregnancy outcome for a national sample of *young* mothers.

Methods

Study Design, Population

We included in this study those women in the ongoing National Longitudinal Survey of Labor Market Experience, Youth Cohort (NLSY) who met study criteria.[10] The NLSY consists of a national probability sample of 12,686 youth who ranged in age from 14–21 years when selected in 1979. The survey intentionally oversampled Blacks, Hispanics, and economically disadvantaged Whites. The use of NLSY sample weights in analyses corrects for this oversampling and allows for generalization to the entire US population for this age group. All analyses presented here incorporate sample weights.

Criteria for inclusion in the study population included: female sex, having had a singleton live birth prior to the 1983 interview, and having worked during the most recent pregnancy. Additional technical criteria necessary for including an NLSY respondent in the analyses were availability of a US Census Bureau occupational and industry code for the job held during pregnancy, of job characteristic information for that job code, and of birth outcome data—birthweight and gestation—for the particular pregnancy (see below). Of the initial 12,686 NLSY respondents, 772 women met all criteria (see Appendix).

Pregnancy Outcome and Risk Factors

The survey respondents provided detailed information concerning family background, social, and educational factors, as well as a thorough job history, at the initial interview in 1979. Since that time, the survey youth have answered an extensive in-person questionnaire annually. Women respondents first gave information concerning their reproductive experiences in 1982, retrospectively describing pregnancy experiences in all previous years. The 1983 interview updated this information and included the weight and number of weeks early or late for the most recent live birth. Only the most recent pregnancy was used for these analyses. The NLSY sample diminished less than 5 percent from 1979 through 1983, with little variation among race or sex groupings.[11]

Using the interview data available from 1979 through 1983, we ascertained information concerning many of the factors known or suspected to influence risk of preterm or low birthweight delivery.[2] Specifically, we abstracted information concerning maternal race (Black, Hispanic, other), maternal age at the time of the infant's birth, maternal federal poverty status during the year of infant birth, maximum grade completed by the mother prior to the infant's birth, grade completed by the infant's maternal grandmother as of 1979, maternal marital status at the time of the infant's birth, smoking during pregnancy, alcohol use during pregnancy,

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month began prenatal care, height, prepregnancy weight, weight gain during pregnancy, prior induced abortions, prior total pregnancy failures, parity, and prior low birthweight delivery. Fertility and obstetric data were obtained from the 1982 or 1983 interviews; sociodemographic data were obtained in most cases from the interview most closely following the infant's birth.

Job Characteristics Data

The yearly NLSY interview obtains a detailed job history, including stop and start dates for all jobs. The NLSY staff provide a US Census Bureau occupation and industry code for each job worked for 20 hours per week for longer than nine weeks. Through the use of this history, the child's birth date, and the estimated gestation, we identified those jobs held during pregnancy. Job codes were available for some women working at a lesser level of intensity during pregnancy, provided that the woman worked over 20 hours for nine weeks in the same job prior to the pregnancy. We also derived a measure of the intensity of work during pregnancy, which encompassed both the proportion of pregnancy worked and the hours worked per week.

We used the Job Characteristics Scoring System developed by Robert Karasek to determine job control, job demands, and work-related physical exertion.[12] This system is based on analyses of the pooled responses to the three Quality of Employment Surveys of 1969, 1972, 1977 by a large, representative national samples of workers.[13] Karasek and colleagues derived several scales by factor analysis of these survey responses. Scales were developed separately for men and women, and apply to most US Census Code Occupational/Industry codes.

High values for job control reflect both the skill and creativity required to perform a job (skill discretion), as well as the freedom to make decisions about the work (decision authority). The measure is both reliable and valid.[12]

The psychologic workload measure reflects a job's pace and pressure (e.g., job requires working very hard). Although somewhat less reliable, this measure maintains face validity.[12]

The physical exertion measure is based on one question asked in two of the three Quality of Employment Surveys, specifically, "Does your job require lots of physical effort?" The measure has both face and concurrent validity.[12,*]

Attitudes Toward Working

All respondents were asked in 1979 and 1982 about their aspirations at age 35 ("What would you like to be doing at age 35?": same job, different job, or having a family). If a woman responded "Having a family," she was then asked "would you like to work in addition to having a family?" For these analyses, we classified a woman as uncommitted toward paid work if she responded in the 1979 survey that she did not want to work at age 35 in addition to having a family.

Analysis

We defined each demand/control quadrant by dichotomizing demand and control at the median level of each among NLSY working pregnant women. We considered three related outcome measures:

- preterm, low birthweight, defined as gestation less than 38 weeks and birth weight 2500 grams or under (5.5 pounds);
- low birthweight itself, i.e., birthweight 2500 grams or under;
- birthweight viewed as a continuous value.

We compared the frequency of preterm, low birthweight delivery, frequency of low birthweight, and mean birthweight between women in job characterized by both high demand and low control (n = 262) with women in jobs with all other combinations of demand and control (n = 510).

We dichotomized the physical exertion measure into high and low exertion groups at the median value among NLSY working pregnant women for those analyses incorporating this measure.

We undertook weighted multiple logistic regression to determine the effect of job-related stress on risk of delivering a preterm/low birthweight or low birthweight infant.[14] In these regressions, we adjusted for the potentially confounding influence of: maternal race (Black, Hispanic, other), maternal age, maternal federal poverty status during the year of infant birth, maximum grade completed by infant's mother prior to birth, education of maternal grandmother of 1979, marital status at time of birth, smoking during pregnancy, alcohol use during pregnancy, month began prenatal care, prepregnancy weight, prior induced abortions, prior total pregnancy failures, parity, and prior low birth weight delivery, as well as work-related physical exertion and our measure of work intensity. Similar weighted multiple linear regression analyses were used to determine the effect of exertion on birthweight.

We explored whether the relationship between stressful work and preterm low birthweight varied among those more or less motivated towards work through stratification and through specification of interaction terms (stress × motivation) in the logistic and linear models.

We weighted all analyses with NLSY Sample Weights, which reflect the probability of selection relative to the US population for this age group, and also reflect non-response in any given year.[15] Design effect factors in the NLSY are generally small (less than 2), and are not included in the analyses presented here.**

With the sample size of 780, we could detect a relative risk associated with job stress on the likelihood of delivering a preterm, low birthweight infant of 1.8 with a power of 90 percent.

Results

Sample Attrition and Missing Data

Among women who worked during pregnancy, several differences existed between those women included in the final sample and those excluded due to missing information (either no Census job code or job characteristic score) (Table 1). Most of the exclusions were due to the absence of a job code, in turn due to these women having worked for limited hours. Excluded women were as a group socioeconomically disad-

*Homer CJ, Beresford SAA, James SA, Siegel E, Wilcox S: Work-related physical exertion and risk of preterm, low birthweight delivery. Paediatric and Perinatal Epidemiology (in press).

**Unpublished technical documentation, Center for Human Resources Research, Ohio State University.

TABLE 1—Comparison of Study Women and Excluded Working Women

Measure	Worked, No Census Code	Worked, Coded, No JCSS Score	Final Sample	All NLSY Mothers
	n = 207	n = 54	n = 786	n = 2375
Maternal				
% Black	16.7	27.3*	15.7	21.9
% Hispanic	10.7	5.7	7.3	8.9
Height (in)	64.0	64.0	64.1	63.9
Weight (lbs)	128.6	122.0	126.9	127.0
Weight gain (lbs)	31.7	29.7	31.6	31.0
Education (years)	11.0*	12.0	11.9	11.2
Prior pregnancy failures (mean)	.15	.09	.15	.13
% Prior LBW	3.3	3.9	2.4	4.0
% Less than federal poverty level	36.9*	36.4	15.9	29.0
% First trimester prenatal care	74.8*	82.0	84.5	80.0
% Smoked	50.3*	32.2	40.1	43.0
Infant				
Birthweight (grams)	3245	3145	3295	3304
% LBW	7.1	5.1	6.25	8.3
% Preterm/LBW	4.3	1.7	3.4	4.7

*Excluded group differs from final sample at $p < .05$, by chi-square or T-test

vantaged relative to other working women, and were more similar to the population of non-working women.

Validation of Measures

The mean birthweight of the infants in the NLSY was 3304 grams. Preterm infants were smaller than term infants, with a mean weight of 2697 grams versus 3389 grams for term infants. Of the 2375 infants included in this study, 8.3 percent weighed less than 2500 grams. Just under 5 percent (4.7 percent) of the NLSY infants were both low birthweight and preterm. NLSY data consistently confirmed those factors well known to influence pregnancy outcome.***

Prominent within the high stress group are jobs of stitchers and sewers in the apparel industry, waiters and food counter workers, nursing aides and orderlies, and cashiers, especially in grocery stores. Low demand, high control work included accountants, mail order salesmen, and bookkeepers. Low demand, low control workers included typists, a variety of cleaning personnel, and household child care workers. High demand, high control occupations included nurses, receptionists, and restaurant managers.†

Impact of Job Characteristics

Women working during pregnancy in jobs characterized by high psychologic demands and low job control were two times as likely to deliver a low birthweight, preterm infant as women working in low exertion jobs (5.1 percent vs 2.6 percent). Similarly, the frequency of low birthweight infants was 1.67 times higher among high stress workers and the mean birthweight of their infants was lower by 78 grams.

Women in high stress jobs were less well educated, had lower prepregnancy weight, and higher smoking frequency***; moreover, almost three-fourths of them in psychologically stressful jobs were also in jobs with high amounts of physical exertion, and physical exertion itself was strongly associated with increased frequency of preterm, low birthweight as well as with the other outcomes (Table 2).

After taking into account the potential confounding effect of job related exertion and other maternal characteristics, we found that the association of job related psychologic stress with preterm, low birthweight, or with any of the other pregnancy outcomes for the sample of women viewed as a whole was no longer important (Table 3).

For those 70 women who stated they did not want to working outside the home after age 35, however, stressful work significantly worsened pregnancy outcomes.

On average their birthweights were almost 500 grams less than infants born to women in less stressful occupations. These differences remained significant when exertion and other maternal factors were considered (Table 4).

Discussion

We found in this study that young women working in jobs characterized by high levels of psychologic demand with little control over the pace and style of response to those demands were somewhat more likely to deliver a preterm, low birthweight infant than women in other jobs, if they were not motivated to continue working.

We considered practically all those factors outside the work experience that are thought to influence pregnancy outcome, and by using an occupational title-based system of job characteristic description, we avoided the possibility of recall bias in determining work stress. The construct of stressful work as being comprised of work with high demands and low control is intellectually appealing; moreover, it, together with the specific instrument used in this study, has been supported in the field of cardiovascular research.[9,16,17]

Despite these strengths, this study does present potential drawbacks in a number of areas. These areas include the nature of the study population, the characteristics of the measures of occupational stress and motivation, and the source of outcome information.

The population used for this study is not representative of all American mothers, but rather represents only those women bearing children during the earlier portion of their potential reproductive years. Such a sample necessarily includes more socioeconomically disadvantaged, unmarried, and minority women than a sample drawn from the entire maternal population. Compared with the women included in the National Family Growth Survey, NLSY mothers are more likely to be Black (21.9 vs 14.1 percent), to have dropped out of high school (29 vs 24.5 percent), and to be unmarried (22.1 vs 17.2 percent).[18] The youthfulness of the sample likely accounts for the somewhat increased preva-

TABLE 2—Pregnancy Outcomes for Women in Jobs of Differing Levels of Exertion (by quartile)*

Job Exertion Level	N	%Preterm/ LBW	%LBW	Birthweight (mean in grams)
Quartile 4 (High Exertion)	n = 196	5.07	9.05	3281
Quartile 3	n = 180	7.01	8.83	3232
Quartile 2	n = 232	1.25	4.35	3423
Quartile 1 (Low Exertion)	n = 164	1.03	3.04	3440

*Results not adjusted for differences between workers at different levels of exertion. Singleton births only.

***Data available on request to the author

†A more complete listing of jobs is available on request to the author

TABLE 3—Impact of Stressful Work on Pregnancy Outcomes*

Outcome	Crude Estimate of Effect	Adjusted Estimate of Effect	95% Confidence Interval
Preterm/LBW	Relative Risk = 2.0	Relative Risk = 1.3	.6,3.1
LBW	Relative Risk = 1.7	Relative Risk = 1.4	.75,6.8
Birthweight	Difference = −78 grams	Difference = .1 grams	−81,+81 grams

*Singleton births only; adjusted estimates based on regression models without interaction.

TABLE 4—Impact of Stressful Work on Pregnancy Outcomes, Accounting for Maternal Motivation to Work

Outcome	Crude Frequency of outcome	Adjusted Estimate of Effect (95% CI)
Preterm/Low Birthweight		
Does not aspire to work outside home (n = 70)	Low Stress 0% High Stress 8.3%	Relative Risk = 8.4 (1.4,50.2)
Does aspire to work outside home (n = 716)	Low Stress 2.8% High Stress 3.85%	Relative Risk = .64 (.23,1.7)
Low Birthweight		
Does not aspire to work outside home	Low Stress 0% High Stress 18.7%	Relative Risk = 8.0 (1.3,37)
Does aspire to work outside home	Low Stress 5.7% High Stress 6.3%	Relative Risk = .86 (.39,1.89)
Birthweight		
Does not aspire to work outside home	Low Stress 3561 grams High Stress 3062 grams	mean effect = −295 grams
Does aspire to work outside home	Low Stress 3300 grams High Stress 3262 grams	mean effect = +38 grams

lence of low birthweight relative to concurrent national figures of 6.6–7.6 percent.[2] These demographic characteristics may also explain the decreased frequency of maternal work during pregnancy (66 percent of married primigravidas in the sample worked during pregnancy, compared with contemporary national figures of 85 percent).[1] Nonetheless, while not reflecting the entire age spectrum, the NLSY's emphasis on this younger child-bearing population is important in its own right. Almost half (47 percent) of all births occur to women less than 25 years old.[18]

Concerning the measure of occupational stress, problems may exist relating to both the precision of the measure and its validity among this young population. The use of an occupational title-based system leaves open the possibility that the job experience of the particular women in this study may not be accurately reflected in the job characteristics measure. The indicator of job demand is particularly suspect on this score. Although these measures are unlikely to be biased in any particular direction, use of any imprecise system makes identification of an association difficult.[19]

Moreover, the construct of high demand/low control work as constituting work-related psychologic stress may not fully encompass factors resulting in work stress for young women at early stages of their work careers. Other factors, such as salary, shift worked, flexibility in hours, or availability of maternity and sick leave, may play an equally important role for this population.

The measure of maternal motivation toward work used in this study is not intended to validly predict a woman's employment status at age 35; rather, this measure is used to indicate the woman's current attachment to working. This measure, unfortunately, is not stable even over short periods. Although 9 percent of women in 1979 and 10 percent in 1982 were classified as not intending to remain in paid work, the correlation between these two groups is low (r = .15, kappa = .15).

Finally, all of the outcome measures in this study are based on maternal recall. Although this defect is not found in other studies relating to work and pregnancy outcome, we found the maternal responses very plausible. Mean White and Black birthweights in the NLSY sample, for example, are extremely close to the estimates from the National Natality Survey (3374 grams and 3095 grams for White and Black infants, respectively, in the NLSY, compared with 3391 grams and 3099 grams for White and Black infants in the NNS).[20] Moreover, these data support most of the well known associations with birthweight, low birthweights, and prematurity, such as race, smoking, maternal weight gain, and prior pregnancy experience.

Assuming that our findings are not simply accounted for by these methodologic limitations, why should the impact of high demand/low control work be so strong among women not intending to remain in the paid work force? We can speculate that such women may feel compelled to work, resent it, and that a demanding and restrictive job provides the "last straw." Conversely the over arching desire to remain a part of the gainfully employed population may limit the influence of high demand/low control work on the majority of women who are motivated to remain workers.

Maternal work during pregnancy is likely to be a balance between helpful aspects, such as financial remuneration and social contact, and harmful components. This study sought to determine whether work-related psychosocial stress is one of these harmful aspects. We found that for those relatively few women not desiring to remain in the paid work force, jobs characterized by high demand/low control were independently associated with adverse pregnancy outcome. This finding, based on small numbers, must be considered preliminary pending confirmation in larger studies.

For the remainder of working women, high demand/low control work did not seem to influence the outcomes examined. Due to the limitations in the measures used, however, this study does not preclude an important role for work-related psychologic factors in influencing the outcome of pregnancy for these women. Further research is necessary to clarify which aspects of work are stressful for young women, how these interact with the woman's motivation to work, and how these factors together may influence pregnancy outcome.

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APPENDIX

Derivation of Study Sample from National Longitudinal Survey of Youth

12,686	youth in initial sample	Excluded from study:	
	↓	6,398	Men
6,288	Women		
	↓	3,913	Childless as of 1983 interview
2,375	Delivered a child before 1983 interview		
	↓	1,328	Did not work during most recent pregnancy
1,047	Worked during most recent pregnancy		
	↓	207	Working pregnant women did not have job code assigned
840	Working pregnant women had jobs assigned census code		
	↓	54	Women with job codes not matched to Job Characteristics Scoring System
786	Women with job codes matched to Job Characteristics Scoring System		
	↓	14	Women not reporting precise birthweight*
772	Women with job data recalling precise birthweight of most recent birth		

*12 of these women do report whether their infant weighed more or less than 5.5 pounds.

Qualitative Health Research Conference: Call for Abstracts

An international, interdisciplinary conference is being planned for next year to explore issues and developments in qualitative methods and to examine the latest qualitative health research. The conference will be held February 22–23, 1991 at West Edmonton Mall, Edmonton, Alberta, Canada.

Interested researchers are invited to submit abstracts, not to exceed 150 words, for consideration. Send three copies of the abstract—including author(s) name(s), address, institutional affiliation, telephone and fax numbers—by April 19, 1990 to: Janice M. Morse, RN, PhD, Professor and National Health Research Scholar, Faculty of Nursing, CSB 3-120, University of Alberta, Edmonton, Alberta, Canada T6G 2G3. Dr. Morse is also the contact for further information.

Effects of Workplace Smoking Bans on Cigarette Consumption

Ron Borland, PhD, Simon Chapman, PhD, Neville Owen, PhD, and David Hill, PhD

Abstract: A sample of staff working in the Australian Public Service (n = 2113) were surveyed two to four weeks before a mandated total ban on workplace smoking was introduced, and again five to six months later. Among the 391 smokers on whom complete data were available, the workplace smoking bans were associated with reduced rates of smoking, particularly among heavier smokers where the reduction in consumption was over 25 percent. (*Am J Public Health* 1990; 80:178–180.)

Introduction

The evidence for effects of workplace bans on smoking cessation is mixed[1–5]; the reduction in prevalence may be no greater than the spontaneous rate of smoking cessation. Three studies which investigated consumption in continuing smokers, however, found evidence of reductions.[1,3,5]

We examined the impact on smoking behavior of the introduction of a workplace smoking ban in the Australian Public Service, Australia's largest employer group, with over 170,000 employees. The introduction of the ban was announced over a year before its implementation deadline and was followed by publicity, pre-deadline phasing in of restrictions at some worksites, and availability of programs to help smokers stop smoking or to control their consumption. This paper reports evidence of changes in smoking behavior in a longitudinal sample of smokers from work settings with varying levels of restrictions on smoking prior to the mandated ban.

Method

A total of 2,113 employees of the Australian Public Service (from six Departments spread across three cities and 44 locations) was surveyed two to four weeks before the deadline for implementation of the ban, and again five to six months after its introduction. There were 4,215 respondents to the initial survey (a 79 percent return rate). However, 26.3 percent did not include their names on the initial questionnaire and another 11.5 percent had left the public service or were on extended leave, leaving 2,623 whom we attempted to recontact. A total of 2,169 matched surveys were returned (a return rate of 83 percent). However, 56 of these were not usable, leaving the final sample of 2,113.

Preliminary analyses revealed that those who completed both surveys were less likely at the pre-test to have been smokers (22.7 percent) compared to 27.6 percent for those who completed the initial survey only. There were slightly more males in the final sample (55 percent) compared to the initial sample (51 percent), but there was no difference in mean age or in overall daily cigarette consumption.

To explore the effects of the ban on workday cigarette consumption, only the 391 smokers who reported being smokers at both surveys were considered. Excluded from this group were 39 occasional smokers who reported in the initial survey that they did not smoke on workdays and five who did not provide consumption estimates.

The initial questionnaire, completed two to four weeks before the deadline for the introduction of the ban, dealt with smoking status, the extent of current restrictions in their workplace, and the manner in which any existing restrictions were brought about. Smokers were asked to estimate the number of cigarettes they usually smoked on both work and non-work days, and to recall the number of cigarettes they smoked in the previous 24 hours, divided into seven time periods (before work, while working indoors, while working outdoors, during coffee breaks, at lunch time, in the hour immediately after work, and in the rest of the evening). They were also asked about their attitudes to smoking and to smoking bans, but these data are reported elsewhere.[6] A similar questionnaire was administered at follow-up five to six months later.

Results

Of the matched sample of 2113, 492 (or 23.3 percent) were current smokers at the time of the initial survey and 471 (or 22.3 percent) were smoking at follow-up, a reduction of 21 smokers. Fifty-seven smokers at the time of the initial survey were not smoking at follow-up, and 36 previous non-smokers reported smoking. This 1 percent reduction in prevalence over the six month period is about the estimated yearly reduction in prevalence of smoking for Australians.[7] Of those who stopped, 58 percent had done so more than four months before completing the follow-up questionnaire, i.e., closely following the introduction of the ban.

To study the effects of pre-deadline restrictions on consumption, restrictions were divided into those where the person could smoke at their work station (43 percent of respondents), and those where they should not (57 percent). As can be seen from Table 1, the pre-deadline daily consumption difference between those who reported that smoking at their work station was not permitted and those where it was permitted was 4.5 cigarettes per day. Table 1 also shows that the mean reduction in smoking following the ban was 5.2 cigarettes where there had been no pre-deadline work station restrictions, and 1.9 cigarettes where there were such restrictions. This smaller reduction in the latter group should be seen in the light of their lower pre-deadline consumption.

To further explore the consumption reduction in respondents for whom there had been no restrictions prior to the mandated ban, levels of reported usual workday consumption before the ban were divided into three categories (light, moderate, and heavy) and the change from reported pre-ban workday consumption to post-ban consumption organized by usual pre-ban consumption (Figure 1). While light smokers did not change consumption, moderate smokers reduced by an average of 5.8 cigarettes per day (29.1 percent) and heavy smokers reduced by 7.9 cigarettes (26.6 percent).

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TABLE 1—Mean Daily Cigarette Consumption before and Six Months after the Workplace Smoking Ban was Introduced, According to Restrictions Previously Imposed

Time of Survey	Pre-ban Smoking Restrictions: Banned at Work Station (n = 221)	Pre-ban Smoking Restrictions: Not Banned at Work Station (n = 170)	Difference (between subjects effect)
Before ban	16.01	20.54	4.53 (2.45, 6.61)
After ban	14.16	15.36	1.20 (−0.55, 2.95)
Before-after difference (within subjects effect)	1.85 (1.00, 2.70)	5.18 (3.94, 6.42)	

NB: 95% confidence intervals in parentheses.

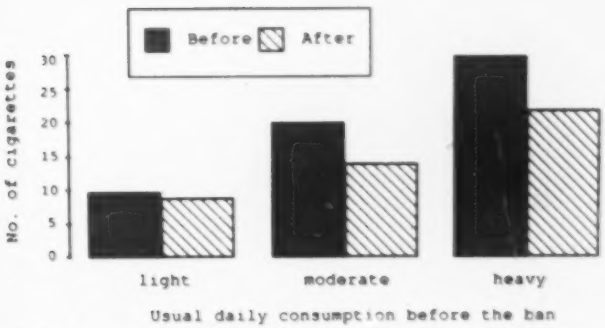

FIGURE 1—Mean Cigarettes Smoked per Day before and after the Ban among Previously Light, Moderate and Heavy Smokers
Light = 1–14 cigarettes/day
Moderate = 15–24 cigarettes/day
Heavy = 25 or more cigarettes/day

As can be seen from Table 2, smokers reduced their average cigarette consumption while working inside by 7.0 cigarettes per day. This was partly compensated for by average increases of 0.7 of a cigarette smoked outside during working hours, and of less than half a cigarette both before work and in the hour after work. The pattern of change was generally consistent for the three smoker categories, except that heavy smokers tended to smoke more outside while working, and the light smokers showed evidence of compensation in the "rest of the evening."

Discussion

Although some smokers may have used the ban's onset and/or the increased availability of smoking cessation resources as stimuli to stop, overall prevalence in the sample changed very little; it is not clear that this resulted in increased rates of smoking cessation compared with normal community rates. The introduction of the workplace smoking ban was associated with reduced levels of cigarette consumption, however. Small increases in rates of smoking outside of the work environment did not compensate for the enforced reduction. Comparison of smoking behavior under two prior levels of restriction on smoking corroborate the evidence that workplace smoking bans are associated with reduced overall levels of smoking, as do data, not reported here,* which showed similar drops in consumption among a small sample who completed 24-hour smoking diaries both before and after the ban.

Relatively little compensatory smoking occurred, and there is no evidence of greater compensation in heavier smokers, who are presumably the most addicted. The fact that smokers did not smoke more at coffee and lunch breaks suggests that the inconvenience of leaving the workplace to smoke was a sufficient obstacle to increased consumption. However, because we did not assess blood nicotine or cotinine levels directly, and did not ask for reports of puff topography, it is not possible for us to show that compensatory changes in puff topography did not occur. Smokers could have increased puff frequency, depth of inhalation, or amount smoked as compensatory mechanisms. However, the environmental restraints imposed by a workplace smoking ban do not favor compensation by changed smoking topography because they reduce opportunities to space cigarettes throughout the day, and compensatory smoking patterns

*Available on request to author.

TABLE 2—Mean Changes in Cigarette Consumption by Usual Smoking Level Reported in the Initial Survey for Those Who Could Smoke at the Work Stations at that Time, Broken Down by Time of Day

	Pre-testing smoking level: Light n = 48	Moderate n = 62	Heavy n = 60	Total n = 170
Before work	0.17 (−0.11, 0.45)	0.31 (0.07, 0.55)	0.80 (0.38, 1.22)	0.44 (0.25, 0.63)
Coffee breaks	−0.04 (−0.44, 0.36)	−0.16 (−0.60, 0.28)	0.07 (−0.42, 0.56)	0.05 (−0.20, 0.30)
Working inside	−2.83 (−3.60, −2.07)	−6.42 (−7.58, −5.26)	−10.97 (−12.79, −9.15)	−7.01 (−7.93, −6.09)
Working outside	0.46 (0.10, 0.82)	0.24 (−0.33, 0.81)	1.37 (0.50, 2.24)	0.70 (0.32, 1.08)
Lunch	0.38 (0.08, 0.67)	−0.13 (−0.37, 0.11)	0.13 (−0.33, 0.60)	0.11 (−0.09, 0.31)
Hour after work	0.46 (0.15, 0.77)	0.31 (0.01, 0.61)	0.55 (0.21, 0.89)	0.44 (0.62, 0.26)
Rest of evening	0.48 (−0.46, 1.42)	0.05 (−0.84, 0.93)	0.13 (−1.18, 1.44)	0.20 (−0.41, 0.81)
Total	0.94 (−2.51, 0.63)	−5.81 (−7.32, −4.30)	−7.9 (−10.62, −5.21)	−5.18 (−6.41, −3.95)

NB: 95% confidence intervals in parentheses.

would result in increased exposure, primarily in the evening when most cigarettes are already reported to be consumed. Nevertheless, if there were compensatory changes, then inferences about the benefits of the reduced consumption would be weakened.

Heavy smokers appear to benefit most from a workplace ban. As heavy smokers typically find smoking cessation most difficult,[8] workplace smoking bans may provide a means by which they can reduce their consumption to less dangerous levels. Although controlled smoking has not been shown to be a generally sustainable strategy under normal conditions,[9] the imposition of environmental restrictions may make long-term controlled smoking more viable in itself, and a useful way station for those who would eventually like to stop smoking completely.

To estimate the impact the ban may have on overall cigarette consumption, we extrapolated our results to the entire Australian Public Service. We estimate that 24.7 percent of 170,000 public servants or about 42,000 are smokers. Taking the estimate of reduced consumption of 5.2 cigarettes a day from the main sample in Table 1, and assuming 240 work days per year, the resultant reduction in cigarette consumption is about 52 million cigarettes. At the current cost of about 10 cents (Australian) for a cigarette, this represents a loss of $5.2 million a year in retail tobacco sales to staff of the Australian Public Service alone. The public health benefits of these restrictions seem clear, but it is also evident what is motivating the tobacco companies to oppose them.

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NICHD Funds Two Research Centers on Learning Disabilities

The National Institute of Child Health and Human Development (NICHD) has announced the establishment of the first two US research centers to combine the biomedical and behavioral sciences to focus specifically on learning disabilities. One of the new centers, headed by Dr. Bennett Shaywitz, will be at Yale University in New Haven, CT; the other, headed by Dr. Martha Bridge Denckla, will be located at Johns Hopkins University in Baltimore, MD. Support for the two centers totals approximately $1.5 million, with $550,000 going to Yale, and almost $1 million to Johns Hopkins.

The centers were established in response to the recommendations of the Interagency Committee on Learning Disabilities (ICLD), which was established by the Health Research Extension Act of 1985 to review proposals for research programs and facilities related to learning disabilities in children. The US Department of Education (1987) reports that 4.73 percent of all school-aged children (representing almost 1.9 million children) receive special education services for learning disabilities.

The Yale Center will include three projects: attention and conduct disorders in children and their interrelationships; attentional aspects of cognition; and a study of the distribution, types, and stability of reading, arithmetic and attention problems in a representative sample of children selected at age five and followed for two years.

The Johns Hopkins Center proposes a multidisciplinary program; through the convergence of biomedicine and education, investigators hope to discover how and why children develop learning disabilities. The program will include neurologists, psychiatrists, geneticists, statisticians, psychologists, special educators, and communication specialists.

The investigators plan to use molecular genetics technology and magnetic resonance imaging (MRI) to study the origins of learning disorders. The research will focus on the psychological processes related to brain development as well as the effects of educational intervention. To gain a better understanding of Attention-Deficit-Hyperactivity-Disorder, investigators will study the visual, spatial and motor deficits in families with a history of known genetic defects. Methodological approaches will include neuropsychological analysis and MRI correlates of intervention-resistant reading disabilities.

Precision and Accuracy of a Portable Blood Analyzer System during Cholesterol Screening

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Abstract: The precision and accuracy of two Kodak Ektachem DT-60 portable blood analyzers were assessed in a model (research) cholesterol screening program in Rochester, New York. Between June and October 1987, a total of 8,573 people underwent a cholesterol screening held in a movable trailer. A wide variety of temperature, humidity, and other potentially adverse conditions were encountered during the screening period. Between-run coefficients of variation ranged from 1.9 percent to 4.8 percent per month; average bias compared to a Reference Laboratory method ranged between +0.2 percent and +2.0 percent. Both precision and accuracy met currently recommended standards for cholesterol testing in the United States. (*Am J Public Health* 1990; 80:181–184.)

Introduction

In 1985, the National Institutes of Health initiated a National Cholesterol Education Program intended to inform health professionals and the general public about the significance of high blood cholesterol levels.[1] It is now recommended that cholesterol testing be performed on all adults in the United States.[2] Interest in blood cholesterol coincides with the recent availability of analytical systems that allow cholesterol concentration to be measured in a matter of minutes with small volumes of whole blood, serum, or plasma. Some of these systems are portable and could facilitate almost-immediate feedback to screening participants. The instruments appear to be highly acceptable to consumers because they avoid venipuncture in favor of fingerstick blood sampling.[3] While found to be reliable when evaluated in physicians' office or laboratory settings, there is little information concerning the portable blood analysis systems over extended time periods under field conditions.[4–6]

A report from the Laboratory Standardization Panel of the National Cholesterol Education Program[7] indicates that well-established clinical laboratories may lack acceptable accuracy in cholesterol measurement. Given the problems that have been noted with major laboratories in cholesterol testing, it is critically important to assess the reliability of the portable testing systems under true field conditions before they are widely recommended for general use in public screenings. We report such an assessment.

Methods

The Rochester, New York Model Cholesterol Screening Program

A model cholesterol screening program funded as a demonstration research project by the National Heart, Lung, and Blood Institute was conducted in the metropolitan Rochester, New York, area during a 13-week period from June to October 1987. Ten grocery stores served as the sites of the cholesterol screenings, and 8,573 screenees participated.

The Analytical System

The Kodak Ektachem DT-60 was evaluated for total cholesterol determinations. Cholesterol measurement on this system utilizes a dry, multilayered, self-contained analysis element coated on a clear polyester support ("slide"). Cholesterol esters are hydrolyzed to cholesterol, which is then oxidized to generate hydrogen peroxide. The hydrogen peroxide oxidizes a leuco dye in a peroxidase-catalyzed reaction to produce a colored compound. Colorimetric measurement by reflectance spectrophotometry provides the basis for the final cholesterol concentration value. A 10 microliter drop of plasma or serum is deposited on the slide by a single-tipped battery-driven pipette and is evenly distributed on the slide by a spreading layer. Once spotted with plasma or serum, the slide is manually advanced into the analyzer for further processing. Assay time is slightly more than five minutes; six samples may be loaded in the analyzer simultaneously, and maximum throughput is about 65 cholesterol slides per hour. Results appear both in a visual display and on a hard copy.

Two DT-60 Analyzers (designated here DT-60A and DT-60B) were purchased from the regular production line. The instruments were operated by a screening team of 20 people who were trained by supervisory personnel. None of the screeners had prior laboratory or chemistry experience.

Training and Prescreening Operation

Screening supervisors learned to operate the instruments during a standard 3 to 4-hour training period conducted by Kodak sales personnel. For each of 10 days prior to the actual screening program, two supervisors prepared the instruments for use and ran four sets of reference materials in pairs, along with actual patient-derived blood samples, twice each day. Thus, each day, each reference sample was run four times on each analyzer. These reference values were compared to target values provided by the manufacturer and were used to establish calibration of each DT-60. Precision data were assessed to determine readiness for actual field operation. The supervisors then trained the screening team, each member receiving 3 to 4 hours of practice and training prior to field operation of the instruments. In the field, the screening team was supervised closely, especially for the first 2 to 3 weeks to avoid technical errors.

Environmental Conditions

Screenings were held in an 8′ × 35′ construction trailer equipped with a room air conditioner. The trailer was not

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heated, and no attempt was made to standardize trailer temperature or humidity. The trailer was allowed to simulate the wide variety of temperature and moisture conditions that might be encountered in community screening programs. On one potential screening day in early October, when outside temperatures reached below 10°C, both DT-60 analyzers failed to reach operating temperature, and screenings had to be canceled. No other obvious malfunctions were noted due to environmental conditions in the construction trailer. The instruments were started and shut down anew each day.

Precision

Instruments were calibrated using Kodak supplies as described by the manufacturer for the DT-60. At the beginning of each day's operation, a series of three separate commercially prepared reference samples was run on both DT-60 Analyzers to assess continued evidence of calibration. Screenee samples were analyzed either on DT-60A or DT-60B, whichever was ready first. Samples were run in duplicate when the first determination for total cholesterol was <3.35 mmol/L or >7.25 mmol/L. Reference material values were "rejected" when a reference value was greater than two standard deviations from the target value, as determined during the prescreening period on replicates of reference materials. The "rejected" specimen was repeated and, if still out of range, the operations protocol specified discontinuation of analyses until the instrument was recalibrated. After analysis of 10 screening samples on a given instrument, a low-range reference sample was run. Following 10 more screening samples, an intermediate-range reference sample was run, and following an additional 10 screening samples, a high-range reference sample was run. Thus, after 30 screenee samples, a series of three reference samples was completed. This procedure was repeated throughout a day's run on both DT-60 instruments. Between-run precision only is reported here.

Accuracy

Accuracy of the DT-60 instruments was assessed using patient samples (venous serum) obtained from hospitalized patients. Fresh samples were split and run on DT-60A, DT-60B, and by the Abell-Kendall Manual Reference Method in a CDC-standardized laboratory. Paired runs were performed three times during the screening program, approximately six weeks apart. Sixty-eight samples were tested on all three instruments/methods. Samples were run on the DT-60s by usual screening team members at the end of the day's screening work.

Results

The precision data during the 10 days of practice (or training) prior to actual screening in the field are shown in Table 1. While coefficients of variation (CV) for the entire 10-day period, for each instrument (DT-60A and DT-60B), were overall quite acceptable, especially in the clinically important values above 5.15 mmol/L, accuracy of the instruments was less acceptable. For example, the CV for the reference sample target of 7.00 mmol/L was 2.22 percent for DT-60A and 2.59 percent for DT-60B over the 10 days. However, mean values on both instruments fell below target values throughout the entire range of reference samples studied. Thus, we considered the instrument/operator team to be acceptable prior to screening in the field in terms of precision, but both instruments were felt to require recalibration before actual field use to correct for the low bias we observed on reference materials during the 10-day prescreening trial runs.

Precision for DT-60A, on a monthly basis during the actual screening program, is reported in Table 2. Coefficients of variation never exceeded 4.8 percent and were typically 3 percent or less. Data for DT-60B were similar and therefore are not repeated. It should be noted that CV values did not substantially improve with either instrument as the experience of the screeners increased.

The results of linear regression analysis comparing each DT-60 separately against the Abell-Kendall Manual Reference Method data are reported in Table 3. As indicated, each DT-60 correlated highly with the Abell-Kendall values ($r = 0.99$ for each), and both showed a slightly negative proportional bias (linear slope <1.0) but a positive y intercept. Table 4 reports the mean and average bias of each DT-60 compared with the Abell-Kendall Reference results for all patient samples run (total cholesterol by reference method ranges from 2.75 mmol/L to 9.55 mmol/L). Figure 1 is a Bias Plot comparing the DT-60 instruments with the matching Abell-Kendall Reference Results. The plot provides visual confirmation of the bias data reported in Table 4.

Discussion

The major criteria by which to assess reliability of cholesterol testing are precision and accuracy.

Precision is defined as the ability of an analytical system to obtain comparable values on repeated testing, typically

TABLE 1—Precision during 10 Days of Pre-Screening/Training Period

Instrument	Number of Observations	Target Cholesterol Value of Reference Material (mmol/L)	Minimum Cholesterol Value Observed (mmol/L)	Maximum Cholesterol Value Observed (mmol/L)	Mean of Cholesterol Observations (mmol/L)	Coefficient Vairation (CV) (%)
DT-60A	40	2.60	2.25	2.70	2.50	3.87
	40	3.15	2.55	3.15	3.00	4.23
	40	7.00	6.45	7.10	6.90	2.22
	40	7.60	6.70	7.60	7.15	2.59
DT-60B	40	2.60	2.05	2.75	2.55	5.50
	40	3.15	2.80	3.25	3.05	3.13
	40	7.00	6.40	7.20	6.90	2.59
	40	7.60	6.80	7.55	7.20	2.33

To convert mmol/L to mg/dl cholesterol, divide by 0.02586

TABLE 2—Precision in a Field Study of Model Cholesterol Screening (DT-60A)

Month	QC Pools Target Value (mmol/L)	Mean Value (mmol/L)	DT-60A (n)	DT-60A CV (%)
#1	2.65	2.65	69	2.8
	6.00	6.05	67	2.6
	7.25	7.30	65	4.8
#2	2.70	2.70	100	3.0
	6.30	6.15	82	2.7
	7.70	7.50	79	3.4
#3	2.70	2.70	88	2.8
	6.30	6.25	81	2.8
	7.70	7.60	72	1.9
#4	2.70	2.70	92	3.3
	6.30	6.35	86	2.7
	7.70	7.65	76	2.3

TABLE 3—Linear Regression Equations for DT-60 Results Compared to Abell-Kendall Reference Method Cholesterol Results

Analytical Instrument	Sample Size (n)	Slope	Y-Intercept	r
DT-60A	68	0.952	14.44	0.99
DT-60B	68	0.946	13.20	0.99

TABLE 4—Mean and Average Bias of Instruments versus Abell-Kendall Method for Cholesterol

Analytical Instrument	Sample Size (n)	Mean (mmol/L)	Average Bias Compared to Abell-Kendall Reference Method**
Abell-Kendall* (Reference Method)	68	6.10	—
DT-60A	68	6.20	+1.3%
DT-60B	68	6.10	+0.2%

*range of values 2.75 mmol/L to 9.55 mmol/L
**Biases calculated using cholesterol values originally determined in mg/dl.

expressed by the *coefficient of variation* (CV), defined as one standard deviation divided by the mean of repeated determinations × 100. The *total* (or between-run) coefficient of variation is a measure of a method's variability over many days, whereas *within-run* precision is a measure of variance during a single run. Lippel, *et al*, noted that total precision is usually worse than within-run precision for cholesterol because there are many more sources of variability from run-to-run than would be likely within a single run.[8] The National Cholesterol Education Program has advised that the total CV should be 5 percent or less, and most laboratories now participating in the College of American Pathologists standardization program report total CV under 3.5 percent.[7]

Accuracy is defined as agreement of the test result with the "true value." The most frequently used cholesterol reference method is the manual Abell-Kendall method utilized by the Centers for Disease Control (CDC), generally assessed by comparing cholesterol values obtained by the test method to those of a laboratory whose accuracy has been "traced" back to an accepted reference method, such as that available at the CDC.[7] The Laboratory Standardization Panel of the NCEP has recommended that laboratories achieve an *average* bias less than or equal to ±3 percent within the next

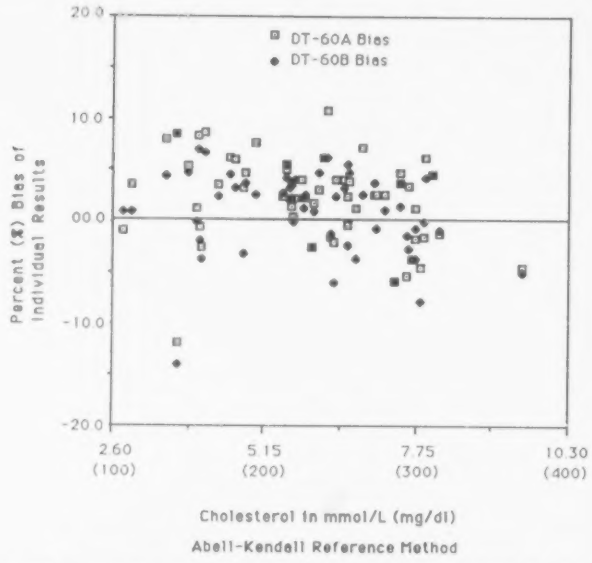

FIGURE 1—Bias Plot Comparing Individual DT-60A and DT-60B Results against the Cholesterol Concentration Determined by the Abell-Kendall Reference Method on Venous Serum Split Samples

five years.[7] Average *bias* is determined by subtracting the mean of a *group* of patient samples run on a new method from the mean of those samples determined using the reference method and then dividing the difference by the mean of the reference method.

Portable cholesterol testing instruments have been available since approximately 1979,[9] and a number of precision and accuracy assessments using portable testing devices have been reported.[4,5,6,10–13] However, previous studies have generally not been performed in screening settings where frequent movement of potentially sensitive equipment, large numbers of different screening personnel with varying degrees of laboratory experience, large patient volume, and changes in environmental temperature and humidity could influence the reliability of test results. The specific purpose of this study was to simulate a community mass screening program and to assess laboratory measurement reliability in a true field setting over an extended time period.

Under the conditions of this study, we noted acceptable results for both precision and accuracy during four months of screening for total cholesterol using either of two DT-60 analyzers we had purchased. Variability remained stable over the entire screening period suggesting that CVs of 3.0 percent may be the best one can achieve under conditions similar to those we encountered. These values for precision and accuracy fall within those recommended as goals for clinical laboratories to achieve by 1993.[7] Thus, assuming operational conditions no worse than those we encountered, one could expect this analysis system to provide both precise and accurate total cholesterol determinations in a public screening setting.

Caution should be exercised in extrapolating results of this study to all screening programs and settings. The results we obtained are probably representative of the range of precision and accuracy values that screening programs might achieve, but a number of variables in other programs could lead to differences. For example, each analyzer is slightly different, as our results showed. Even when instruments are

calibrated using identical materials and operated by the same personnel, results from one instrument to another will vary. We would recommend, therefore, that screening personnel perform assessments of instrument accuracy and precision before undertaking public screenings and ensure that these variances are within accepted laboratory standards. We do not recommend that screening teams rely exclusively on reported biases or precision levels since, as the literature now clearly shows, these may vary from instrument-to-instrument and from operator-to-operator.[6,12,14] These results also cannot necessarily be extrapolated to other types of portable blood analyzers such as the Reflotron by Boehringer-Mannheim or the Vision system by Abbott. To date, reports based on the Reflotron have suggested that neither accuracy nor precision are as acceptable in screening as the results reported here.[15,16] No data have been reported concerning the Abbott system in a screening situation.

In conclusion, we found that a trained group of screening personnel using a portable blood analyzer system yielded acceptable precision and accuracy in a demanding screening-type field setting. We expect that similar results could be achieved by other groups when utilizing equally rigorous surveillance of results and comparable instrumentation.

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Report Shows 16 Alzheimer's Medicines in Development

A new report has been released by the Alzheimer's Association and the Pharmaceutical Manufacturers Association (PMA) showing that 16 medicines are being developed by 11 pharmaceutical companies to treat Alzheimer's disease (AD) and its related symptoms.

The report, titled *Alzheimer's Medicines in Development*, lists chemical and trade names of each drug, as well as its developer and development status. All the medicines in the report are in human testing in the United States.

Alzheimer's disease costs Americans close to $80 billion a year and currently affects an estimated 4 million Americans.

Although none of the test medicines is expected to be a cure, they do represent important progress toward easing the symptoms of Alzheimer's disease.

A copy of the report may be obtained by writing the Alzheimer's Association, 70 E. Lake Street, Suite 600, Chicago, IL 60601; or the Pharmaceutical Manufacturers Association, 1100 15th Street, NW, Suite 900, Washington, DC 20005.

Demographic and Dietary Determinants of Constipation in the US Population

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Abstract: We investigated the association between self-reported constipation and several demographic and dietary variables in 15,014 men and women 12–74 years of age examined between 1971–75 during the first Health and Nutrition Examination Survey. Overall, 12.8 percent reported constipation. Self-reported constipation correlated poorly with stool frequency. Nine percent of those with daily stools and 30.6 percent of those with four to six stools/week, reported constipation. Constipation was more frequent in Blacks (17.3 percent), women (18.2 percent), and those over age 60 (23.3 percent); after adjusting for age, sex, and race it was more prevalent in those with daily inactivity, little leisure exercise, low income, and poor education. Constipated subjects reported lower consumption of cheese, dry beans and peas, milk, meat and poultry, beverages (sweetened, carbonated and noncarbonated), and fruits and vegetables. They reported higher consumption of coffee or tea. They consumed fewer total calories even after controlling for body mass and exercise. (*Am J Public Health* 1990; 80:185–189.)

Introduction

Constipation is a significant problem in the United States with over 330 million dollars spent each year on over-the-counter laxatives.[1] Our understanding of both the epidemiology and pathophysiology is limited.

Studies of constipation are handicapped by lack of a standard definition of the condition. Connell, *et al*,[2] inquired about the bowel habits of 1,055 persons not seeking medical advice in an industrial community. They found that 99.4 percent had three or more bowel movements per week. Based on this work, individuals with fewer than three stools per week are usually considered to be constipated. While physicians and investigators often define constipation by stool frequencies that lie below the usual range, healthy subjects tend to define constipation in terms of function (straining) and stool consistency (hard stools).[3]

Because of the lack of a standard definition, because bowel habits change with time, and because constipation depends on age and sex, the range of reported prevalence is quite broad. In a group of healthy young adults not seeking health care, Sandler and Drossman found that 7.3 percent reported that they were constipated more than 25 percent of the time.[3] When constipation was defined as straining more than 25 percent of the time, Thompson and Heaton found that 10.3 percent of subjects were constipated.[4] In a prospective study of 1,064,004 men and women who were surveyed by American Cancer Society volunteers, 18.5 percent of men and 33.7 percent of women reported "constipation."[5]

Sonnenberg and Koch have recently performed detailed analyses of constipation using data from the National Hospital Discharge Survey, the National Health Interview Survey, the National Ambulatory Medical Care Survey, and the Vital Statistics of the United States.[6] Constipation was the most frequent digestive compliant in the United States with an estimated four million people complaining of "frequent constipation." There were over 92,000 annual hospitalizations with constipation listed among the hospital discharge diagnoses, and 900 deaths from diseases associated with or related to constipation. Using data from the National Disease and Therapeutic Index, the same authors reported 2.5 million physician visits for constipation.[7] The data are limited in several ways. Most people with constipation do not visit physicians, are not hospitalized, and do not die. The reliability of the prevalence estimates for chronic constipation based on the National Health Interview Survey are uncertain because one member of the household provided health information for all members of that household. The published reports of constipation have not attempted to link constipation with dietary and lifestyle factors that might be responsible for this symptom.

The first National Health and Nutrition Examination Survey (NHANES-I) was conducted by the National Center for Health Statistics between 1971–1975.[8] The study involved a nationwide probability sample of 20,749 persons. In addition to detailed socio-demographic information, the survey included data on dietary intake, exercise, and bowel dysfunction. We took advantage of unique data available from the NHANES-I to describe the distribution and determinants of constipation in the United States.

Methods

The NHANES-I was a large-scale, multistage probability sample designed to closely reflect the US non-institutionalized civilian population, 1–74 years of age. The design and methodology have been described in detail elsewhere.[6] The analyses reported here involve 15,014 Black and White persons older than age 12 at the time of the survey, who completed both an examination and a dietary interview.

Constipation, in the present study, was based on responses to the question: "Do you have trouble with your bowels that makes you constipated or gives you diarrhea?" Those who answered "yes—constipated," were defined as having self-reported constipation and were compared to those who responded negatively to the question. Those with diarrhea (621), constipation and diarrhea (73), and those with missing information (72) were excluded. Subjects were also asked: "How often do you usually have a bowel movement?" Stool frequency was coded by the National Center for Health Statistics as less than two stools per week, two to three per week, four to six per week, one per day, and two or more per day. It was not possible to analyze stool frequencies apart from these categories. An alternative definition of constipation was fewer than four stools per

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week. No time period for self-reported constipation or stool frequency was specified.

Height and body weight were obtained through standardized procedures.[6] Body mass index (BMI) was calculated using the formula BMI = weight (kg.)/height $(m)^2$ to provide an index of weight controlled for height. Daily calories were determined using standardized food composition tables applied to foods in each person's 24-hour dietary recall record. Subjects were also asked the frequency with which they consumed a variety of foods with categories chosen to provide reasonably balanced numbers in each category. Race was dichotomized as White or Black.

Analyses were performed using the Statistical Analysis System (SAS Institute, Cary, NC). The SESUDAAN program, which takes into account the complex sample design of the survey, was used to compute standardized proportions and standard errors.[9] The RTILOGIT program[10] was run under SAS to perform logistic regression analyses that took account of the sample weights and sample design. Age, body mass index, and calories were entered into models as continuous variables. Categorical variables were coded as described in the Tables.

Results

Overall, 12.8 percent (S.E. 0.48) of the United States population reported constipation (Table 1). Self-reported constipation was more common in Blacks (17.3 percent), women (18.2 percent), and those over age 60 (23.3 percent). When we defined constipation as fewer than four stools per week, constipation was again more common in Blacks and women. The elderly did not consistently report fewer stools.

There was poor correlation between the reported stool frequency and self-reported constipation (Table 2). Only 67.8 percent of those with fewer than two stools per week reported constipation. Nine percent of those with daily stools, and 30.6 percent of those with four to six stools per week considered themselves constipated. Of the 180 persons with less than two stools per week, 137 (76 percent) were women, supporting the fact that constipation is more common in women.

Self-reported constipation was most prevalent in those with daily inactivity and little recreational exercise with a distinct gradient from the most active to the least active at all ages (Table 3). Those with low income and education were more likely to be constipated within each age category. The effect of marital status was inconsistent. Reported constipation was slightly more common in those living in the South. When we stratified by race and sex (not shown), all of these findings persisted. They also persisted when we compared those with fewer than four stools per week (constipated group) to those with more than four.

Table 4 presents data on a number of specific dietary constituents. Constipated individuals reported lower consumption of cheese and cheese dishes, dry beans and peas, milk, fruits and vegetables, meat and poultry, and sweetened, carbonated and non-carbonated beverages, and higher consumption of coffee and tea. There was no consistent relationship with cereal or bread. These associations largely persisted for White men and women and when we defined constipation as fewer than four stools per week. The results for Blacks were not as consistent but were based on much smaller numbers within individual dietary categories.

In order to assess the independent effects of these variables, we examined them in a logistic regression model that also contained terms for age, race, sex, body mass index, and total daily calories. Each individual dietary item was

TABLE 1—Proportion of Persons with Self-Reported Constipation in the US Population

Group	No.	% Constipated*	Standard Error
Overall	15,014	12.8	0.48
Race			
White	12,178	12.2	0.48
Black	2,836	17.3	1.40
Sex			
Men	6,042	7.0	0.50
Women	8,972	18.2	0.70
Age (years)			
12–29	5,660	8.1	0.54
30–59	5,470	13.9	0.61
60–75	3,884	23.3	1.21

*Percents are US population estimates derived by applying sampling weights to survey data.

TABLE 2—Comparison of Stool Frequency to Self-Reported Constipation

Stool Frequency[a]	No. (%)	% Constipated[b]
<2/week	180 (1.0)	67.8
2–3/week	1,342 (7.6)	58.3
4–6/week	1,884 (10.6)	30.6
1/day	12,442 (70.0)	9.0
≥2/day	1,930 (10.9)	7.0

[a]"How often do you have a bowel movement?"
[b]"Do you have trouble with your bowels which makes you constipated?"

TABLE 3—Proportion of Persons with Self-Reported Constipation in the US Population by Age and Personal Characteristics

	Age (years)		
	12–29	30–59	60–75
	% SEM[a]	% SEM[a]	% SEM[a]
Nonrecreational Exercise[b]			
Very active	6.9 (0.6)	11.7 (0.8)	17.9 (1.2)
Moderately active	8.7 (0.7)	15.9 (0.9)	25.8 (1.7)
Inactive	15.9 (3.1)	17.2 (2.3)	29.0 (3.2)
Recreational Exercise[c]			
Much exercise	6.0 (0.6)	8.9 (1.0)	18.0 (2.4)
Moderate exercise	7.5 (0.8)	12.7 (0.9)	19.3 (1.7)
Little exercise	14.3 (1.3)	17.1 (1.1)	27.8 (1.7)
Income			
$<6,999	9.6 (1.2)	20.6 (1.9)	28.8 (1.6)
7,000–9,999	8.3 (0.8)	14.9 (1.3)	18.4 (2.1)
10,000–14,999	7.9 (0.9)	11.5 (1.0)	17.1 (3.5)
>14,999	6.4 (0.9)	9.8 (1.2)	10.3 (1.9)
Education (years)			
0–6	9.8 (2.6)	25.6 (3.1)	33.5 (2.8)
7–11	7.0 (0.7)	15.8 (1.2)	26.6 (1.7)
12	8.6 (0.8)	12.7 (8.9)	16.6 (2.5)
13+	8.9 (0.9)	10.7 (0.9)	15.4 (2.1)
Marital Status			
Never Married	6.6 (0.6)	20.0 (2.3)	23.4 (3.7)
Married	10.8 (0.8)	12.8 (0.6)	21.8 (1.5)
Region			
Northeast	7.8 (0.8)	13.3 (1.1)	18.4 (2.1)
Midwest	7.1 (0.9)	12.7 (0.9)	23.5 (1.9)
South	9.6 (1.6)	16.6 (1.7)	28.4 (3.2)
West	8.1 (0.9)	13.4 (1.2)	23.0 (2.6)

[a]Standard error of the mean
[b]Subjects were asked: "In your usual day, aside from recreation, how active are you?"
[c]Subjects were asked: "Do you get much exercise in things you do for recreation?"

TABLE 4—Proportion of Persons with Self-Reported Constipation in the US Population by Dietary Constituents Consumed

	Frequency of Consumption[a]				
	low 1	2	3	4	high 5
	% SEM[b]	% SEM	% SEM	% SEM	% SEM
Cheese	15.9 (1.3)	14.1 (1.1)	12.9 (0.6)	11.2 (0.6)	12.6 (1.1)
Cereal	11.9 (0.8)	13.9 (0.9)	11.8 (0.6)	16.1 (1.3)	11.9 (9.4)
Beans	20.6 (1.4)	12.7 (0.7)	12.6 (0.6)	10.7 (0.8)	9.2 (1.0)
Bread	15.4 (6.0)	13.4 (3.9)	13.3 (1.0)	12.6 (0.6)	12.9 (0.6)
Milk	15.3 (1.2)	14.8 (1.7)	14.2 (0.8)	12.4 (0.6)	9.9 (0.8)
Fruit/vegetables	2.6 (2.1)	18.4 (4.1)	14.0 (1.3)	12.3 (0.6)	13.1 (0.7)
Meat/poultry	28.2 (11.1)	22.1 (6.2)	15.9 (1.3)	12.4 (0.5)	5.9 (1.3)
Coffee/tea	7.9 (0.8)	10.3 (1.7)	9.6 (1.1)	13.8 (0.6)	
Beverages	18.0 (1.0)	15.8 (1.0)	11.5 (0.7)	10.2 (0.7)	

[a]Cheese refers to cheese and cheese dishes; beans to dry beans and peas; beverages to sweetened, carbonated and non-carbonated. The categories for the following specific items were: *milk*: (1) never, (2) less than once a week, (3) 1–6 times a week, (4) once a day, (5) more than once a day; *meat and poultry, fruits and vegetables, bread*: (1) never, (2) less than once a week, (3) 1–6 times a week, (4) 1–2 times a day, (5) more than twice a day; *cheese, beans*: (1) never, (2) less than once a week, (3) 1–2 times a week, (4) 3–6 times a week, (5) once or more a day; *cereal*: (1) never, (2) less than once a week, (3) 1–6 times a week, (4) one or more times a day. *Beverages* (sweetened, carbonated and non-carbonated) and *coffee or tea* were ranked in four categories by the NCHS (1) never, (2) less than once a week, (3) 1–6 times a week, (4) once or more a day.
[b]SEM = Standard error of the mean.

statistically significant ($p < 0.001$) in the same direction as the univariate analyses shown in Table 4.

Body mass index was similar for those who reported constipation and those who did not at every age and in both sexes, whereas the constipated group consumed fewer calories (Table 5). In order to assess the independent effects of various demographic and lifestyle factors on reported constipation we performed a logistic regression analysis that simultaneously controlled for all variables in the model and took into account the sample weights and sample design (Table 6). The most important determinant of constipation was sex. Women were 2.4 times more likely to report constipation after controlling for other factors. The other results were in the same direction as the univariate analyses. Constipation was more common in Blacks, the elderly, and those with less education, low income, and inactivity. Marital status and body mass index were not important. Constipated subjects consumed fewer calories even after controlling for the other variables in the model.

TABLE 5—Age-Specific Mean Body Mass Index and Daily Calorie Consumption by Constipation Category[a]

	Age (years)		
	12–29	30–59	60–75
Women			
Body Mass Index[b]			
Constipated	22.7 (0.3)	25.6 (0.2)	27.1 (0.3)
Not constipated	22.2 (0.1)	25.5 (0.1)	26.4 (0.2)
Calories			
Constipated	1698.9 (48.3)	1493.8 (31.1)	1229.4 (33.7)
Not constipated	1762.7 (27.5)	1554.4 (18.8)	1350.5 (23.7)
Men			
Body Mass Index[b]			
Constipated	23.9 (0.7)	26.2 (0.4)	25.3 (0.4)
Not constipated	22.8 (0.1)	26.1 (0.1)	25.5 (0.1)
Calories			
Constipated	2655.5 (125.9)	2195.0 (84.4)	1789.8 (83.1)
Not constipated	2847.2 (39.7)	2440.2 (34.4)	1944.1 (38.6)

[a]Numbers in parentheses represent the standard error of the mean.
[b]Body mass index = weight (kg)/height $(m)^2$

Discussion

Our results are representative of the non-instiutionalized civilian US population. The large size of the study (15,014 subjects) permitted us to look at several demographic and dietary characteristics in order to present a more complete description of constipation. The results confirm other reports and indicate that recreational and nonrecreational inactivity is associated with self-reported constipation independent of dietary intake. Exercise is thought to increase propulsive movements in the large intestine[11] although there are not good studies to support the use of exercise in constipated subjects.[12]

The NHANES-I survey was conducted between 1971 and 1975. There has been a modest decrease in the prevalence of "frequent constipation" between 1968 and 1986.[6] We chose the first NHANES-I because subjects were asked about both stool frequency and self-reported constipation. There were only 180 subjects with fewer than two stools per week, too few to permit detailed analyses. We performed all analyses using stool frequency less than four per week (8.6 percent of the population) as well as self-reported constipation, and found no major differences in the results for diet and demographic features with one exception. The elderly did not report consistently fewer stools than the young even though they reported more constipation.

In the absence of an agreed upon definition of constipation, we believe that self-reports of the symptom of constipation may be preferable to stool frequency since infrequent stools may be asymptomatic and simply represent the lower end of the continuum. Self-reported constipation may refer to several different symptoms including infrequent stools, feelings of incomplete evacuation, straining and hard stools[3] which may, in turn, be the consequence of several discrete pathogenetic mechanisms. It is possible that the differences that we observed in constipation prevalence by age, sex, and race are due to differences in definitions used by respondents. The similarity of our age and sex findings to those based on physician diagnosis, however, argues against this interpretation.[7]

There is value to studying self-reported constipation since it is this symptom that distresses patients. Epidemiol-

TABLE 6—Constipation in the US Population, Logistic Regression Analysis[a]

Variables	Beta	S.E.	Odds Ratio
Female sex	0.8803	0.0007	2.41
Black race	0.2849	0.0008	1.33
Age (10)	0.2063	0.0002	1.23
Nonrecreational activity	0.1813	0.0004	1.20
Recreational activity	0.1522	0.0004	1.16
Married	−0.0090	0.0001	0.99
Body mass index (10)	−0.0279	0.0005	0.97
Calories (1,000)	−0.1391	0.0004	0.87
Income	−0.1369	0.0003	0.87
Education	−0.1503	0.0003	0.86

[a]Adjusting simultaneously for all other variables in model using RTILOGIT[10] to take account of sample weights and sample design. For education, income, nonrecreational activity, and recreational activity, betas (and odds ratios) reflect comparisons between individual categories described in Table 3. Age and body mass index were entered as continuous variables with betas calculated for intervals of 10. Total calories were entered as a continuous variable with the beta reflecting an interval of 1000 calories.

ogists have been reluctant to study conditions for which there may not be pathologic, radiologic, or laboratory confirmation. We need to study the epidemiology of symptoms and symptom complexes, however, since they represent a large part of human morbidity and suffering.[13]

Diet, particularly dietary fiber, has often been regarded as an important determinant of constipation,[14,15] although personality factors may be important as well.[16] The large sample size and detailed dietary data available in the NHANES-I permitted us to examine the association between diet and constipation. We found that those who ate more fruits and vegetables had fewer complaints of constipation. In addition, however, we found that those who ate more of a number of dietary constituents including cheese, dry beans and peas, milk, and meat and poultry also had less self-reported constipation. As one might expect, these individuals also had a higher caloric intake. When we controlled for caloric intake, however, these dietary components were still significant.

One of the most interesting findings was that constipated subjects ate fewer calories. The constipated group was not leaner, and their lower calorie consumption was not completely explained by decreased physical exercise or differences in other demographic characteristics, although these could have been incompletely controlled in the logistic model (residual confounding). The inverse association between calorie consumption and self-reported constipation may offer clues about etiology. Caloric intake is based on body size, physical activity, metabolic efficiency, and net energy balance (i.e., change in body fat or muscle).[17] The mechanisms of metabolic efficiency, including differences in absorption and thermogenesis, are poorly understood, but individual differences must exist since even under controlled conditions some subjects will gain more weight than others eating similar calories.[18]

One might hypothesize that individuals with constipation are more efficient at absorbing or utilizing food in their small intestines. They eat less, present less to their colons, and have more constipation as a consequence. Those free of constipation, on the other hand, are less efficient. Food that escapes absorption enters the large bowel where fecal flora may produce short chain or volatile fatty acids that protect against constipation.

The absorption of dietary constituents by the human small bowel is known to be incomplete. Kramer, *et al,*[19] found that several different dietary components increased the ileal effluent in seven colectomized subjects. The starch moiety of most staple foods (wheat, corn, oats, potatoes) is incompletely absorbed by normal subjects,[20] and there may be considerable variation among individuals. Stephen, *et al,*[21] found that while some individuals absorbed starch well, others failed to absorb 15–20 percent. It is possible that functional constipation could be due to overly efficient handling of the carbohydrate that normally escapes absorption in the small bowel.[22]

In summary, we found that self-reported constipation was more common in women, Blacks, the poor, the elderly, and those with less education and little physical exercise. Those who ate fewer calories were more constipated even after controlling for exercise and body mass index. In order to reconcile all of the available information, one might view constipation as the end result of both exogenous factors (diet and exercise) and endogenous factors (sex hormones, personality, and metabolic efficiency). Further studies on energy balance and absorption in constipated subjects may be important in helping us to understand and treat this common symptom. Such studies will require large numbers and careful consideration of symptom definition.

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Nation's First Medical School Celebrates Its 225th Anniversary throughout 1990

The University of Pennsylvania School of Medicine—the first medical school established in the United States in 1765—marks its 225th anniversary during 1990, at the same time the University notes its 250th anniversary. A year-long celebration is planned to celebrate this landmark in the history of American medicine. A blend of history, contemporary scholarship, and a look at the future will be linked with festivities to make 1990 a memorable year for alumni, faculty, students, and friends.

The continuing theme throughout the year will be the evolution of medicine in the United States and the critical role the University of Pennsylvania School of Medicine played in the process, accentuated by exhibits, lectures, symposia, and exchanges to highlight the major contributions of the school. Three peak celebration periods are planned for the year—occurring in January, May and October.

- The January program emphasized frontiers of science in medicine, and centered around the dedication of a new clinical research building on the site of the former Philadelphia General Hospital. Louis Sullivan, MD, US Secretary of Health and Human Services, delivered the keynote address which focused on national health policy. Other events in the January program included the 27th annual Women in Medicine dinner, and a scientific symposium entitled "Molecular Mechanisms of Disease" featuring three of the world's foremost medical scientists: Philip Leder, MD, Harvard University; Sydney Brenner, MD, Cambridge, England; and Joshua Lederberg, PhD, Rockefeller University.
- The "May Festival" will take place May 16–20, and will include dedication of the Seymour Gray Foundation for Molecular Medicine: Seymour Gray Lecture in Molecular Biology. Keynote speaker will be Michael S. Brown, MD, Nobel Laureate and Professor of Molecular Genetics, University of Texas-Southwestern Medical School. "Medical Alumni Weekend" a major annual event—will be incorporated into the festivities, with numerous social activities, and a scientific symposium entitled "Recent Developments in Cancer Research."

Also in May, continuing medical education credit will be given for a series of faculty-alumni exchanges that are part of the Medical School's 225th anniversary.

- The "Future of Medicine and Health Care" will be the focus of the October festivities, with a symposium on October 12 featuring three keynote speakers: Arnold Relman, MD, Editor, *New England Journal of Medicine*; Samuel O. Thier, MD, President, Institute of Medicine/National Academy of Sciences; and Uwe E. Reinhardt, PhD, Professor of Political Economy, Princeton University.

For further information, contact: Office of the 225th Anniversary Celebration, University of Pennsylvania School of Medicine, 196 John Morgan Building, 36th and Hamilton Walk, Philadelphia, PA 19104; or the Office of Public Information, University of Pennsylvania Medical Center, 2 Blockley Hall, 3400 Spruce Street, Philadelphia, PA 19104-6021. Tel: (215) 662-2560.

Epidemiologic Assessment of Screening Tests for Antibody to Human T Lymphotropic Virus Type I (HTLV-I)

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Abstract: We tested 196 sera from a human T lymphotropic virus type I (HTLV-I) risk group (prostitute women) with two commercial "research" enzyme-linked immunoabsorbent assays (EIA) for HTLV-I antibodies. All tested sera were characterized by HTLV-I Western immunoblots and by HTLV-I radioimmunoprecipitation assays. The estimated sensitivities of the EIA tests were 93.8 percent and 100 percent, and the specificities were 98.8 percent and 95.8 percent, respectively, using recommended criteria for seropositivity (requiring reactivity to both *gag* p24 and *env* gp46 or gp61/68). Calculated negative predictive values remained excellent (>99.9 percent and 100 percent, respectively) at lower seroprevalence rates but the positive predictive values were only 7.3 percent and 2.3 percent when calculated for a seroprevalence rate of 0.1 percent. These results emphasize the importance and need for additional HTLV-I supplementary serologic testing when screening populations with low HTLV-I seroprevalence rates. (*Am J Public Health* 1990; 80:190–192.)

Introduction

Human T lymphotropic virus type I (HTLV-I), the first human retrovirus discovered,[1] is causally linked to adult T-cell leukemia/lymphoma (ATL)[2] and has been more recently associated with a progressive myelopathy known as tropical spastic paraparesis (TSP) in the Caribbean and as HTLV-I-associated myelopathy (HAM) in Japan.[3,4] HTLV-I infection is endemic in southwestern Japan, the Caribbean, and in part of Central America and Africa.[5] In the United States, elevated rates of infection have been reported in intravenous drug users[6,7] and in recipients of multiple blood transfusions,[8] raising concerns about the safety of the nation's blood supply. A recent study of random blood donors in eight US cities showed a seroprevalence of 0.025%.[9]

Screening tests for antibodies to HTLV-I were licensed by the Food and Drug Administration (FDA) in November 1988, and the FDA has recommended that these tests be used to screen blood donated for transfusion.[10] The purpose of our study was to evaluate two commercially available HTLV-I enzyme-linked immunoabsorbent assays (EIA)* using a set of sera from a population at high risk for HTLV-I infection, and to estimate the predictive values expected with these tests when testing populations or when screening blood donors from populations with low rates of infection.

Methods

Serum Specimens

The 200 serum specimens used in this study were systematically chosen (every 7th specimen until a total of 200 were obtained) from a set of 1,541 sera from US prostitutes with a high HTLV-I seroprevalence rate (7 percent).[11] All specimens had been frozen and stored at the Centers for Disease Control (CDC) at −20°C and were thawed for this study.

HTLV-I Antibody Testing

All serum specimens were tested by two HTLV-I EIAs (Dupont Co., Wilmington, DE, and Cellular Products Inc., Buffalo, NY); both used viral lysates from HuT-102-infected cells and differed mainly in their respective manufacturer's recommended cutoffs: 0.6 × mean of positive controls for Dupont's and 0.25 × mean of positive controls for the Cellular Products'. Initially reactive sera were retested in duplicate and classified as reactive or nonreactive for the test based on the manufacturer's recommendation. Because we needed to know whether each specimen was positive, indeterminate, or negative (see following), 196 of the 200 serum specimens, including all those reactive in either screening test, were further tested by Western immunoblot (WIB) and by radioimmunoprecipitation assay (RIPA); four specimens, negative on both EIAs, were too small for us to do WIB or RIPA and therefore were excluded from further analysis.

WIB used an HTLV-I-infected cell lysate purified from MT-2 cells (Hillcrest Biologicals, Cypress, Calif).[12] HTLV-I antigen was suspended in a sample buffer, heated at 95–100°C for four minutes, and subjected to electrophoresis in a single well of a 10 percent polyacrylamide gel (PAGE); PAGE-resolved proteins were electrophoretically transferred to nitrocellulose sheets and washed sheets were cut into 3.0 mm strips. Individual strips were incubated with 1:100 dilutions of serum overnight at 4°C, washed, incubated for 60 minutes at room temperature with 5 μg/ml biotinylated goat anti-human IgG (Vector Laboratory, Burlingame, CA). Strips were washed again and then incubated with avidin-biotin-peroxidase complex according to the manufacturer's instructions (Vector Laboratory, Burlingame, CA). Color reactions were developed with 3, 3'diaminobenzidine, nickel chloride and H_2O_2 (Sigma Chemical Co., St. Louis, MO).

For the RIPA, HTLV-I-infected MT-2 cells were metabolically labeled with [^{35}S]cysteine and [^{35}S]methionine (200 uCi of each nucleotide/ml/1 × 10^7 cells), disrupted in lysing buffer, centrifuged, and incubated with the sera. Immunocomplexes were precipitated with protein A-sepharose (Sigma Chemical Co., St. Louis, MO). Samples were analyzed by PAGE followed by autoradiography of dried gels.

In each test run, banding patterns were compared with those of a known positive serum, and a negative serum was included as a negative control. Specificity of banding patterns

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*At the time of this study these tests, which had not been licensed, were approved for research only.

was assessed by comparison with banding patterns on strips that had been incubated with monoclonal antibodies against HTLV-I-specific proteins (p19, p24 for WIB and gp61/68 for RIPA).[13,14]

Definitions

In this study, a seropositive serum sample was defined by the presence of antibodies to *gag* (p24) and *env* (gp46 or precursor gp61/68) on WIB or RIPA.[10] A serum sample with immunoreactivity to at least one HTLV-I gene product but not satisfying these criteria was called indeterminate. All remaining specimens were designated negative.

Calculation of Predictive Values

Negative and positive predictive values were calculated using sensitivity, specificity, and prevalence as outlined in Fleiss.[15]

Results

Of the 196 sera included in the analysis, 16 (8 percent) were HTLV-I seropositive by WIB or RIPA. Of those, seven fulfilled the seropositivity criteria on WIB alone; nine showed only *gag* reactivity on WIB and required RIPA to demonstrate antibody to *env*. Fourteen specimens (7 percent) were indeterminate after WIB and RIPA; all showed reactivity to *gag* gene products only, and no specimen showed reactivity on RIPA that did not also show reactivity on WIB. The remaining 166 specimens were negative.

Of the 16 seropositive specimens, 15 were repeatably reactive by Dupont EIA, for a test sensitivity of 93.8 percent, and all 16 were reactive by Cellular Products' EIA, for a test sensitivity of 100 percent (Table 1). Excluding the 14 serum specimens with indeterminate HTLV-I serologic results, the specificities of the tests were 98.8 percent and 95.8 percent, respectively.

Table 2 shows the positive and negative predictive values of these tests, calculated for different seroprevalence rates; the calculations were based on the sensitivities and specificities estimated in this study: the predictive value of a negative test result increased with lower seroprevalence levels and was >99.9 percent for Dupont's EIA at seroprevalence levels of ≤0.1 percent; the negative predictive value of Cellular Products' EIA was 100 percent for all seroprevalence rates. In contrast, the predictive value of a positive test result decreased with lower seroprevalence rates and was <8 percent for Dupont's and <3 percent for Cellular Products' at seroprevalence rates lower than 0.1 percent.

TABLE 1—HTLV-I Antibody Test Results of 196 Serum Specimens by Repeatable Reactivity in Two HTLV-I Enzyme-Linked Immunoassays

Results of Tests for HTLV-I	No. Repeatably Reactive	
	by Dupont's EIA*	by Cellular Products' EIA
Positive (N = 16)	15	16
Indeterminate (N = 14)	2	10
Negative (N = 166)	2	7

*At the time of the study these tests, which had not been licensed, were approved for research only; the recommended cutoff for the Dupont test has changed with licensure.

TABLE 2—Performance Characteristics of Two EIAs and Resulting Predictive Values for Different Seroprevalence Rates*

	Dupont's EIA		Cellular Products' EIA	
Sensitivity (b)	93.8%		100%	
Specificity (a)	98.8%		95.8%	
if:	PPV[§]	NPV[¶]	PPV	NPV
p = 8%	87.2%	99.5%	67.4%	100%
p = 1%	44.1	99.9%	19.4%	100%
p = 0.1%	7.3%	99.9%	2.3%	100%
p = 0.05%	3.4%	99.9%	1.2%	100%
p = 0.025	1.7%	99.9%	0.6%	100%

*Sera with indeterminate HTLV-I reactivities were excluded from these analyses.

[§]PPV = positive predictive value = $\frac{(b)(p)}{(b)(p) + (1-a)(1-p)}$, where b = sensitivity, a = specificity, p = prevalence

[¶]NPV = negative predictive value = $\frac{a(1-p)}{a(1-p) + (1-b)p}$

Discussion

This paper is not intended to be a comparison of the performance of the two tests. Neither test was licensed by the FDA at the time of the study and the recommended cutoff for the Dupont test has in fact changed with licensure* and is now similar to the Cellular Products' test. Rapid developments in the field may result in additional changes in these tests in the future. Rather, our data are intended to illustrate that both tests have low positive predictive values in groups with low prevalence of infection. Repeating the study with the currently licensed tests would not have changed this conclusion. Our data emphasize the need, with current assays, for more specific supplementary serologic testing before interpreting a sample as seropositive. Further serologic confirmatory testing must precede any decision to counsel a tested individual.

Our study suggests that, in populations with HTLV-I seroprevalence rates <0.1 percent, less than 10 percent of repeatably reactive test results might be found to be truly positive. While quite accurate for populations where sexually transmitted diseases and other infections are prevalent, these estimates of positive predictive value (PPV) must be more cautiously interpreted in screening US blood donors. In the American Red Cross study of 39,898 blood donors, for example, 10 of 68 specimens repeatedly reactive in a screening test were confirmed as positive for HTLV-I antibody, yielding a PPV of 15 percent.[9] This discrepancy is probably due to a higher specificity of the screening tests in the blood donor population. Specificity could not be calculated accurately in the American Red Cross study because supplementary testing was done only on EIA-reactive specimens, but specificities of 99.3 percent to 99.9 percent (compared with 98.8 percent and 95.8 percent in our study) have been estimated in blood donor populations.[10] Additionally, our

*Human T-lymphotropic virus type I (HTLV-I) Dupont HTLV-I ELISA: Product Insert Sheet

lower estimates of PPV could be the result of differences in the cutoff used to interpret these tests, and to technical differences in the supplementary serologic tests. Despite these discrepancies, it is still clear that PPVs are low in low-prevalence populations.

Our criteria for seropositivity have been recommended by the US Public Health Service Working Group[10] and require reactivity to both *gag* and *env* gene products; they currently necessitate at least Western blotting and in some instances WIB and RIPA as *env* reactivity appears to be better on RIPA than on WIB. The significance of indeterminate HTLV-I serologic results is not fully understood; these may represent nonspecific reactivity to cellular proteins, cross-reactivity with other unknown retroviruses, or early seroconversion to HTLV-I. The uncertainty regarding the significance of HTLV-I indeterminates resembles the situation with human immunodeficiency virus (HIV) where the full significance of sera with "indeterminate" HIV WIB remains to be determined[16]; further studies including serial serologic testing of persons with indeterminate HTLV-I serologies and examination of peripheral mononuclear cells with the new polymerase chain reaction (PCR) technique,[17] we hope, will provide evidence for or against viral infection. In analyzing our data, we excluded the indeterminates and thus avoided the issue of whether to count them as positives or negatives. Including the indeterminates with the positives would lower the calculated sensitivities of the screening tests; including them with the negatives would lower their specificities. Either way the resulting PPVs would be lower than the ones we calculated. For example, at a prevalence rate of 0.1 percent, including the indeterminates with the positives produces a PPV of 4.5 percent for Dupont's (compared with 7.3 percent) and 2.0 percent for Cellular Products' (compared with 2.3 percent); including them with the negatives changes the PPVs to 4.1 percent and 1.0 percent, respectively.

Based on our data, 11 to 21 percent of repeatably reactive specimens on EIA may be negative for HTLV-I antibodies (no bands on WIB). As blood is discarded from donors whose EIA results are repeatably reactive after screening, as much as 21 percent of discarded blood may be HTLV-I-negative. However, the objective of screening is an HTLV-I-free blood supply because HTLV-I is transmissible by transfusion of HTLV-I infected blood[18]; screening of the blood supply should achieve this. A limitation of current HTLV-I serologic tests, including WIBs and RIPAs, is that they cannot distinguish infection with HTLV-I from infection with HTLV-II, a closely related human retrovirus with no clear disease associations.[19] At present, HTLV-I and HTLV-II can be separated by amplification of viral specific sequences in infected cells using polymerase chain reaction (PCR).[20]

Acronyms Used

ATL, adult T-cell leukemia/lymphoma; CDC, Centers for Disease Control; EIA or ELISA, enzyme-linked immunoabsorbent assay; FDA, Food and Drug Administration; HAM, HTLV-I-associated myelopathy; HIV, human immunodeficiency virus; HTLV, human T lymphotronic virus (I and II); PAGE, polyacrylamide gel; PCR, polymerase chain reaction; PPV, positive predictive value; RIPA, radioimmunoprecipitation assay; TSP, tropical spastic paraperesis; WIB, Western immunoblot.

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Public Health Briefs

Comparison of Two Methodologies to Measure Agricultural Occupational Fatalities

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Abstract: Agricultural occupational fatalities in Pennsylvania for the years 1985–87 were followed up. Supplemental data concerning the occupation of the deceased and circumstances of the fatal accident were obtained from a family member. The number of fatalities designated as agriculturally and occupationally related by the National Traumatic Occupational Fatality (NTOF) system was compared with the number of fatalities identified by using alternative criteria for classification of agriculturally and occupationally related fatalities. There may be nearly a 30 percent error in the NTOF method resulting in a 20 percent undercount. (*Am J Public Health* 1989; 80:198–200.)

Introduction

The National Safety Council (NSC) estimates that agricultural industry fatalities for 1984* were 46 deaths per 100,000 workers[1] while the National Institute for Occupational Safety and Health (NIOSH) estimates them at 18.36 for the same year.[2]

The reliability and validity of the data used to generate these estimates have been questioned. Of specific concern is that these data are derived from information on death certificates regarding usual occupation, type of business or industry, and injury at work categories.[2–6] In agriculture, occupational work is difficult to distinguish from non-occupational work since the worksite is often the place of residence. This combination results in exposure to worksite hazards in both working and non-working situations and in both occupational and non-occupational activity.[7–9]

Additionally, the agricultural labor force is composed in part of individuals less than 16 years of age, individuals who work in other occupations on a full-time basis, and family members and relatives who may lend a helping hand only occasionally. These labor characteristics are not normally the case in other industries. Furthermore, agricultural land is often used for recreational purposes such as hunting, swimming, riding, etc, and for both non-occupational and occupational work. Lastly, differing methodologies are used to record and classify agricultural fatalities by the NSC and NIOSH.[1,2]

This paper compares the number of fatalities designated as both agricultural and occupational by the National Traumatic Occupational Fatality (NTOF) system, a system developed by NIOSH, with the number of fatalities identified by using alternative criteria for classification.

Methods

NIOSH developed the NTOF classification system to monitor occupational fatalities for all industrial sectors in the United States, including agriculture. The NTOF system collects and analyzes data on traumatic occupational fatalities using information from death certificates recorded by vital statistics reporting units in all 50 states and the District of Columbia.[2] These selected death certificates are then coded for the "usual industry" of the victim by divisional level industry categories listed in the Standard Industrial Classification Manual.[2,10] The assumption is made that "usual industry" at the divisional level is a proxy for the occupation engaged in at the time of the fatality. The number of death certificates coded into the agricultural industry are then considered agricultural occupational fatality cases.

We obtained copies of all death certificates for the years 1985–87 that indicated deaths that were accidental, and included "farm" or "agriculture" in any category (N = 141) from the Pennsylvania Department of Health, Division of Health Statistics and Research. For the same time period, a clipping service forwarded all newspaper accounts of farm and agricultural accidents and fatalities occurring in Pennsylvania.

The sample of fatality cases was reduced to 100 since only the death certificates that could be matched with a newspaper clipping containing the name and address of the deceased member's family were used. Of these 100 fatalities, 66 (66 percent) individuals from families of the deceased agreed to participate in the study. This group will hereafter be referred to as the supplemental data group.

A family member, usually a spouse, parent, or child of the deceased, was contacted by either telephone or mail to obtain supplemental data on the deceased's primary or usual occupation, secondary or part-time occupation, and the relationship between the activity at the time of the fatality and the deceased's primary or secondary occupation, non-occupational work, recreational pursuits, or other activities. The terms "primary or usual occupation" and "secondary or part-time occupation" were not further defined to the individuals questioned. Further information on the methodology

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*The agricultural industry, as defined by Standard Industrial Classification (SIC) codes 01 through 09 includes production agriculture (farms, ranches, commercial orchards, truck gardens, etc.), various agricultural services, commercial hunting and fishing, and forestry, excluding logging.

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used to collect the supplemental data are detailed elsewhere.**

The alternative criteria for designating an agricultural occupational fatality included cases whose victims were:

- engaged in a work activity related to *either* their primary or secondary occupation of agriculture at the time of the fatality,
- under the age of 16 and engaged in a farm work activity, or
- under the age of 16 and injured by exposure to an agricultural worksite hazard although not engaged in an agricultural work activity.

Results

Our alternative criteria and the supplemental data were used as one data source when comparisons, explained below, were made. Because of the single data source, the effect of the alternative criteria and the supplemental information cannot be separated in this analysis. Table 1 compares characteristics of three groups: cases with supplemental data, cases that did not respond to our survey, and cases for which there was no match with a newspaper clipping. There are no differences between the 66 cases followed up and the other two groups.

Twenty-six of the 66 cases (39 percent) were identified by the NTOF criteria as agricultural occupational fatalities; the remaining 40 cases (61 percent) were excluded. Using the alternative criteria and supplemental information, 39 of the 66 cases (59 percent) were classified as agricultural occupational fatalities; the remaining 27 cases (41 percent) were excluded.

**Seltzer B, Murphy D, Yesalis C: A methodology for the collection of supplemental information on agricultural fatalities. Paper submitted for publication, September 1988.

TABLE 1—A Comparison of Selected Variables between the Supplemental Data Group and the Non-response and Death Cerficate Only Groups

Variables Compared	Groups		
	Supplemental Data Group (%)	Non-response Group (%)	Death Certificate Only Group (%)
Sex			
Male	63 (95)	33 (97)	35 (85)
Female	3 (5)	1 (3)	6 (15)
Age (years)			
Under 16	9 (14)	8 (24)	7 (17)
16 or over	57 (86)	26 (76)	34 (83)
Time of death			
AM	22 (33)	6 (18)	9 (22)
PM	44 (67)	27 (79)	31 (76)
Unknown	—	1 (3)	1 (2)
Injury at work			
Yes	31 (47)	19 (56)	13 (32)
No	28 (42)	14 (41)	25 (61)
Blank	7 (11)	1 (3)	3 (7)
Accidental injury			
Yes	63 (95)	31 (91)	40 (98)
No	—	1 (3)	—
Blank	3 (5)	2 (6)	1 (2)
Medical examiner			
Yes	56 (85)	30 (88)	35 (85)
No	4 (6)	2 (6)	3 (7)
Blank	6 (9)	2 (6)	3 (7)
Autopsy			
Yes	22 (33)	12 (35)	16 (39)
No	42 (64)	20 (59)	24 (59)
Blank	2 (3)	2 (6)	1 (2)

Of the 26 cases designated by NTOF as agricultural occupational fatalities, 3 (12 percent) were found to be non-agricultural occupational; the misclassification was due to relying on "usual industry" as a proxy for the occupation engaged in at the time of the fatality.

Sixteen of the 40 cases (40 percent) excluded by the NTOF criteria should have been classified as agricultural occupational fatalities. There were three reasons why these cases were missed by NTOF:

- In three cases, the "injury at work" category on the death certificate was either marked "no" or left blank.
- In seven cases, the fatality was from the secondary occupation of agriculture and secondary occupations are not listed on the death certificate.
- In six cases, the victim was under the age of 16 and was involved in farm work or was fatally injured from exposure to a worksite hazard.

Of the agricultural occupational cases NTOF did identify, three should *not* have been included. This means 19 of 66 cases were misclassified. However, because 16 cases were undercounted and three cases were overcounted, the net error was an undercount of 13 (20 percent) agricultural occupational fatalities.

The 27 cases that were excluded by our alternative criteria and supplemental data were examined to determine the specific reasons for their exclusion. These cases fell into four groups (Table 2) and are clearly non-agricultural and/or non-occupational.

Discussion

Our research suggests that there may be nearly a 30 percent error in the NTOF method for designating agricultural occupational fatalities which resulted in a 20 percent undercount of these fatalities. This error is due to problems of incomplete and inaccurate information on the death certificates, NTOF's limiting criteria for inclusion as an agricultural occupational fatality case, and the use of "usual industry" as a proxy for work engaged in at the time of the fatality.

Adjusting the NIOSH estimate (18.36 per 100,000 workers) upwards by 20 percent still leaves their estimate well short of the National Safety Council estimate (46 per 100,000 workers). There are at least two possible explanations for the remaining difference. One reason may be the different denominators each used. NIOSH used Census Bureau data for their denominator,[2] while the NSC used Bureau of Labor Statistics data.[1]

A second reason is that the NSC estimate may include some cases similar to the 27 cases that were excluded by our alternative criteria and supplemental data (we found 15 cases that involved non-occupational work). Death certificates do not differentiate

TABLE 2—Supplemental Data Group Identification of the 27 Non-agricultural Occupational Fatalities

	Number
Non-Occupational Work	15
Recreational	4
Underlying Medical Condition	5
Operator/Passenger in a Licensed Motor Vehicle*	3
Total	27

*A fatality where the victim was an operator or passenger in a licensed motor vehicle and was involved in a collision with agricultural machinery, livestock, or other agricultural hazard. A public roadway accident is the primary example.

between occupational and non-occupational work, but unless supplemental data are collected, it is difficult to differentiate these fatalities. Differentiating fatal work accidents that are both agriculturally and occupationally related from those that are simply work related or that happen to occur on a farm or in a rural area is critical if agriculture, as an occupation, is to be fairly and accurately compared with other occupations.

The supplemental data collected in this study have additional benefits besides helping to more accurately identify agricultural occupational fatalities. This information can also yield information on non-occupational related injury and health problems. The Cooperative Extension, which has responsibility for farm and home safety education, can use this information to guide safety education and injury prevention activities. Yet another benefit is to assist state agencies to appropriately allocate resources for injury prevention.

The agricultural industry's unique workforce and workplace characteristics pose a greater challenge than most other industries for the tracking of its occupational work fatalities. The NTOF system is a good start for enumerating fatal agricultural occupational work cases at the national level and it is replicable for a single industry at the state level. This allows those interested in single industry state level data to conduct follow-up studies such as we have reported. These state studies can be used as a basis for state statistics, and for adjusting particular industry totals at the national level.

This study should be replicated in several states to better quantify errors of both the NTOF system for agriculture, and non-occupational fatal injuries in agriculture.

ACKNOWLEDGMENTS

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Use of OSHA Inspections Data for Fatal Occupational Injury Surveillance in New Jersey

MARTHA STANBURY, MSPH, AND MARCIA GOLDOFT, MD, MPH

Abstract: Occupational Safety and Health Administration (OSHA) computerized inspections data, death certificates, and medical examiner records identified 204 fatal occupational injuries in New Jersey, 1984–85. OSHA computerized data uniquely identified seven cases. They did not identify 35 fatalities under OSHA's jurisdiction, of which 24 were investigated by OSHA but not recorded, four were not considered work-related, and seven were not known to OSHA. Eighty-seven were outside OSHA's jurisdiction; 28 were among the self-employed who are not under the health and safety protection of any governmental agency. (*Am J Public Health* 1990; 80:200–202.)

Introduction

Surveillance data for fatal occupational injuries have been compiled in several states.[1–5] These data systems are based largely on death certificates, but are usually supplemented by one or more sources of data such as workers' compensation reports and medical examiner reports. Each source independently captures some fraction of cases. Another potential source of data for surveillance systems is data from occupational fatality investigations carried out by the Occupational Safety and Health Administration (OSHA). Investigation data are maintained in a computer database, OSHA's Integrated Management Information System (IMIS).

We examined the usefulness and completeness of these OSHA inspection data for New Jersey's fatal occupational injury surveillance system. The objectives were to determine: whether OSHA investigation data identified cases not captured on death certificates or medical examiners' records; and whether OSHA investigation data were available for all work-related deaths within OSHA jurisdiction that were identified from other data sources in the surveillance system.

Methods

Unintentional occupational fatalities were ascertained for calendar years 1984–85. A fatal occupational injury was defined as a death resulting from an unintentional workplace injury. New Jersey cases were identified from the New Jersey computerized death certificate file by a code that indicated unintentional injury at work and from medical examiner records by manually sorting through reports in the state medical examiner's office. Cases from each source were then merged into one computerized file by matching on the name of deceased.

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The New Jersey data were compared to OSHA fatality investigation data identified initially from the OSHA IMIS file. The OSHA IMIS file is intended to contain reports of all workplace investigations conducted by OSHA area office inspectors. Name of deceased, name of employer, and other investigative information are included in each record. Names of deceased in the IMIS file were matched with names from the merged death certificate/medical examiner file.

Next, a list of decedents was compiled from cases in the death certificate/medical examiner file for whom a record was not matched in the IMIS file, but whose workplaces appeared to be under OSHA jurisdiction. Jurisdiction was determined by reviewing information about each decedent's workplace available on the death certificate and medical examiner report. Employers in the public sector are not under OSHA jurisdiction. Almost all employers in the private sector are covered by OSHA, except where other federal agencies including the US Department of Transportation and the Federal Aviation Administration have jurisdiction.

The list of decedents' workplaces potentially under OSHA jurisdiction was sent to the OSHA regional office to determine whether these employers were, in fact, under OSHA jurisdiction. If so, the regional office was asked to determine whether an investigation was carried out that was not recorded in the IMIS computer file.

Results

OSHA IMIS data listed 77 fatalities for 1984–85. Sixty-three (82 percent) of these matched with cases in the medical examiner/death certificate file. Four of the remaining 14 cases were cardiovascular deaths and therefore did not meet the case definition. No death certificate could be found for three of the 14. This could have been due to name mispelling or to coding errors in the IMIS file. The remaining seven of the 14 met the case definition and were not captured in the New Jersey data sources (Table 1). Death certificates were obtained for these seven cases; coding errors explained why they had not been captured.

Two-hundred and four fatalities were counted by combining the IMIS data with the death certificate/medical examiner file. Table 2 shows OSHA jurisdiction of the 204 cases. Eighty-seven (43 percent) of the fatalities were outside of OSHA jurisdiction. Of the 105 under OSHA jurisdiction, OSHA IMIS identified 70 (67 percent) cases. Twenty-four investigations were carried out by OSHA area offices but were not recorded in the IMIS system. Of the remaining 11 fatalities under OSHA jurisdiction, OSHA claimed that in four cases the deaths were not work-related because the decedents had preexisting medical conditions (e.g., seizure disorder) related to the fatal episode, and therefore an OSHA investigation was not required. Seven cases of the 105 under OSHA jurisdiction were not investigated because OSHA was unaware of the fatality.

TABLE 1—Occupational Fatalities Identified in OSHA IMIS Data: 1984–85

	Number	%
Matches with death certificate/medical examiner file	63	81
False positives (cardiovascular deaths)	4	5
No death certificate found	3	4
True positives not matching death certificates/medical examiner file	7	9
Total cases in IMIS data file	77	100

TABLE 2—OSHA Jurisdiction for 204 Occupational Fatalities Identified in New Jersey 1984–85

Cases	Number (%)
Not under OSHA Jurisdiction	
Transportation	51
Government	8
Self-employed	28
Total not under OSHA jurisdiction	87 (43)
Under OSHA Jurisdiction	
Investigated according to IMIS data	70
Investigated according to OSHA regional office, not in IMIS	24
Not work related, according to OSHA Regional office	4
Not investigated; OSHA regional office unaware of fatality	7
Total under OSHA jurisdiction	105 (51)
Insufficient Data to Determine OSHA Jurisdiction	12 (6)
Total fatalities	204 (100)

Discussion

This study showed that the OSHA IMIS data file identified occupational deaths identified through no other source, but that it did not serve as a sole source of information because it did not capture all work-related deaths. Because IMIS data capture cases independent of other data sources, these data should be included in state-based fatal occupational injury surveillance systems. In addition, these data could be used as another source to estimate the true magnitude of fatal occupational injuries nationwide, following the procedures of Suruda.[6] However, there are several problems in utilizing these data, including the failure of IMIS data to accurately reflect the numbers of fatality investigations completed by OSHA area offices and underascertainment due to limited OSHA jurisdiction. The national OSHA Office of Data Analysis is addressing the first problem.

In a previous report, with incomplete case follow-up by the OSHA regional office, it appeared that as many as 34 percent of cases with employers apparently under OSHA jurisdiction were not investigated.* Other states using data on employers' fatality reports to OSHA had similar findings.[2,5] After exhaustive examination of case files by the OSHA regional office, it has been determined that a smaller proportion (7 percent) of cases under OSHA jurisdiction were not actually investigated by OSHA. Currently, death certificate and medical examiner data in New Jersey are being shared with the OSHA regional office to ensure that all occupational fatalities within OSHA's jurisdiction are investigated.

These data showed that nearly half of these New Jersey occupational deaths were outside of OSHA jurisdiction, of which 28 were among the self-employed, who are not under the health and safety protection of any governmental agency. Investigative data from OSHA have certain details about the circumstances of fatalities that are not in data from other

*Stanbury MJ, Goldoft M, O'Leary K: Data Sources for a Fatal Occupational Injury Surveillance System in New Jersey. Paper presented at the Annual Conference of the Society for Occupational and Environmental Health, April 6–8, 1987.

sources. Such details, which are important for injury prevention programs, are not available on these cases. Legislative changes are needed to address the large number of occupational deaths not under OSHA's jurisdiction.

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Workplace Health Hazards: Analysis of Hotline Calls Over a Six-Year Period

JULIA QUINT, PHD, MARGARET HANDLEY, BA, AND KATE CUMMINGS, MPH

Abstract: Between 1981–1986 a state-based occupational health telephone hotline received more than 8,000 inquiries on over 3,000 hazardous agents. Major caller groups were employees (37%), employers (20%), health care providers, primarily physicians (19%), government agencies (12%), and labor unions (6%). Employees were the fastest growing caller group. Callers inquired about general health hazards of chemicals (65%), the relation of symptoms to work (22%), and risks to pregnancy (13%). (*Am J Public Health* 1990; 80:202–204.)

Introduction

In 1979, the California legislature enacted a right-to-know law with several support resources including the Hazard Evaluation System and Information Service in the Department of Health Services. As described elsewhere,[1] staff (toxicologists, physicians, industrial hygienists) perform a variety of functions, including operation of a publicly accessible telephone inquiry response system. Over 12,000 inquiries involving a cross-section of California workplaces have been received from 1980 through early 1989.

We present and discuss descriptive statistics which characterize inquiries received from 1981 through 1986.

Methods

Inquiry Intake Procedure

Information in the following categories is obtained from callers and recorded on a standard form after assuring confidentiality. Callers' concerns are grouped into one of three areas: effect on pregnancy; relation of symptoms to work; general health hazard information. Only one concern is recorded as primary; the order of priority (highest to lowest) is pregnancy, symptoms, general information. Callers are categorized as follows:

- *Employees:* persons calling on their own behalf regarding exposures in their workplace, self-employed individuals, and friends and relatives who call on behalf of employees.
- *Employers:* supervisors and managers, company health and safety and medical personnel and industrial hygenists.
- *Health care providers:* physicians, nurses, genetics counselors, poison control center staff and paramedics.
- *Governmental representatives:* California Occupational Safety and Health Administration (OSHA) personnel, city, county, state and federal personnel.
- *Others:* attorneys, news media representatives, volunteer organizations, educational institutions, etc.

Callers' characteristics and primary concerns were tallied from 1981 through 1986. Primary concerns by caller group were tallied from 1986 logs. Data on employee industries, occupations and agents and repeat callers were tallied from the 4th quarter, 1986. Industry data were available for 466 of 562 total inquiries.

Results

During the first four years of full service, there was a modest increase in the annual volume but in 1985 and 1986, the number of inquiries increased by 49 percent and 43 percent, respectively (Figure 1). The average number of inquiries during 1985 and 1986 (2,067) was approximately double the average (1,038) during 1981–84. The sudden increase in inquiry volume appears to be due to increased awareness regarding the potential health effects of chemical exposures at work.

In 1986, of the 365 employer inquiries received, 40 percent were from managers and supervisors, 29 percent were from health and safety personnel (other than industrial hygienists), and 24 percent were from industrial hygienists.

Of the health care providers who called in 1986, 67 percent were physicians (70 percent from private practices). Nurses (12 percent) and genetics counselors (9 percent) were the other major groups who called in 1986.

Approximately one-third of the government agency inquiries in 1986 were from California OSHA. The majority of these callers were industrial hygienists requesting information for worksite inspections.

Analysis of a subset of 1986 callers (562 inquiries received in the fourth quarter of 1986) showed that 39 percent of the employers, 36 percent of the health care providers, 33 percent

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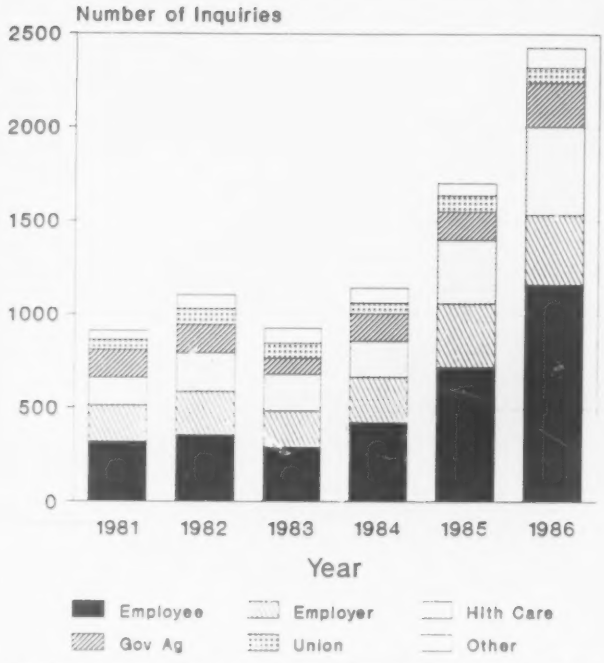

FIGURE 1—Number of Telephone Inquiry Response System Inquiries by Caller Group, 1981–86 (Caller groups defined under Methods)

of the union representatives, and 26 percent of the callers from government agencies were repeat callers. By comparison, only 2 percent of the employees had used the hotline previously.

Based on 466 inquiries from all callers in the fourth quarter of 1986, 48 percent concerned employment in service industries, 28 percent in manufacturing industries, and 8 percent in construction industries. The remaining inquiries concerned employment in transportation, communication, electric, gas, sanitary services (6 percent), agriculture (4 percent), public administration (4 percent), and retail (2 percent) industries. As a point of reference, the service industry was about 24 percent and the manufacturing industry about 18 percent of all non-agricultural California employees in 1986.[2] The types of industries included under services and manufacturing, along with typical occupations and agents of concern, are described in Tables 1 and 2, respectively.

Each year, multiple (10 or more) inquiries are received on 40 to 50 agents. The top 40 agents for which consultations were provided in 1986 are listed in Table 3.

Most caller concerns fall into three major categories. Average percentages of total inquiries for each category from 1981 through 1986 were 65 percent for general health hazard information, 22 percent symptom-related, and 13 percent pregnancy outcome.

The number of pregnancy-related inquiries received between 1981 and 1986 varied greatly. From 1981 through 1983 an average of 56 inquiries were received per year compared to an average of 375 inquiries per year from 1984 through 1986. A complete analysis of the pregnancy inquiries will be published in a separate report.[3]

Further analysis of 584 symptom-related inquiries received in 1986 showed that most were from employees (51 percent) and health care providers (26 percent), mainly physicians. Small percentages of symptom inquiries were from employers (8 percent) and government agencies (7 percent). California OSHA industrial hygienists inquiring about symptomatic employees encountered during worksite inspections represented about 60 percent of the symptom-related inquiries from government agencies.

Discussion

The preceding statistics clearly demonstrate that there is an increasing demand for acute and chronic health effects

TABLE 1—Service Industries,[a] Typical Occupations and Agents Inquired about during 4th Quarter, 1986

Industry	Occupation	Agents
Business (N = 86)[b,c]	Office workers	Video display terminals (VDTs), carbonless copy paper, photo copier chemicals, paint, carpet glues, indoor air contaminants, pesticides, asbestos
	Indoor pesticide applicators	organophosphate pesticides
	Photographers, photo lab technicians	amines, formaldehyde, hydroquinone
	Custodians	alkalis, alcohols, cleaners containing glycol ethers
Personal (N = 34)	Cosmetologists	hair dyes
	Manicurists	acrylic nail products (acrylates)
	Dry cleaners	perchloroethylene
	Laundry workers	alkalis (various products)
Health (N = 32)	Dental lab technicians	mercury, epoxy resins
	Histology, pathology lab technicians	formaldehyde, xylene, alcohols
	Hospital lab technicians	biological hazards, radiation
	Dental assistants	nitrous oxide, methyl methacrylate, mercury, formaldehyde
	Nurses	antineoplastic drugs, ethylene oxide, organic solvents
	Anesthesiologists	nitrous oxide, anesthetic waste gases
Educational (N = 18)	Teachers	PCBs, carpet glues, indoor air contaminants, photocopier chemicals, asbestos, fluorescent lights
	Librarians	asbestos
Miscellaneous (N = 18)	Artists	lead, dyes, pigments
Miscellaneous Repair (N = 16)	Welders	welding fumes, metals
	Furniture refinishers	methanol, methyl ethyl ketone
Auto Repair (N = 9)	Mechanics	asbestos
	Car painters and strippers	methylene chloride, toluene, xylene, phenols, isocyanates (specialty paints)

[a]Coded using a combination of industry, occupation, and narrative information.
[b]N = Total inquiries.
[c]Total Service Industry Inquiries = 222. Industries with less than nine inquiries are not shown.

TABLE 2—Manufacturing Industries,[a] Typical Occupations and Agents Inquired about during 4th Quarter, 1986

Industry	Occupations	Agents
Electrical Machinery Equipment and Supplies (N = 29)[b,c]	Assemblers, Parts cleaners, Wafer fabricators/processors	soldering fumes, organic solvents, epoxy resins, methyl ethyl ketone, glycol ethers, arsine, fluorides
Transportation Equipment (N = 14)	Boat builders	plastics, fiberglass, ketones
	Ship builders	epoxy resins
Chemicals and Allied Products (N = 12)	Chemical workers	acrylamide, metals, sulfur compounds
Printing (N = 11)	Printers	methylene chloride, perchloroethylene, glycol ethers
	Silk screener	organic solvents, acids
Stone, Glass, Concrete Products (N = 11)	Glass factory employees	organotin, silica, kerosene
Fabricated Metal Products (N = 10)	Machinists	glycol ethers, organic solvents
	Welders	welding fumes
	Solderers	solder fumes, fluorocarbons
Machinery (Non-Electrical) (N = 7)	Machinists	lead, thorium, cadmium
Food and Kindred Products (N = 7)	Food processors	chlorine, phosgene, sulfur dioxide
	Winery employees	carbon dioxide, nitrogen compounds
Rubber and Misc. Plastic Products (N = 6)	Plastics/Teflon industry employees	organic solvents, polymers

[a]Coded using a combination of industry, occupation and narrative information
[b]N = Total inquiries
[c]Total Manufacturing Industry Inquiries = 130. Industries with less than six inquiries are not shown.

TABLE 3—Agents Most Frequently Inquired about in 1986

Agent	Number of Inquiries[a]
glycol ethers	119
methylene chloride	108
trichloroethane (1,1,1)	89
lead and lead salts	82
acetone	76
asbestos	74
formaldehyde	73
fluorocarbons	71
acrylates (including acrylic nails)	67
toluene	64
xylene	61
methyl ethyl ketone	57
solvents, not otherwise specified	54
isopropanol	49
polychlorinated biphenyls	48
ammonia	43
paints	43
video display terminals (VDTs)	41
malathion	39
ethylene oxide	39
methanol	38
isocyanates	37
mercury (organic, metallic, inorganic)	35
silica and silicates	33
chromium and chromium salts	33
sulfuric acid	32
perchloroethylene	32
hydrochloric acid	31
arsenic and arsenic salts	29
epoxies	27
chlorpyrifos	26
sodium hydroxide	24
monophenol	21
trichloroethylene	20
fluorescent light fixtures	19
chlorine	18
diazinon	18
nitric acid	17
benzene	17
carbon monoxide	17

[a]Number of times agent was inquired about. May not represent distinct consultations since one consultation often involved multiple agents.

information for workplace exposures. Provision of this information is the core of the Hazard Communication Standard.

Eighty percent of all employers calling in 1986 wanted general health hazard information (such as acute and chronic health effects and industrial hygiene information), suggesting that this group needs further assistance to meet fully the challenge of its responsibility.

Of the various callers, employees are the largest and fastest growing subset. In 1986, 38 percent of all employees were primarily concerned with general health hazard information while 36 percent called about pregnancy concerns and 26 percent called because they had symptoms. Calls from health care providers closely paralleled this distribution. Both employee and health care provider symptom calls focused largely on acute effects and required, for the most part, information and industrial hygiene interventions to reduce exposures.

The telephone hotline is sometimes a limited forum for disseminating useful information. Specifically, some callers need to be educated about basic principles of toxicology before their complex questions (e.g., what is the cancer risk?) can be answered.

The purpose of hotlines is to provide ready access to information rather than education. Perhaps the service would have broader impact if it targeted those individuals who can use and disseminate the technical information provided. Callers who would be potential targets for a technical assistance hotline (health care providers, government agencies, union representatives, and employers) are by virtue of their jobs in contact with many employees. Thus, the information provided to them may benefit many employees. The high percentage of repeat callers among these potential target groups indicates that the hotline, to some extent, has already established an identity as a technical assistance service.

Since hotlines do not necessarily present the best forum for some types of callers, we also publish and disseminate chemical fact sheets and other educational material.

ACKNOWLEDGMENTS

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Incentives and Competition in a Worksite Smoking Cessation Intervention

LEONARD A. JASON, PHD, SHYAMALANITA JAYARAJ, CARYN C. BLITZ, MARK H. MICHAELS, AND LORI E. KLETT

Abstract: We evaluated a worksite smoking cessation program that employed multicomponents including support groups, incentives, and competition. The combination of incentives and other components increased participation rates to over 80%. Forty-two percent of smokers were abstinent at six months, compared to 13 percent of a control group (difference 29 percent, 95% CI 9, 49). (*Am J Public Health* 1990; 80:205–206.)

Introduction

Klesges, Cigrang, and Glasgow[1] and Klesges and Cigrang[2] reviewed seven controlled worksite programs having incentives and/or competition. The average post-test cessation rate was 44.8 percent, and the follow-up cessation rate was 45.9% (if one study by Stachnik and Stoffeimayr[3] with an exceptionally high rate is excluded, the average 13-month follow-up rate was 26.1 percent).

In January of 1988, we began pilot work with a worksite incentive system, that built on our previous work.[4,a] We report the results below.

Methods

The intervention was composed of two parts: a three week quitting program, and six months of follow-up meetings. The two group leaders of the two groups were employees of the Experimental (intervention) company, who were provided training and consultation. One of the two groups met on company time in the late afternoon, the second group met during lunch. For the first three weeks, there were two meetings each week. For the next two weeks, there were weekly meetings. For the next four weeks, there were meetings every other week. For the last four months, there were group meetings on a monthly basis. Each meeting lasted from 40 to 50 minutes.

Incentives

Participants also could earn $10 for attending each meeting regardless of their smoking status. Following the end of the initial three-week program, participants could also earn $1 each day that they were abstinent up to $180 (six months), and for every 30 continuous days of abstinence an extra $30. At the end of the program, each participant who had been abstinent for at least the last two months was eligible to participate in a lottery, with the company contributing $50 for each person who quit in the program. (The company changed this policy at the end of the program by actually putting in $50 for each person who signed up for the smoking cessation program.)

Participants had the opportunity to compete in a team effort for additional incentives. Teams consisted of three members who worked together to quit smoking. The team with the largest number of days abstinent won a cash prize of $300 at the end of the program.

Participants also had the option of selecting a coworker "buddy" to aid in those times of temptation to start smoking again. At the program's end, each buddy was given an opportunity to select an item from a list of gifts.

Participants were not required to join a group. They could still participate and be eligible for other incentives. On a pre-intervention survey, 81 percent of Es indicated they wanted the group program, 4 percent only wanted the self-help book, 2 percent wanted to quit on their own, and 11 percent indicated that they might quit smoking. As for incentives, 91 percent requested payment at meetings, 94 percent payment of $1 a day for quitting, 98 percent for $30 for a month of continuous abstinence, and 93 percent for the lottery. Forty-five percent of Es selected a team and 57 percent selected a buddy. Participants earned an average of $237. Fifty-three percent of Es indicated they would not have tried to quit if the program had not been offered.

Control group smokers were not provided any program or incentives; the high rates of control participation can partially be explained by their interest in obtaining prepoint expired air carbon monoxide (CO) tests. In the CO test, participants first exhale completely, then take a deep breath, hold it for 20 seconds, expire half the air, then breathe into an impermeable plastic bag to provide a sample of carbon monoxide in their breath to be measured.[5]

Results

Pretest Comparability

Two companies agreed to participate in our worksite smoking cessation study. There were approximately 300 employees at the Experimental company and 260 at the Control company. Forty-two of the 52 Control company smokers and 53 of the 63 smokers in the experimental company agreed to be involved in this study.

The two populations were rather similar (Table 1). Experimentals had smoked longer than controls and had higher status jobs.

Program Impact

The experimental manipulation of support, incentives, and competition had a substantial effect on abstinence rates at the immediate post-test, six-month end-of-group meetings, and at the six-month follow-up (Table 2). Experimentals also had higher continuous abstinence rates and overall worksite quit rates (i.e., the percentage of all smokers, both participants and non-participants).

Most of the experimentals selected incentives and the support group, but several preferred to accept other incentives unrelated to group attendance. For two who only selected the self-help book plus incentives, one quit at the post-test and six-month end-of-group meeting, and both had quit by the 12-month follow-up. One who decided to quit on his own and also accept incentives was successful at the three

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[a]Jason LA, Lesowitz T, Michaels M, Blitz C, Victors L, Dean L, Kimball P: A worksite smoking cessation intervention involving the media and incentives. 1989. Manuscript under review. DePaul University, Chicago.

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TABLE 1—Demographic Characteristics and Preintervention Measures on Experimental and Control Conditions

	E	C	Difference (95% CI)
Gender			
% Male	20	22	2% (−16,20)
% Female	80	78	2% (−16,20)
Race			
% Black	8	11	3% (−9,15)
% White	92	89	3% (−9,15)
Occupation			
% Labor/Craft	37	42	5% (−17,27)
% Service	12	33	21% (3,39)
% Prof/Managerial	51	25	25% (4,46)
Education[a]	4.4	3.9	.5 (−0,1)
Age (mean years)	36.8	33.3	3.5 (−4,7.4)
Mean years smoking	19.3	14.7	4.6 (.9,8.3)
Preintervention smoking			
Number of cigarettes per day	24.6	20.1	4.5 (−.8,9.8)
Tar (mg/cig)[b]	12.1	11.3	.8 (−1.3,2.9)
Nicotine (mg/cig)[b]	.81	.77	.04 (−.08,.16)
Carbon Monoxide (mg/cig)[b]	11.9	10.9	1.0 (−.8,2.8)
% Tried to quit before	82	67	15% (−5,35)
Mean number of prior quit attempts	3.4	3.1	.3 (−1.3,1.9)
% Previously stopped smoking 6 months or more	43	39	4% (−18,26)
Mean number of times stopped smoking 6 months or more	1.8	1.8	.0 (−1.2,1.2)

[a]A 7-point scale was used (Elementary School = 1, Professional School = 7)
[b]The participants told interviewers their brand of cigarette and these brands were checked with the January 1985 Federal Trade Commission Report to obtain tar, nicotine, and carbon monoxide levels.
E = Experimental
C = Control

TABLE 2—Immediate Post-test, 6-Month, and 12-Month Measures on Abstinence, Continuous Abstinence, and Worksite Smoking Reductions

	E	C	Difference (95% CI)
Post-Test (95)*	(53)	(42)	
% Abstinence	49	9	40% (21,59)
% Worksite Smoking Reductions	41	8	33% (17,49)
6-Month (92)*	(53)	(39)	
% Abstinence	42	13	29% (9,49)
% Continuous abstinence	34	5	29% (13,45)
% Worksite Smoking Reductions	35	10	25% (10,40)
12-Month (84)*[a]	(47)	(37)	
% Abstinence	36	16	20% (1,39)
% Continuous abstinence	21	5	16% (2,30)
% Worksite Smoking Reductions	32	13	19% (3,35)

*Total numbers in groups
[a]Of the 6 Es who left the company, 3 left abstinent; whereas more of the 5 Cs who left the company were abstinent.

time points. Of the six experimentals who indicated maybe they would quit but would also accept incentives, only one quit at the immediate post-test, relapsed at the end of six-month group meetings, and had quit again at the six month follow-up.

Biochemical Verification

CO confirmations (ppm ≤ 9) were conducted at both experimental and control settings at the post point and end of the six-month meeting period. There was an 85 percent accuracy rate with self-reports. When discrepancies occurred, there are a number of explanations that could have explained the findings (e.g., exposure to second hand smoke, poor ventilation at home or in the car).

Discussion

The present study indicates that incentives and competition can be used effectively to increase participation in worksite smoking cessation programs. Overall worksite reductions were high because a large percentage of smokers participated in the program. It should be noted that worksite policies that allowed smoking at designated areas were introduced in both settings prior to the start of the intervention. Some of the effectiveness of the present program might be due to the smoking restriction policies at the experimental company.

One of the advantages of the present study was that experimentals could select those parts of the program that most appealed to them. Although most chose the groups and incentives, several of those who quit selected other materials and/or incentives. This suggests that different individuals might prefer different types of programs.

There are some limitations to the present study. There were several prepoint differences between the experimental and control companies, and these differences might have played a role in the outcomes. In addition, only two companies were used, and thus this research can only be considered a case study until it is replicated with larger samples.

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Retirement and Primary Cardiac Arrest in Males

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Abstract: We investigated the association between retirement and primary cardiac arrest (PCA) in 126 male cases and controls, 25–75 years of age, without prior heart disease or comorbidity. After adjustment for age alone, retirement was not associated with an increased risk of PCA, (OR = 1.1; 95% confidence intervals = 0.5, 2.4). This lack of association was not uniform across age strata, however. In 10 of 19 discordant pairs 60 or more years of age, the control subject had been retired; in all seven discordant pairs under 60, the case had been retired (lower 95% CI of the relative risk = 1.9). (*Am J Public Health* 1989; 79:207–208.)

Introduction

Clinicians have long suspected that retirement might increase the risk of coronary heart disease (CHD).[1] Retirement represents one of several profound exit/loss events, such as widowhood, death of friends or relatives, divorce, and relocation, that become more prevalent with increasing age.[2] The literature on retirement and physical health is fairly small and marked by contradictory findings and important methodologic limitations.[3–5] We analyzed data from a community-based study of potential risk factors for primary cardiac arrest[6] to determine the influence of retirement. Since our study was restricted to persons who were clinically healthy, we were able to minimize potential bias related to the fact that poor health is one determinant of retirement.[7]

Methods

The design of this study has been presented in detail elsewhere.[6] Briefly, from reports by trained paramedics in Seattle and suburban King County, Washington between December 1979 and January 1981, all 1,250 cases of out-of-hospital primary cardiac arrest were identified. Cases were characterized by a sudden pulseless condition without evidence of a non-cardiac condition as the cause of cardiac arrest.

Because we required detailed information regarding the prior experience of each subject, we restricted the study population to married residents of King County, 25–75 years of age (779 cases). In addition, based on the emergency medical service report or a brief telephone screening interview with the spouses of potential cases, persons with the following conditions were excluded: a history of prior clinically recognized heart disease (492 cases), such as angina pectoris, myocardial infarction, coronary artery bypass surgery and graft, congestive heart failure, arrhythmias, cardiomyopathy, congenital or valvular heart disease; or comorbidity that "limited their usual activity" (103 cases), such as cancer, chronic lung, renal or liver disease. Thus, there were 184 cases eligible for inclusion in this study; they represented persons who appeared and acted as if they were "healthy" until the time of their primary cardiac arrest.

After each case was identified, a control subject matched for age (within seven years), sex, marital status, and urban or suburban residence was selected from the community using random digit dialing.[8] Spouses of potential controls were queried by telephone in a fashion similar to those of cases; potential controls with a history of prior clinically recognized heart disease or comorbidity were excluded. Of eligible subjects identified, the spouses of 89 percent of the cases and 85 percent of the controls complied with a more extensive in-home interview. Thus, there were 163 cases and 163 controls in our study population; 18 percent of subjects were female, 94 percent were White, and the mean age was 58 years.

The home interview verified the absence of prior clinically recognized heart disease or comorbidity and covered factors such as history of hypertension treatment, current cigarette smoking, habitual vigorous leisure-time activity, history of diabetes mellitus, and alcohol consumption. We also asked whether the subject was currently employed and, if not, whether the subject was retired and formerly employed, not working currently but temporarily laid off and seeking employment, or never employed. Information regarding the specific age of retirement and reasons for retirement was not obtained. These investigations were completed in 1981.

We restricted this analysis to the 127 male case-control pairs in which both members were either employed or retired. Among the six male case-control pairs that were excluded, four of the cases and two of the controls were temporarily laid off and seeking employment. One additional pair was excluded because of missing data related to employment status.

An unadjusted matched-pairs analysis and a conditional multiple logistic regression analysis for matched case-control data were used in this study.[9] Factors evaluated simultaneously using the matched-pair multiple logistic model included history of hypertension treatment, current cigarette smoking, habitual vigorous activity, and alcohol consumption. We included age as a continuous variable to control for residual confounding related to age. To explore possible differences between early and conventional retirement (as represented by age at interview), we used a dichotomous variable for age (<60 years, >60 years).

Results

Cases were more likely to be retired compared to controls (Table 1); comparing retired men with currently employed, the estimated relative risk was 1.7 (95% confidence intervals = 0.8, 3.4). However, the estimate of the relative risk was 1.1 either after adjustment for age alone (95% CI = 0.5, 2.4) or after simultaneous adjustment for age, history of hypertension treatment, current cigarette smoking, habitual physical activity, and alcohol consumption (95% CI = 0.5, 2.8).

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TABLE 1—Distribution of Primary Cardiac Arrest Case-Control Pairs According to Retirement Status, King County, Washington, December 1979–January 1981

Retirement by Control	Retirement by Case	
	Yes	No
Yes	27	12
No	20	68

Among case-control pairs less than 60 years of age, cases were more likely to be retired compared to controls (Table 2); in all seven discordant pairs it was the case who had been retired (relative risk = infinity, lower 95% CI = 1.9). Of note, among subjects less than age 60, the prevalence of cigarette smoking, obesity, diabetes, family history of heart disease, and alcohol consumption was similar among retired and employed men; however, prior hypertension treatment was more common among retired (62 percent) than employed (25 percent) men. There was no relation between retirement and the risk of primary cardiac arrest among men greater than or equal to 60 years of age, as reflected by an estimated relative risk of 0.9 (95% CI = 0.3, 2.7).

Discussion

There are several limitations of our analysis: we did not obtain specific information on the reasons for retirement; we do not know the extent to which retirement was involuntary. However, our study was restricted to persons without clinically recognized heart disease or comorbidity that limited their usual activity; only 8 percent of subjects had more than three restricted bed days over the prior six months and less than 1 percent had more than six days. For these reasons, we consider it unlikely that physical disability was a major determinant of retirement in our study. Since we did not obtain information on the time since retirement began, we were not able to determine if this factor influenced the risk related to retirement. Finally, because of the small number of retired men in our analysis, we were not able to fully explore potential confounding related to other risk factors for the age-specific estimates of risk related to retirement.

We chose a cut-point of age 60 for early retirement because we considered retirement prior to that age unambiguously early. For a later cut-point, such as age 62, the magnitude of the increase in risk related to early retirement decreased (relative risk = 2.8, 95% confidence intervals = 0.9, 8.2) but the direction of the interaction between age and retirement on the risk of primary cardiac arrest remained the same. Nevertheless, for the reasons noted above, our finding of an increase in the risk of primary cardiac arrest among younger but not older men should be viewed as preliminary. Because of the magnitude of the association of retirement with primary cardiac arrest that we observed in relatively young men, the benefits and risks of early retirement deserve further study.

TABLE 2—Distribution of Primary Cardiac Arrest Case-Control Pairs* According to Age and Retirement Status, King County, Washington, December 1979–January 1981

Age	Retirement by Control	Retirement by case	
		Yes	No
<60	Yes	0	0
	No	7	57
>60	Yes	27	10
	No	9	4

*10 case control pairs where both subjects were not in the same age stratum were excluded from this table.

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Seroconversion to Human Immunodeficiency Virus in Prison Inmates

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Abstract: We evaluated the prevalence and incidence of human immunodeficiency virus (HIV) infection in 3,837 inmates of a state prison system. Ninety-two (2.4 percent) were HIV-seropositive. The highest proportion of HIV-seropositive inmates was found among Blacks (5.4 percent), females, and those 30 years of age or older. HIV-seropositivity of entering inmates was also 2.4 percent and was unchanged over the three years of the study. Seroconversion occurred in two inmates while in prison, a rate of one conversion per 604 person-years, but HIV infection could have occurred before entry. Seroconversion to HIV was rare in inmates in this correctional facility (*Am J Public Health* 1990; 80:209–210.)

Introduction

Acquired immunodeficiency syndrome (AIDS) was first reported in prison inmates in 1983.[1,2] As of October 1, 1988, 3,136 AIDS cases had been reported in a survey of state and federal correctional facilities.[3] Since inmates frequently report intravenous (IV) drug use both before and during incarceration,[4,5] human immunodeficiency virus (HIV) infection in this population is not surprising. Studies of persons entering prison in the United States have shown rates of HIV seropositivity ranging from 0 to 17 percent.[3,6]

Several state prison systems, including the Nevada Department of Prisons, routinely screen incoming inmates for antibody to HIV. In Nevada, this is mandated by law. In August 1987, the Nevada Department of Prisons also began testing all inmates for antibody to HIV at the time of their release. To estimate how frequently HIV seroconversion occurs after inmates are admitted to a correctional facility and to assess whether seroconversion is the result of HIV exposure while in prison, we surveyed the results of HIV antibody testing of all inmates released from the Nevada Department of Prisons between August 1987 and July 1988.

Methods

The Nevada Department of Prisons operates 14 correctional facilities: one maximum and three medium security facilities for men, one women's facility, seven minimum security facilities, and two transitional centers. All persons admitted to the system beginning August 1985 received HIV antibody testing. In addition, all inmates in the system were tested in a prevalence survey from September through December 1985. Post-test counseling is offered to all inmates tested.

HIV antibody was determined by enzyme immunoassay (EIA; Electronucleonics Inc., Columbia MD, and Abbott Laboratories, Chicago, IL). Repeatedly EIA positive specimens were tested by the Western blot method at the University of California, Davis. Presence of an envelope band and a *gag* or *pol* band were required for Western blot confirmation.

Seropositivity of groups was compared by chi-square analysis. Confidence intervals were calculated using the Taylor series method.

Results

In September through December 1985, 92 (2.4 percent) of the 3,837 inmates* tested were HIV-seropositive. The highest proportion of seropositive inmates was found among Blacks (5.4 percent), females, and those 30 or more years of age (Table 1).

HIV seropositivity in incoming inmates from August 1985 to July 1988 was 124/5,216 (2.4 percent). In the first year of the study, the rate was 43/1,623 (2.6 percent); in year two, it was 43/1,758 (2.5 percent); and in year three, it was 38/1,835 (2.1 percent). No variation in age or race distribution of the affected inmates was seen over time (data not shown).

A total of 1,105 inmates had initial samples obtained on entry or in the prevalence survey and again on leaving the prison system. Of these 1,105 inmates, 1,066 (96.5 percent) were seronegative on initial testing and at exit; 36 (3.3 percent) were seropositive on both tests, two seroconverted, and one inmate was a possible seroconverter (Table 2). The seroconversion rate was therefore 2/1,069 or 0.19 percent of susceptibles. The 1,069 inmates whose initial test was negative were followed a total of 1,207 person-years; the seroconversion rate could therefore also be expressed as one conversion per 604 inmate-years.

To determine whether transmission had occurred within the prison system, we also assessed the HIV serostatus of all inmates in the system during the study period who had

*Racial-ethnic distribution of prison population: White 58.1 percent; Black, 33.3 percent; Hispanic 6.3 percent; Other 2.3 percent.

TABLE 1—Prevalence of Antibody to Human Immunodeficiency Virus (HIV) in Nevada State Prison System Inmates, December 1985

Demographic Features	HIV Seropositive/ No. Tested (%)	Odds Ratio	(95% CI)**
Race			
White	21/2231 (0.9)		
All Other*	71/1606 (4.4)	4.87	(2.98, 7.96)
Sex			
Male	74/3594 (2.1)		
Female	18/243 (7.4)	3.81	(2.23, 6.48)
Age (years)			
Less than 30	32/1792 (1.8)		
30+	60/2045 (2.9)	1.66	(1.08, 2.57)

*Race	Number	(% Seropositive)
Black	1279	(5.4)
Hispanic	243	(0.8)
Native American	51	(0)
Other	33	(0)

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TABLE 2—Seroconversion to HIV among Nevada State Prison System Inmates

Inmate Case	Age (years)	Sex	Race	Time in Custody before Testing	Risk Activity for HIV	Entry Test, Result	Exit Test Result
1	32	M	Black	130 days	Intravenous Drugs	EIA −	EIA +, WB +
2	35	M	Black	20 days	Intravenous Drugs, homosexuality	EIA −	EIA +, WB +
3	22	M	White	7 days	None known	EIA −	EIA +, WB indeterminate (p24 only)

entered on or before December 31, 1981; 313 such inmates were identified. Fifteen (4.8 percent) of these were HIV seropositive; however, all 15 had had their incarceration interrupted by parole at some time since 1982, and thus might have acquired HIV infection outside the prison.

Discussion

IV drug use has been cited as the major risk behavior for HIV seropositivity among prison inmates.[2,3] Our observation of a higher rate of HIV seropositivity in non-White inmates may therefore be a reflection of higher rates of IV drug use in non-White inmates or of higher rates of HIV infection in non-White IV drug users,[7] or both. The increased rate of HIV infection in incarcerated women in our survey may reflect an increased percentage of incarcerated women who are either IV drug users or sex partners of drug users. In Nevada, incarceration for a drug-related offense is more common among non-White than White inmates (18 percent versus 11 percent) and among female than male inmates (24 percent versus 13 percent); we therefore suspect that increased drug use in these groups accounts for the increased HIV seropositivity (Nevada Department of Prisons, unpublished data). Increased HIV seropositivity in older inmates may be related to duration of high-risk behavior for acquisition of HIV.

The rate of HIV seropositivity in incoming inmates (2.4 percent) remained stable over the three years of our study. Rates in incoming inmates in Maryland, although higher (7.2 percent), have also remained stable during the past three years.[8] The 2.4 percent seropositivity rate in incoming inmates in Nevada is higher than that reported in 20 of 25 prison systems responding to a recent Department of Justice survey.[3] Thus if HIV seroconversion within prisons such as those reported in the Department of Justice survey were occurring at a substantial rate, evidence of such seroconversion would have been seen in our survey.

Two definite cases of seroconversion to HIV were detected, while a third inmate developed only antibody to the HIV *gag* protein, p24, on Western blot. Reactivity to p24 alone is considered an "indeterminate" result but can be indicative of early seroconversion.[9] Because this inmate denied activity which would have put him at risk for HIV infection and refused retesting after his release, he could not be definitively classified.

The seroconversions could have resulted from exposure to HIV either before or after entering a correctional facility; although inmate 1 (Table 2) was in continuous custody for the four months before his first HIV antibody test, seroconversions occurring more than four months after exposure have been reported.[10] The overall rate of seroconversion (one case per 604 person-years) was even lower than the rate of one per 244 inmate-years reported from a study of inmates in the Maryland State Prison system.[6] However, in that study only a self-selected subset of exiting inmates were tested.

Both IV drug use and homosexual intercourse occur in prisons; in a study of inmates in Tennessee, 26 percent reported intravenous drug use and 17 percent reported homosexual activity while in prison.[5] Presumably, needle sharing and homosexual intercourse are also occurring among inmates in the Nevada Department of Prisons facilities. However, such occurrences are apparently not frequent enough between HIV-infected inmates and susceptibles to result in a high rate of HIV transmission. Because exposures could have occurred before entry into the Nevada State Prison System, the seroconversion rates reported here are maximum estimates of the transmission of HIV in prison inmates. We conclude that transmission of HIV among inmates incarcerated by the Nevada Department of Prisons is rare.

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Suicide Clusters: An Examination of Age-Specific Effects

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Abstract: The age specificity of time-space clusters of suicide was examined using National Center for Health Statistics data for 1978–84. Significant clustering of suicide occurred primarily among teenagers and young adults, with minimal effect beyond 24 years of age. Clustering was two to four times more common among adolescents and young adults than among other age groups. (*Am J Public Health* 1990; 80:211–212.)

Introduction

Approximately 29,000 people killed themselves in 1985 in the United States.* Suicide is rare before the age of 12, increases dramatically during adolescence, and reaches a peak between ages 20 to 24 at which time the suicide rate plateaus until another increase is witnessed among elderly men. Suicide among teenagers and young adults has increased markedly since 1960, moving suicide from the fifth leading cause of death to the second leading cause of death among teenagers in 1985.*

The recent occurrence of several highly publicized clusters of youth suicides has focused attention on the problem of suicide "epidemics"—unusually high numbers of suicides occurring in a small area and brief time period. Such outbreaks have been reported from ancient times through the twentieth century in diverse populations of varied ages,[1,2] but these reports are difficult to interpret without systematic surveillance and reporting of suicide clusters. Moreover, only recently[3] have statistical techniques for the detection of clusters of suicide in time and space been used, although several studies have examined temporal clustering after suicide stories.[1] Few studies have examined temporal fluctuations, in general[4–7] or spatial patterns.[7] Some clustering of suicides occurs by chance alone, even if suicides are occurring at random, so measurement of the rate of "outbreaks" must account for the clustering expected by chance variation.

Our earlier work indicated that clustering of suicides occurred among 15–19 year olds to a greater extent than would be expected by chance alone.[3] The present report examines the whole life span to determine whether suicide clusters are unique to adolescents or represent a more general phenomenon.

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*Mortality Statistics Branch, Division of Vital Statistics, National Center for Health Statistics. Vital Statistics of the United States (Unpublished data for 1985).

Methods

Data Sources

Vital statistics mortality data tapes 1978–84 were obtained from the National Center for Health Statistics (NCHS). Separate analyses were performed for the following age groups: 15–19, 20–24, 25–29, 30–34, 35–44, 45–54, 55–64, and 75+ years.

A suicide was defined as any individual whose cause of death had been listed as codes E950–959 of the Ninth Revision of the International Classification of Diseases (ICD-9). The late effects of a self-inflicted injury (E959) was included in the analyses because the timing of the death, in addition to the initial suicidal act, may influence a subsequent suicide. However, this classification of death only accounted for up to 0.2 percent of the total number of suicides within any age group and, therefore, it is unlikely that it had an impact on the analyses.

Statistical Method

The Knox procedure[8] was used to assess the degree of time-space clustering. Details on alternative procedures and the reasons for selection of the Knox procedure are given elsewhere.[3] The Knox procedure considers all possible pairs of cases and the time and space distances between them. Critical values for time and space to define "closeness" must be specified in advance. The test statistic involves a comparison of the observed number of pairs close in time and space with the expected number, which is the product of the proportion of pairs that are close in time with the number of pairs close in space. The test statistic approximately follows a normal distribution, $Z = (O-E)/\text{variance}$. The variance of the statistic was estimated following the method of David and Barton.[9]

Definition of Cluster

There are no explicit definitions of a "suicide cluster" in the literature. Initial operational definitions and limits for time and space parameters were examined in an exploratory analysis of NCHS data for 15–19 year olds for 1978 and state mortality data for 1978 through 1985, as described elsewhere.[3] Four space units (county and town of residence and occurrence) and five critical time units (3, 7, 14, 30 and 60 days) were examined to define closeness between pairs of cases. Exploratory analyses indicated that county of residence yielded a space unit more sensitive to the definition of a cluster. There was little difference between the five time units in the exploratory examination. The data are reported for 7, 14 and 30 days.

Results

As shown in Table 1, 1,420 close pairs of suicides (same county, 0–7 days) occurred among 15–19 year-olds, whereas 1,339.2 were expected. The 6 percent elevation above chance had an associated p-value of .011. The effect of clustering is primarily observed in the younger age groups (15–19 and 20–24 year olds) at the three critical time units. Very little clustering occurred at older ages, with the exception of the 55–64 year old age group at the seven-day critical time unit.

TABLE 1—Results of Knox Procedure on 1978–1984 NCHS Mortality Detail File

		Window = 7 Days				Window = 14 Days				Window = 30 Days			
		Close-Close Pairs				Close-Close Pairs				Close-Close Pairs			
Age (years)	N	Expected	Observed	O/E	P	Expected	Observed	O/E	P	Expected	Observed	O/E	P
15–19	12135	1339.2	1420	1.060	.011	2756.7	2842	1.041	.047	6000.7	6177	1.029	.009
20–24	25511	6335.5	6523	1.030	.008	13126.1	13481	1.027	.001	28642.6	29602	1.033	.000
25–29	23275	7126.1	7155	1.004	.365	14775.8	14888	1.008	.174	32247.7	32681	1.013	.008
30–34	19534	4805.0	4834	1.006	.338	9991.0	10035	1.004	.328	21794.4	21978	1.008	.106
35–44	29092	9707.5	9822	1.012	.119	20086.4	20271	1.009	.094	43780.9	44066	1.007	.087
45–54	26002	6976.1	7103	1.018	.060	14530.8	14629	1.007	.205	31622.9	31633	1.000	.478
55–64	25624	6399.6	6623	1.035	.002	13277.2	13401	1.009	.136	28928.5	29260	1.011	.024
65–74	19621	3334.8	3395	1.018	.139	6877.7	6976	1.014	.111	15002.1	15247	1.016	.020
75+	14718	2373.0	2367	0.997	.550	4876.8	4888	1.002	.435	10567.0	10554	.998	.551

The effect size was two to four times greater among the adolescents than in other age groups.

Discussion

Suicide clusters appear to occur primarily among teenagers and young adults, although clusters do occur occasionally at other ages. This is consistent with the findings of Phillips and Carstensen[10]: the impact of suicide stories on subsequent suicides was greatest for teenagers. In addition, although there is no formal surveillance system for suicide clusters in the United States, most of the suicide clusters reported to the Centers for Disease Control have primarily involved adolescents, young adults, or both.[1]

The magnitude of the clustering effect is relatively small, even among teenagers and young adults. Estimates of the percentage of teenage suicides that occur in clusters averages between 1 percent and 2 percent with considerable variation by state and year, yielding a range of estimates from less than 1 percent to 13 percent.[3] However, the relative risk for suicide given exposure to the suicide of one or more other persons may be quite great. Studies at the individual level will be needed to define this relative risk. Moreover, although the number of youth suicides that occur in clusters may be relatively small, they represent a class of suicides that may be particularly preventable. Suicide prevention efforts that focus on high-risk populations is a recommended strategy.[11]

The Centers for Disease Control has recently developed recommendations for a community plan for the prevention and containment of suicide clusters.[12] Targeting multiple risk groups in prevention strategies will be necessary to reduce the suicide rate substantially.

ACKNOWLEDGMENTS

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Misclassification of Childhood Homicide on Death Certificates

GARRY D. LAPIDUS, PA-C, MPH, DAVID I. GREGORIO, PHD, MS, AND HOLGER HANSEN, MD, DRPH

Abstract: Suspect classification of homicide deaths of Connecticut residents under 20 years of age was noted for 29 percent of cases examined. Misclassification was attributed to incomplete or erroneous information recorded on the death certificates, rather than errors in the designation of ICD-9 homicide codes. The results have important implications in the interpretation of vital statistics when homicide is listed as the cause of death and underscore the value of record linkage systems. (*Am J Public Health* 1990; 80:213–214.)

Introduction

The causes of death reported on death certificates represent important national, state, and local statistics that are used in epidemiologic research as well as the planning/evaluation of health service delivery.[1,2]

Unfortunately, accuracy of death certificates regarding the cause of death presents problems.[3] In particular, underrecording of childhood homicide is known to be a serious problem.[4] It is thought to occur for a variety of reasons including ascertainment difficulties,[5] reporter unwillingness to consider homicide as a diagnosis,[6] and misclassification of homicides as "accidents" or as "cause of death unknown."[7]

This report addresses a complementary issue: the overrecording of childhood homicides on vital statistics.

Methods

Death certificates for all reported homicides between 1980–85 among Connecticut residents 0–19 years of age (N = 166) were reviewed by the authors. Supplementary information available from the State's Chief Medical Examiner Office on circumstances leading to the death of subjects (i.e., police investigation reports, eyewitness accounts and hospital records, laboratory and autopsy reports) was examined for the 161 deaths which occurred within the state.

The manner of death recorded on the death certificate (unintentional injury, homicide, suicide, or undetermined) was evaluated for consistency with supplementary information obtained from files of the State's chief medical examiners office. Homicide is defined in the ICD-9 CM manual as "injuries inflicted by another persons with intent to injure or kill by any means."[8] This definition is consistent with the legal interpretation of criminal homicide, i.e., "lethal violence (unlawfully) inflicted by a second person (or persons) with the intent to do bodily harm."[9] Non-criminal homicide deaths (i.e., negligence, justifiable homicide),[9] by contrast, would be excluded by a strict application of the ICD definition.

Certificates attributing death to the intentional use of a firearm, stabbing with a sharp instrument, other physical assault, arson and the like, when corroborated with statements by the assailant(s) or other eyewitnesses, and/or information from medical records and police investigations were considered accurate. Typical examples would be: an adolescent's death due to firearm injury where review of the medical examiner's report and police investigation report an argument following alcohol and/or illegal drug use in the company of friends or an acquaintance; a victim under three years of age for which a sworn confession by a parent attributes death to assault by blunt force was considered an appropriate designation of homicide.

Death certificates for which supplementary information was at odds were judged suspect for the purposes of this study. Our decisions were based on whether the police investigation documented criminal intent to injure or kill.

A preliminary review of circumstances leading to the death of cases suggested six causes of death for which an application of the ICD-9-CM homicide definition might be suspect; negligent use of a motor-vehicle, (e.g., motor vehicle/pedestrian collisions, without evidence of intent to injure, but rather hit and run incidents where the legal transgression was leaving the scene of a crash without stopping to give aid), unintended discharge of a firearm, (e.g., play with firearms by children, resulting in the unintended discharge of the weapon), suicide, legal intervention, arson, and drowning (e.g., where intent to harm was not documented).

Results

For 47 of 161 deaths studied (29 percent), we judged the manner of death reported on death certificates not to conform to strict interpretation of the ICD definition of homicide when supplementary information was taken into account. These included: 16 deaths due to arson, 13 motor vehicle pedestrian or pedal cycle collisions, 11 unintentional discharge of a firearm, four legal interventions, two suicides, and one drowning.

Table 1 characterizes those 47 cases. Error (20 percent) was less common regarding cause of death among 15–19 year olds, and most common (65 percent) among 10–14 year olds.

In addition, slight differences were noted by the race and sex of victim. Cause of death was misclassified in 17 of 74 cases among Whites (23 percent) compared to 30 of 87 among others (34 percent). Twenty seven percent of male cases compared to 33 percent of female cases were misclassified.

Discussion

Consistent use of the ICD-9 definition of death was found to be problematic in our investigation. Excluding such deaths from rate calculations would yield a corrected homicide rate of 2.13 per 100,000 persons, a reduction of some 30 percent of the current reported annual homicide rate for this age group.

We recognize the subjective nature of our effort and that additional information on the cases reviewed here (e.g., the legal disposition of criminal charges in such matters) may shed further light on the circumstances of intent to injure.

From the Department of Community Medicine and Health Care, University of Connecticut School of Medicine (all authors) and the Connecticut Coalition to Prevent Childhood Injuries (Lapidus, Hansen). Address reprint requests to Garry D. Lapidus, PA-C, MPH, Department of Community Medicine and Health Care, University of Connecticut Health Center, Farmington, CT 06032. This paper, submitted to the Journal December 23, 1988, was revised and accepted for publication June 23, 1989.

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TABLE 1—Suspect Homicide Deaths by Sex and Age of Victims; Connecticut Residents 0–19 Years, 1980–85

		Male Suspect cases							Female Suspect cases					
Age (years)	All Cases	Hit and run unintended injury	Playing with gun	Legal intervention	Suicide	Drowning	Arson	All Cases	Hit and run unintended injury	Playing with gun	Legal intervention	Suicide	Drowning	Arson
0–4	11		1				1	13	1					2
5–9	7					1	2	6		1				1
10–14	16	3	2	1			2	10	2	2				5
15–19	66	5	3	3	2		1	32	2	2				2
Total	100	8	6	4	2	1	6	61	5	5				10

47 of 161:29%

Furthermore, we recognize that underrecording of childhood homicides (i.e., false-negative cases) likely exceeds the discrepancies noted here. Our analysis focused exclusively on deaths for which the designation of homicide as a cause of death was problematic (i.e., false-positive cases). Attention to the complementary issue (i.e., the designation of criminal homicide deaths as unintentional injury, death due to other causes or cause of death unknown) was beyond our current resources but remains an area requiring further attention.

Our study pointed out several instances of failure to identify the cause of death correctly in circumstances involving the unintended use of firearms (e.g., playing with guns), hit and run motor vehicle collisions, legal intervention by police officers, arson, suicide and drowning. Patterns of misclassification related to the age of victim and mode of injury were detected. However, we were unable to specify systematic biases in the way data were recorded. Misclassification apparently resulted from incomplete or erroneous information on death certificates, rather than errors of ICD-9 homicide coding. Death certificates used for medical examiner cases require listing the manner of death as "accident, homicide, suicide, or undetermined." Presumably, initial homicide determination on the part of the medical examiner when forwarded to the state health department was then "properly" assigned an ICD-9 homicide code.

The importance of maintaining procedures for updating medical examiner files with related information available from police and hospital sources as well as revising death certificates on the basis of supplementary information that may be received is pertinent here. Permanent misclassification of deaths as homicides become more likely as procedures for revising certificates are inconsistent. In several instances we noted incomplete police reports. In others, supplementary information was dated after the certification of death date.

Problems stem, as well, from deficiencies of the ICD-9 classification system which do not allow accurate coding of "hit and run" motor vehicle pedestrian collisions. Roughly one-fourth (27 percent) of misclassified cases (13 of 47) were situations of this type. The coding of such cases as homicide rather than negligent use of a motor vehicle implies criminal intent by drivers. Such certificates provide little information to injury control researchers regarding the true circumstances of the event. Of the 161 cases reviewed only one true vehicular homicide was noted.

ACKNOWLEDGMENTS

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The Incidence of *Haemophilus Influenzae* Meningitis in a Midwestern Metropolitan County

RANDY B. RICE, BS, ANNE D. WALLING, MD, AND KEN J. KALLAIL, PHD

Abstract: The incidence of *H. influenzae* meningitis in children under five years of age was monitored during the years 1983–87 by a retrospective study of records from all hospitals serving a metropolitan county in Kansas. The mean annual incidence rate for the five years studied was 26.0 per 100,000. This compares with a mean of 56.0 per 100,000 measured in the same county during 1979–82. (*Am J Public Health* 1990–80:215–216.)

Introduction

Haemophilus influenzae type b (Hib) is the most frequent cause of bacterial meningitis in the United States,[1] accounting for 48.3 percent of all cases of bacterial meningitis[2] and causing an estimated 12,000 episodes of illness annually. Approximately 85 percent of *H. influenzae* meningitis cases occur in children below the age of five years.[1,2] Annual incidence rates show wide variation from 19 to 69 cases per 100,000 children under five years of age.[3] Of the six antigenic types of *H. influenzae*, type b accounts for 95 percent of the cases of meningitis.[4]

The purpose of this study was to describe the epidemiology of *H. influenzae* meningitis in children under age five in a midwestern community for the period 1983–87 and to compare the results with a similar study in the same community covering 1979–82.[5]

Methods

The study involved children residing in Sedgwick County, Kansas. The estimated total county population grew from 378,000 in 1983 to 394,000 in 1987 (Table 1). The number of children in the county under age five was estimated each year by the Metropolitan Long-Range Planning Department and updated by birth, mortality, and migration rates. In the under five population, the numbers of males and females were calculated to be equal and the racial composition was determined as 82 percent White, 11 percent Black, and 7 percent "other."

Hospital records were identified for all cases of *H. influenzae* meningitis in residents of Sedgwick County during the years 1983–87. The study included all hospitals serving the county population. Noncounty residents admitted to these hospitals were excluded from the study on the basis of zip code information. Cases missed by outflow to other medical centers are unlikely as the closest large population centers are more than 150 miles from Wichita. Examination of city/county health department records did not reveal any cases which could have died before hospital admission. Cases were identified by primary or secondary ICD codes and confirmed by culture and gram stain of cerebrospinal fluid.

Besides age-specific incidence rates, the variables measured included race, sex, and seasonal distribution. Data on *H. influenzae* meningitis in children under five years of age were extracted from the earlier investigation[5] which had studied meningeal infection of all etiologies in all age groups.

Results

Forty-one cases, all under age five, were identified. Forty were serologically type b and one was type d. These children accounted for all cases of *H. influenzae* meningitis in the community during the study period. The largest number of cases occurred between 12 and 17 months of age, followed by children younger than 6 months (Figure 1). Estimates of the population at risk were used to calculate annual incidence rates. As shown in Table 1, these ranged from 21.7 to 37.9 per 100,000 with a mean for the five years of 26.0 per 100,000 person-years.

H. influenzae meningitis occurred in 22 males and 19 females which corresponds to incidence rates of 27.9 per 100,000 for boys and 24.1 for girls during the five years studied. Thirty-six cases occurred in White children and five in Black (race-specific incidence rates were 27.8 for White children and 28.8 for Black). There were no cases in the "other" racial group. The differences by sex and by age were not statistically significant.

During the five years of the study, *H. influenzae* meningitis showed a bi-modal distribution with the highest number of cases occurring in late autumn and in the spring. The disease was least common in the summer months May through September.

Two White males with severe symptoms died within 48 hours of hospital admission during the study period. The case-fatality rate was 4.9 percent. The mean hospital stay for nonfatal cases was 12.2 days. At the time of hospital discharge, sequelae were recorded in 24 percent of hospital records. These sequelae included hearing loss, mental retardation, coma, acquired hydrocephalus, and respiratory impairments.

Comparison with Previous Study

Data from the earlier study[5] were recalculated to provide the incidence of *H. influenzae* meningitis in children under

TABLE 1—Incidence of *H. influenzae* Meningitis in Children below the Age of Five Years in Sedgwick County, Kansas

Year	Estimated Total Population	Population <5 Years of Age	Cases *H. influenzae* Meningitis	Hib Incidence in Children <5 years*
1983	378,000	30,890	7	22.7
1984	381,500	31,168	8	25.7
1985	387,800	31,667	12	37.9
1986	391,000	31,920	7	21.9
1987	396,000	32,239	7	21.7
Total	1,932,300	157,884	41	Mean = 26.0

*per 100,000 person years

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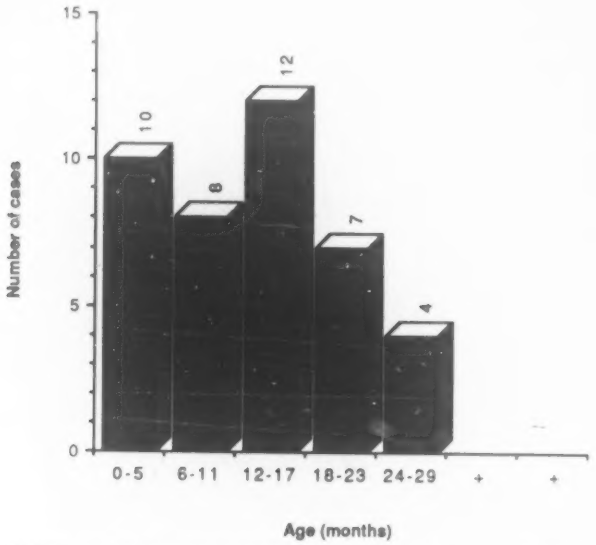

FIGURE 1—The Number of Cases by Age of *H. influenzae* meningitis in Sedgwick County, Kansas during 1983 to 1987

five years of age. The 65 cases observed over the four years (1979–82) corresponded to a mean annual incidence rate of 56 cases per 100,000 children under five, compared to a mean of 26.0 for the five years 1983–87, measured in the present study (95% confidence intervals of difference = 14,36). Methodology was similar for both studies.

Discussion

The assumption that all cases would be admitted to hospital in the county appears valid from our knowledge of clinical practices in the community. Outflow of cases to another city was unlikely as was the possibility of cases being treated without hospital admission. Cases could only have been missed through misdiagnosis and/or miscoding.

The most striking finding is the fall in incidence compared to the previous study. This could be due to several factors such as increased resistance of the population, lowered exposure to *H. influenzae*, decreased virulence of the organism, or the wide variations in annual incidence that occur naturally with this disease.[6] In view of the controversy over vaccination, it is tempting to attribute the fall to vaccination which was introduced to this community in 1986 and is generally offered at 24 months of age. It was not possible to identify data on the proportion of children immunized against *H. influenzae* in the county and information on vaccination was not recorded in the hospital charts. The age of the cases makes widespread immunization unlikely. Immunization could still be responsible for the fall in incidence by reducing cases in older children. This has the dual action of both directly reducing the number of cases and preventing infection being passed to vulnerable younger siblings.

Although the incidence of *H. influenzae* has fallen, the disease remains a potential cause of significant morbidity and mortality. The mortality rate of 4.9 percent confirms that reported by the Federal Centers for Disease Control.[1] One-fourth of all cases had evidence of serious sequelae at discharge and more pathology could be anticipated with long-term follow-up. In addition, the long length of stay represented considerable use of resources. While welcoming the fall in incidence, *H. influenzae* meningitis is a disease which commands respect and continued preventive efforts.

ACKNOWLEDGMENTS

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Trends in Hospitalized Discharge Rates for Head Injury in Maryland, 1979–86

ELLEN J. MACKENZIE, PHD, SHARON L. EDELSTEIN, SCM, AND JAMES P. FLYNN, MD, MPH

Abstract: Hospital discharge data from all acute care hospitals in Maryland were used to examine trends in hospitalized head injury incidence and outcome by severity. From 1979 to 1986, discharge rates increased by 3.4/100,000 per year; the largest percent increase was for more severe injuries. Discharge rates increased the most for adults ages 15–24 and ages 75+ but declined for children ages 0–4. Coinciding with the increase in head injury discharges was a decrease in the hospital case-fatality rate across all severity groups. (*Am J Public Health* 1990:80:217–219.)

Introduction

The public health importance of head injury as a major source of mortality and morbidity is well recognized. Recent studies have reported incidence rates that range from 152 to 200 per 100,000 population depending on case definitions and case ascertainment methodologies.[1–9] Despite differences in the magnitude of these estimates, findings regarding the patterns of incidence by age, race, sex, and severity are highly consistent. There is only one published report, however, in which these patterns have been examined over time (Olmstead County Minnesota, 1935–1974).[1] This paper describes the incidence of hospitalized head injury in Maryland over the eight-year period 1979–86.

Methods

Data Sources

Incidence rates of hospitalized head injury, 1979–86, were computed for the State of Maryland using uniform hospital discharge abstract data maintained by the State's Health Services Cost Review Commission for all 56 acute care, non-federal, hospitals as mandated by law. Excluded from the database are injuries treated on an outpatient basis as well as deaths that occur at the scene, in transport, or in the Emergency Department. Data abstracted include patient demographics, up to five ICD-9CM coded discharge diagnoses, up to three procedures (ICD-9CM coded), total and ICU length of stay, total and component charges, discharge status, and expected principal source of payment. There is a high degree of confidence in the quality of the discharge abstract data, including the diagnostic information, as they are used in paying all hospitals in the State.

To compute discharge rates the total number of hospital discharges (both residents and non-residents) was divided by the State resident population for that year as estimated by the Maryland Department of Health and Mental Hygiene. This approach assumes that the number of state residents treated in out-of-state hospitals roughly equals the number of non-residents treated in-state. All age-adjusted rates are based on direct adjustment using the 1980 census as the reference population.

Definitions

A head injury case was defined as any discharge with a principal or secondary diagnosis of a skull fracture and/or intracranial injury (ICD-9CM codes 800, 801, 803, 804, 850–854). A secondary head injury discharge was included only if the principal diagnosis was a traumatic injury to another body system; complications (ICD 9CM 958) and late effects (ICD 9CM 905–909) of injuries were excluded.

Injury severity was determined through the use of the software ICDMAP which maps ICD-9CM coded diagnoses into Abbreviated Injury Scale (AIS) scores. Studies have shown that AIS scores derived from ICDMAP are generally lower than those obtained by reviewing the entire medical record.[10] Discharges with more than one head injury diagnosis were classified according to the most severe (highest ICD/AIS score) head injury. Discharges with a head injury of ICD/AIS 1 or 2 are classified as minor, ICD/AIS 3 as moderate, 4 as serious, and 5 as severe.

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TABLE 1—Age-Adjusted Head Injury Discharge Rates (per 100,000) by Year and Severity, Maryland, 1979–86

Year	Severity of Head Injury				
	Mild (ICD/AIS 1–2)	Moderate (ICD/AIS 3)	Serious (ICD/AIS 4)	Severe (ICD/AIS 5)	Total
1979	85.7	18.1	8.2	1.7	113.7
1980	82.1	18.3	8.4	2.9	111.7
1981	85.4	21.0	8.2	3.3	117.9
1982	87.6	20.3	8.3	3.6	119.8
1983	88.1	19.6	9.7	4.3	121.7
1984	92.5	20.3	11.1	5.0	128.9
1985	95.2	20.8	12.0	5.7	133.7
1986	94.3	23.1	11.8	5.0	134.2

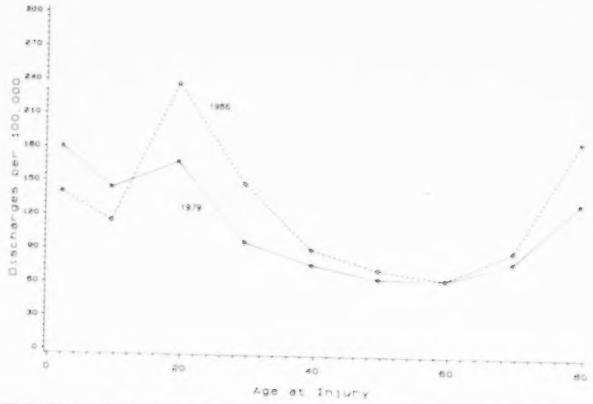

FIGURE 1—Head Injury Discharge Rates (per 100,000) by Age at Injury

Results

Since 1979 there has been an overall increase of 18 percent in the age-adjusted head injury discharge rate from 113.7 to 134.2 per 100,000 (Table 1). A linear regression fit to these data predicted an average increase of 3.4 per 100,000 discharges per year (R^2 = .93). The largest percent increase over the eight-year period was noted among the most severe (ICD/AIS 5) injuries which nearly tripled from 1.7 to 5.0 per 100,000.

Discharge rates for young children ages 0–4 actually declined (180 per 100,000 in 1979 vs 140/100,000 in 1986) whereas rates for adolescents and young adults (ages 15–24) increased (167 vs 236), as did rates among the elderly age 75+ (145 vs 188) (Figure 1). Very little change in discharge rates was noted for adults aged 40–64. The relative risk of age-adjusted head injury discharge rates for males versus females remained relatively constant during the study period with males twice as likely as females to be hospitalized for treatment of a head injury (Table 2). The relative risk for Whites versus others, on the other hand, has declined slightly (1.3 to 1.0).

Hospital case fatality rates have declined between 1979 and 1986 (Table 3) for all severity groups with the greatest relative decrease observed among the moderately severe (ICD/AIS 3) injuries (8 to 5 percent). The percent discharged home has decreased for each severity group while the percent discharged to an extended care facility has increased. This shift in discharge disposition was most notable among individuals with the most severe injuries. Among the ICD/AIS 5 injuries, there was an 8 percent decrease in discharges to home (31 to 23 percent) and a 5 percent decrease in fatality (53 to 48 percent) accompanied by a 15 percent increase (9 to 24 percent) in discharges to an extended care facility.

Discussion

Hospital discharge abstract data generally provide a good source of information on the incidence of injury severe enough to require hospitalization,[9,11,12] and provides readily available population-based data specific to severity. Further, since these databases are maintained over time, they provide the basis for examining temporal trends in incidence.

Since the majority (80 percent) of head injuries in young children were of minor severity, the decrease may reflect changes in admission practices; moreover, in 1983, Children's Hospital in Washington, DC was designated a regional referral center for pediatric trauma, and an increasing proportion of pediatric head injuries occurring in the DC Met-

TABLE 2—Age-Adjusted Head Injury Discharge Rates by Sex and Race (per 100,000), Maryland, 1979–86

	Sex			Race		
Year	Males	Females	Relative Risk	White	Other	Relative Risk
1979	132.4	63.5	2.1	121.0	94.5	1.3
1980	131.5	61.2	2.2	119.4	91.1	1.3
1981	135.5	63.6	2.1	125.2	99.3	1.3
1982	138.7	67.6	2.1	126.5	102.9	1.2
1983	141.2	67.1	2.1	126.3	111.0	1.1
1984	144.6	71.5	2.0	134.7	116.8	1.2
1985	147.0	68.1	2.2	135.4	132.3	1.0
1986	144.2	67.4	2.1	134.0	137.7	1.0

TABLE 3—Discharge Status by Severity of Head Injury, Maryland, 1986 vs 1979

	Severity of Head Injury							
	Minor		Moderate		Serious		Severe	
Discharge Status	1979	1986	1979	1986	1979	1986	1979	1986
% Home	96	94	83	80	70	66	31	23
% Transferred to another acute care facility	1	2	4	4	5	7	7	5
% Transferred to a rehabilitation or extended care facility	1	3	5	10	7	14	9	24
% Died	2	1	8	5	18	13	53	48

ropolitan Area may have been triaged there and would not be recorded in the Maryland hospital discharge abstract database. A child restraint law enacted, in 1984 may also have contributed to the decline.[13,14]

The increased use of ICD-9CM coded discharge diagnoses as a basis for hospital payment has been shown to result in more attentive coding which might explain a shift toward more severe injuries among adolescents, young adults, and the elderly.[15] However, Maryland hospitals have been paid under a per-case payment system since the mid 1970s so that changes in coding practices are not likely to have occurred over the study period. The increased availability and use of CT (computed tomography) scans in the early 1980s may account for increased specificity in diagnosis of specific brain lesions which could result in a shift toward higher ICD/AIS scores. However, neither coding practices nor improved diagnostic procedures would likely account for the increase in overall discharge rates.

The increase in discharge rates accompanied by a decline in the hospital care fatality rates may reflect improved emergency medical services, or increased use of seat belts.[16] The increase in hospital discharge rates over the last eight years accompanied by a small decrease in the case fatality rate has resulted in an increase in the number of survivors with head injury severe enough to result in hospitalization. Several studies have documented the nature of sequelae resulting from severe as well as minor head injury.[17–25] As the number of head injury survivors increases, more hospital-based as well as community services aimed at rehabilitating the head-injured individual will be needed.

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The Impact of Legalized Abortion on Adolescent Childbearing in New York City

THEODORE J. JOYCE, PHD, AND NACI H. MOCAN, PHD

Abstract: In this paper we estimate the impact on adolescent childbearing of the liberalization of the New York State abortion law in 1970. Using Box-Jenkins time series techniques to analyze monthly data on the number of births to White and Black adolescents from January 1963 to December 1987, we found that the level of births to Black adolescents living in New York City fell 18.7 percent, approximately 142 fewer births per month, after the law became effective; the level of White births fell 14.1 percent, approximately 111 fewer births per month. Projections based on the fitted model suggest that a ban on legalized abortion today would have a major impact on adolescent childbearing in New York City as well as other parts of the country, although the magnitude of the change would vary according to local conditions. (*Am J Public Health* 1990; 80:273–278.)

Introduction

There has been great speculation in the popular press whether the US Supreme Court will overturn the 1973 decision in *Roe versus Wade*, the case which legalized abortion across the United States. A likely outcome of such a reversal is that the authority to regulate abortion will be given to individual states. To understand how a dramatic shift in the availability of legalized abortion might affect adolescent childbearing, we examined the changes in the number of births to New York City teenagers following the 1970 New York State law which liberalized abortion. It is our contention that the decline in teenage childbearing between 1970 and 1971 is a good approximation, in reverse, to what would occur today if legalized abortion were no longer available.

A number of studies have noted the decline in births after the New York State Law which liberalized abortion became effective.[1–3] However, the results in each of the studies were based on annual changes over a very short time span. Such unrefined estimates provide only a crude understanding of what might occur if the legalization of abortion were reversed. Pooled time-series, cross-sectional studies have examined the effect of liberalized abortion laws on annual, age-specific fertility rates within and across states.[4–6] The findings suggest that the legalization of abortion had an important impact on fertility rates. Again, the variation over time was limited to at most seven years.

Our study differs substantially in that it is a time-series analysis with monthly data that spans 25 years. The large number of observations allows for a more sophisticated means of fitting the data. In particular, we used Box-Jenkins methods to determine the magnitude and statistical significance of the change in adolescent childbearing that followed the liberalization of abortion in New York City in 1970. Based on the fitted model, we estimate the number of unintended births that were averted due to the widespread availability of legalized abortion. The estimates shed light on the changes in adolescent childbearing that would occur if legal abortions were to become unavailable to New York City residents.

Address reprint requests to Theodore J. Joyce, PhD, National Bureau of Economic Research, 269 Mercer Street, 8th floor, New York, NY 10003. Dr. Joyce is also with the Baruch College/Mount Sinai School of Medicine, City University of New York. Dr. Mocan is with the National Bureau of Economic Research and the Manhattan College. This paper, submitted to the Journal May 25, 1989, was revised and accepted for publication August 8, 1989.

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Methods

Data

Monthly figures on the number of Black and White live births to New York City residents less than 20 years of age are from vital statistics maintained by the New York City Department of Health. Each year of individual birth records has been aggregated by month for Blacks and Whites separately. The number of birth records with unknown age in any one year was less than .03 percent; those records were deleted from the aggregation. The final series consisted of 300 monthly observations for Whites and Blacks from January 1963 to December 1987. Plots of the race-specific series are shown in Figures 1 and 2.

The analysis was limited to Whites and Blacks because ethnicity was not identified on New York City birth certificates until 1978. However, a substantial proportion of the adolescents who are White are of Hispanic origin or descent. Data from the 1970 Census indicate that 13.4 percent of all women 15 to 19 years of age were of Puerto Rican descent.[7] The number of other Hispanic adolescents in 1970 is not known. By 1980 17.0 percent of adolescents 15 to 19 years of age were Puerto Ricans, while the total proportion of all Hispanic adolescents of the same age stood at 25.9 percent. Moreover, in 1984, the first year in which data on births to Hispanic women were published, 75 percent of the White adolescents who gave birth were of Hispanic origin.[8]

The number of births as opposed to birth rates were analyzed because monthly population figures for New York City were unavailable over the period under study. Census data could have been used to estimate monthly population figures between the census years, but such crude estimates would have introduced measurement error. Furthermore, month-to-month changes in the population are minor compared to the 10 to 20 percent drop in the number of adolescent births that were observed between 1970 and 1971.

Statistical Analysis

Two approaches were used to measure the impact of the 1970 legislation on adolescent childbearing. Both approaches used Box-Jenkins time-series analysis.[9] The first approach fitted an autoregressive integrated moving average (ARIMA) model to the pre-legislation data (January 1963–June 1970). Based on the fitted model, we projected the number of adolescent births that would have been expected after July 1970 had abortion not become legal. The difference between the projected births and the actual births is one estimate of the number of births that were averted by the liberalization of abortion.

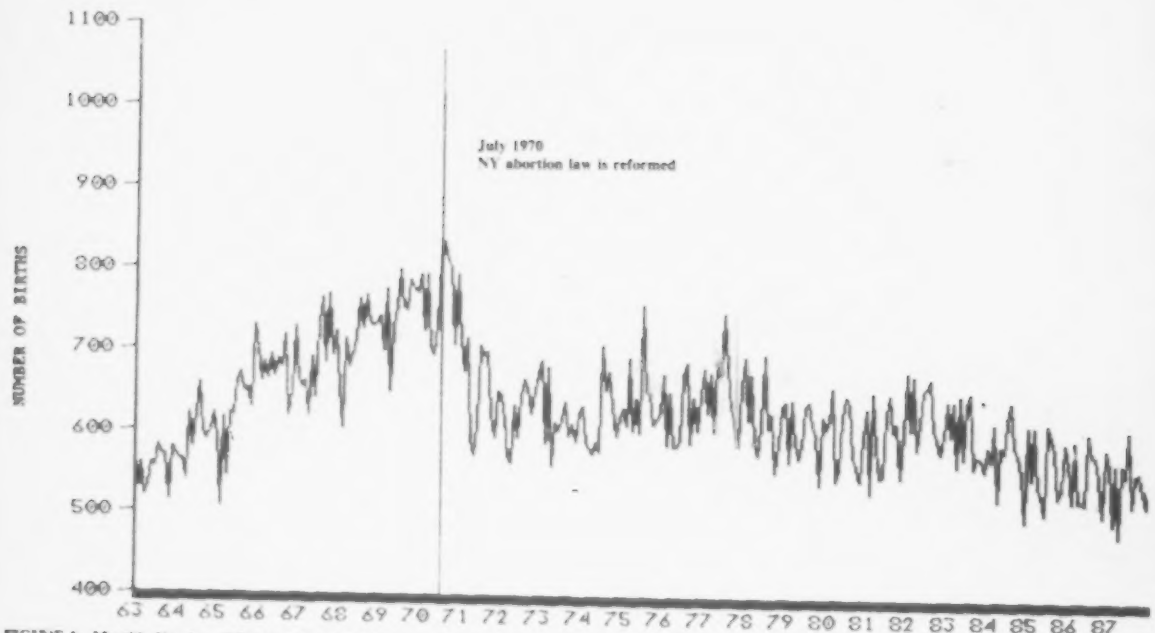

FIGURE 1—Monthly Number of Births to Black Adolescents Living in New York City, January 1963–December 1987

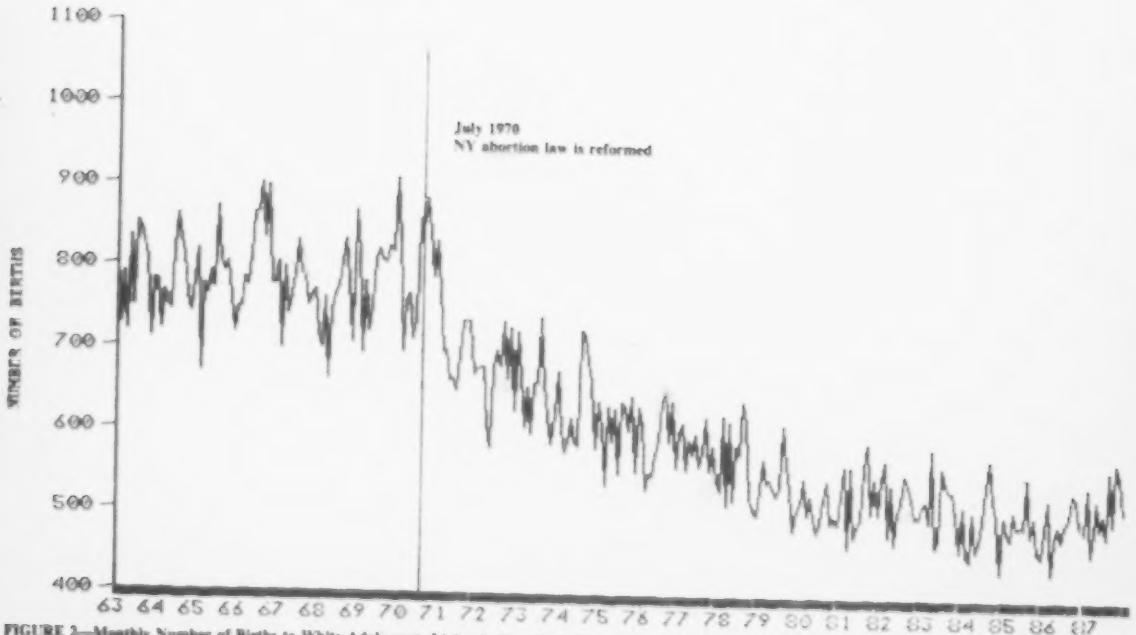

FIGURE 2—Monthly Number of Births to White Adolescents Living in New York City, January 1963–December 1987

The second approach used intervention analysis, an extension of the Box-Jenkins methodology, with the entire data set (January 1963–December 1987).[10,11] The coefficient of the intervention component measures the change in the monthly level of births between the pre- and post-intervention series. The advantage of the intervention analysis is that it is a straightforward means of determining whether the change in the series was statistically significant. Another advantage is that the magnitude of the change is based on a fitted model from observed data over the entire range of the series. The first approach is based on projections generated by the pre-intervention data only. The advantage of the first approach is that if the data were trending prior to the intervention, then the projections would incorporate the trend. The implications of the differences between the two approaches are discussed below.

To successfully apply intervention analysis, it is necessary to know the starting point of the event as well as the general shape of the response of the series to the event. The hypothesis maintained in this study is that the 1970 New York State Law which liberalized abortion had an important impact on the number of adolescent pregnancies that resulted in live births. The law became effective on July 1, 1970. However, because the law did not apply to pregnancies greater than 24 weeks, the effect of the law on the number of live births would not be observed for at least 16 to 20 weeks later. Thus, November 1970 became the starting point of the intervention. Furthermore, the full impact of the law on births to adolescents would not be realized until April 1971 when the pregnancies of the first cohort of adolescents who conceived on or after July 1, 1970 reached term. Consequently, the intervention variable was specified in such a manner that the law's impact on the number of adolescent births increased gradually from November 1970 through April 1971.

The rate at which the law's impact grew between November and April was based on the distribution of abortions by gestational age to New York City residents the first year the law was in effect. For example, 6.1 percent of all abortions to New York City residents were to women whose pregnancies were beyond the twentieth week.[2] Assuming the distribution of abortion by gestational age was the same for every month in the first year,the proportion of the law's full impact that would be felt in November was .061. Thirty percent of all abortions performed in the first year were to women whose pregnancies were between 13 and 20 weeks gestation. We assumed that 15 percent were performed between 13 and 16 weeks gestation, and the other 15 percent were performed between 17 and 20 weeks gestation. Thus, in December, the proportion of the law's full impact would be .216. This accounts for the 6.1 percent of the women who aborted in August 1970 whose pregnancies were greater than 20 weeks gestation and for the 15 percent of the abortions in July 1970 to women whose pregnancies were between 17 and 20 weeks gestation. Following this algorithm and noting that 64 percent of all abortions were performed in the first trimester, the figures for the remaining months were as follows: .361 in January, .574 in February, .785 in March, and 1.0 in April and all months thereafter.

Results

Figures 1 and 2 present the monthly number of births to Black and White New York City adolescents from January 1963 through December 1987. For Blacks, the reversal of a seven-year upward trend between 1970 and 1971 is dramatic. In the case of Whites, a relatively stationary series up to 1970 falls substantially between 1970 and 1971 and then continues downward until approximately 1986. Both figures suggest a major alteration in adolescent childbearing that is coincident with New York State's liberalized abortion law which became effective July 1970.

The ARIMA specification for the pre-intervention series is presented in Table 1. The data are expressed as natural logarithms in order to control for non-stationarity in the variance. A first-order difference transformation was applied to the logarithms of births in order to remove any trend; a twelfth-order difference was used to eliminate seasonality. Based on the autocorrelation and partial autocorrelation functions of the transformed series, Black and White births can be characterized as a first-order moving average with a first-order seasonal moving average. The coefficients of the models are displayed in Table 1. The Q-statistics in Table 1 indicate that the residuals from the estimated models are "white noise" (random variation) processes.[12] Another approach for determining if the errors are "white noise" is to test whether the first difference of the residuals follows a first-order moving-average process with the moving-average parameter equal to 1, and the first autocorrelation equal to −.5. For both Blacks and Whites this was confirmed (results not shown).

Based on the fitted model in Table 1, we projected the number of race-specific adolescent births 24 months beyond June 1970. The projections assume that abortion was not liberalized. A comparison of the actual number of births to the projected number of births is presented in Figures 3 and 4. Subtracting the actual number of births from the projected number and summing over the 24 months indicate that 4,091 Black births and 3,128 White births were averted by the legalization of abortion over the initial two-year period under the law. Note these figures include the months immediately following the new law in which the change in adolescent childbearing was relatively minor. If one estimates the *monthly* number of averted births based on the first year over which the liberalization realized its full impact (April 1971 through March 1972) then 226 Black births and 178 White births were averted per month.

To more formally test whether the 1970 law liberalizing abortion may have caused the precipitous drop in adolescent births, we used all the data to re-estimate the ARIMA structure of each series. As outlined in the previous section, an additional variable was added to the specification to

TABLE 1—Estimated ARIMA Equations for NYC Births to Black and White Adolescents: 1964–1970

BLACKS	θ_1	Θ_1	Q*(24)	R^2
$B_t = -\theta_1 e_{t-1} - \Theta_1 e_{t-12} + e_t$	.735 (9.16)	.801 (9.24)	15.01 .92	.63
WHITES	θ_1	Θ_1	Q(24)	R^2
$W_t = -\theta_1 e_{t-1} - \Theta_1 e_{t-12} + e_t$	.772 (9.38)	.644 (5.70)	19.41 .73	.56

B_t and W_t are the natural logarithms of the Black and White births respectively. θ_1 and Θ_1 are the coefficients and e_{t-1} is the error term. The numbers in parentheses are the t-ratios.
*The Ljung-Box Q statistic determines the randomness in autocorrelations of residual errors, and has a Chi-square distribution.[12] The numbers below the Q-statistics are the marginal significance levels, i.e., the probabilities of the null hypothesis that the autocorrelations of the errors are not different from zero.

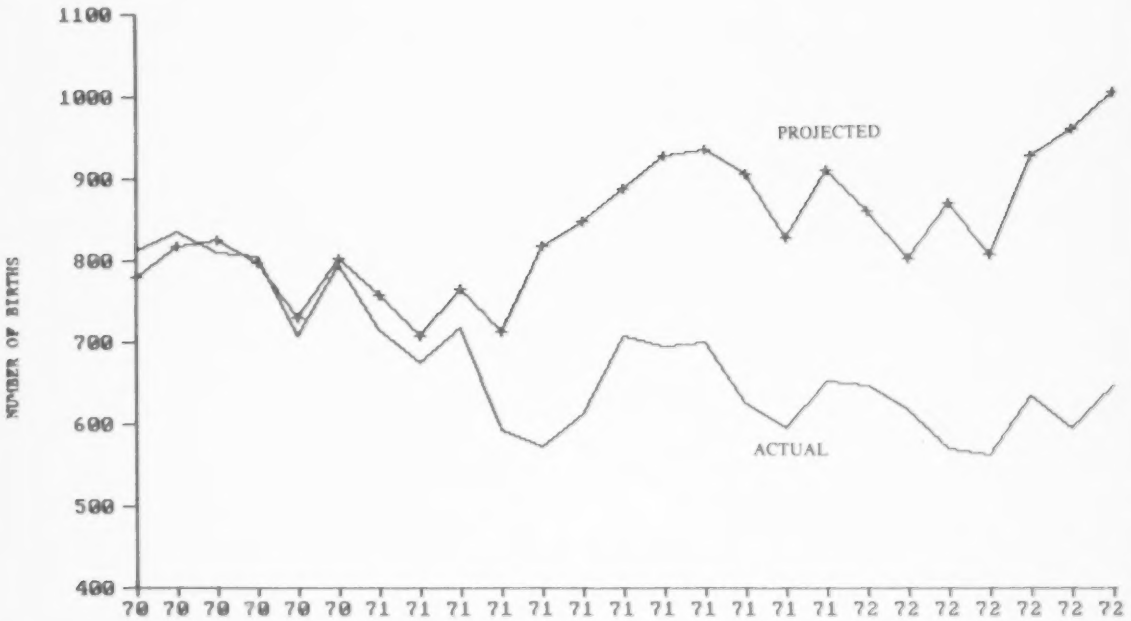

FIGURE 3—Monthly Number of Actual and Projected Births to Black Adolescents Living in New York City, July 1970–July 1972

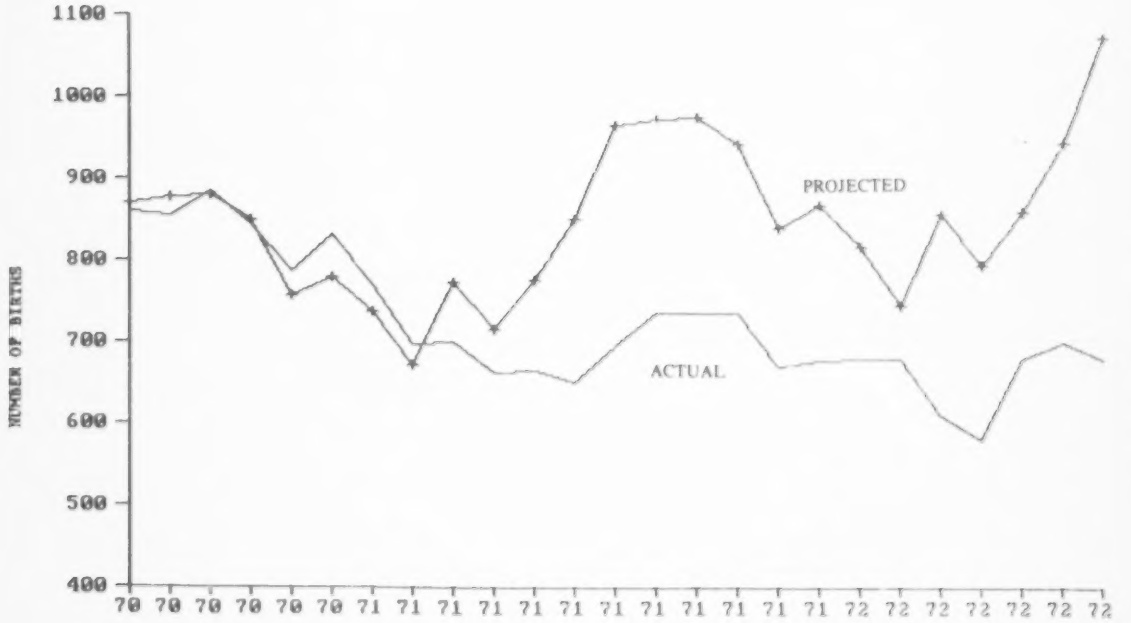

FIGURE 4—Monthly Number of Actual and Projected Births to White Adolescents Living in New York City, July 1970–July 1972

control for the impact of the law. The results are shown in Table 2. Except for the intervention component, the ARIMA structure is unchanged for Blacks. In the case of Whites, a second-order seasonal moving-average component improved the model's fit. The coefficient of the intervention variable, α, is statistically significant for Blacks and Whites. Thus, the data reveal that the decline in the level of births after October 1970 was a change that could not be explained by the normal variation in the series.

The magnitude of the change from the pre- to post-intervention level of the series can be obtained by exponentiating the coefficient on the intervention variable, α, and subtracting it from one. Expressed as a percentage, the level of Black adolescent births fell 18.7 percent after the liberalization of abortion in July 1970 (95% confidence interval = 25–12). White births fell 14.0 percent (95% CI = 20–8). If we use the average number of births over the 12 months prior to July 1970 as an estimate of the pre-intervention level of births, then 2,588 Black adolescent births and 1,998 White adolescent births were averted in the 24 months after July 1970. If the number of averted births is estimated over the first 12 months in which the law's full impact was realized (April 1971–March 1972), then 142 Black births and 111 White births were averted per month.

Discussion

Using monthly data on the number of White and Black births to New York City adolescents, we found that the liberalization of the New York State abortion law in 1970 had a substantial impact on adolescent childbearing. We also found that Blacks were more affected than Whites by the liberalization of abortion. The finding suggests that Whites may have had more access to illegal abortions than Blacks prior to the liberalization.

We used two approaches to estimate the number of births that were averted by the widespread availability of legalized abortion. We consider the estimates obtained from the intervention analysis to be conservative estimates for they do not take into account the upward trend in the number of births especially among Blacks prior to the 1970 liberalization (see Figure 1). The advantage of these estimates is that the level of births from which the changes are estimated is based on actual, as opposed to projected, data. The estimated number of averted births that were obtained by subtracting the actual number of births from the projected number of births should be viewed with some caution. Although they incorporate underlying trends, the projections are based on only seven years of data.

Finally, it should be noted that the decline in births to adolescents in the two years after July 1970 would not necessarily lead to a decline in completed fertility if the teenagers having abortions simply delayed their childbearing until a later age. However, the evidence to date indicates that early childbearers have more children and more unwanted children than women who delay childbearing.[13]

The analysis was limited to New York City because of the special circumstances surrounding the liberalization of New York abortion laws in 1970. Prior to the change in 1970, pregnant adolescents had essentially no access to legal abortion. Although Hawaii, Alaska, and Washington State has laws similar to New York's by the end of 1970, each state had residency requirements. Consequently, the magnitude of the change in adolescent childbearing among New York City residents after the 1970 law became effective was not diminished by migration to other states. The same was not true for residents of other states after the passage of the New York State law. Between July 1970 and June 1971, 75.4 percent of the 33,964 abortions performed on adolescents in New York City were to out-of-state residents.[2]

To gain some insight into the impact on teenage childbearing if legal abortion were unavailable to New York City residents, we applied the coefficients on the intervention variable in Table 2 to the projected number of births in 1988 and 1989. We adjusted for the gradual impact of a ban by the gestational age distribution of abortions prevailing today. If legal abortion were banned January 1, 1988 there would have been 2,618 (18.7 percent) additional Black births and 1,223 (14.0 percent) White births to New York City adolescents in 1988 and 1989 above what would have been expected had the laws regarding abortion remained unchanged.*

The projections are based on a number of assumptions that should be made explicit. First, we assume that the reversal of *Roe versus Wade* would result in a nationwide ban of legalized abortion. Although such an outcome is unlikely, the projections remain instructive because they provide upper-bound estimates based on the most restrictive scenario possible. This scenario cannot be dismissed lightly given that President George Bush, US Attorney General Richard Thornburgh, and Health and Human Services Secretary Louis Sullivan have publicly stated their opposition to *Roe versus Wade*.

A more likely scenario if *Roe versus Wade* were overturned is that New York State will continue to permit legalized abortion. New York is one of only 13 states plus the District of Columbia that currently funds abortions to Medicaid-eligible women and one of only eight states that does so voluntarily.[14] Nevertheless, New York's 1970 law passed the State Senate by five votes and the State Assembly by only one. The debate in both houses was tumultuous.[15]

A second assumption upon which the projections are based is that the use of abortion by adolescents in the early 1970s is unchanged. In fact, the percentage of pregnancies (live births plus induced abortions) terminated by induced abortion has risen from 40.2 percent for Whites and 47.1

TABLE 2—Estimated ARIMA Equations for NYC Births to Black and White Adolescents with the Intervention Component 1964–1987

BLACKS $B_t = -\theta_1 e_{t-1} - \Theta_1 e_{t-12} + e_t + \alpha I_t$

θ_1	Θ_1	α	Q*(48)	R^2
.776	.838	−.207	34.20	.64
(20.68)	(24.87)	(−5.32)	.93	

WHITES $W_t = -\theta_1 e_{t-1} - \Theta_1 e_{t-12} - \Theta_2 e_{t-24} + e_t + \alpha I_t$

θ_1	Θ_1	Θ_2	α	Q(48)	R^2
.824	.740	.122	−.150	42.16	.61
(23.94)	(12.04)	(1.95)	(−4.10)	.71	

B_t and W_t are the natural logarithms of the Black and White births respectively. θ_1 and Θ_1 are the coefficients and e_{t-1} is the error term. I_t is the dummy variable for the intervention; α is its coefficient. The numbers in the parentheses are the t-ratios.

*The Ljung-Box Q statistic determines the randomness in autocorrelations of residual errors, and has a Chi-square distribution.[12] The numbers below the Q-statistics are the marginal significance levels; i.e., the probabilities of the null hypothesis that the autocorrelations of the errors are not different from zero.

*A detailed description of how the projections were obtained is reported in the National Bureau of Economic Research Working Paper No. 3002 by the authors.

percent for Blacks in 1972 to 54.8 and 59.2 percent, respectively, in 1986.** In this respect, our projections may underestimate the impact of a ban on legalized abortion.

One explanation for the increased use of abortion by adolescents is that the availability of abortion has engendered less effective contraceptive behavior; the more extreme version is that abortion has served as an alternative method of fertility control. Thus, if abortion were banned, the pregnancy rate may fall because of better contraceptive behavior. There is no evidence to support either explanation. A 1982 survey reported that only 0.4 percent of women at risk of an unintended pregnancy but using no contraception admitted doing so because they relied on abortion.[16] And although little is known about the relationship between abortion availability and the effective use of contraceptives, a teenager who aborted a pregnancy was less likely to become pregnant again over the next 24 months than was a comparable adolescent who carried her first pregnancy to term.[17]

As noted above, the proportion of births to White adolescents of Hispanic descent is substantial. Based on the little data that exists, Hispanic adolescents are more sexually active than their White counterparts who are not Hispanic. At the same time, Hispanic adolescents are more likely to be married and less likely to abort.[18,19] To the extent these tendencies offset each other, the proportion of Hispanics adolescents at risk of an unintended pregnancy may be similar to that of other Whites.

There is a voluminous literature on the social and economic consequences of adolescent childbearing. As the most recent and comprehensive review makes clear, adolescents who become parents will complete less schooling, have lower wages, experience greater marital instability, and be more dependent on welfare programs than their adolescent peers who delay childbearing.[13,18] Moreover, the children of teenage mothers will experience greater health, cognitive, and socioemotional difficulties.

Nor would a ban on legalized abortion be costless to taxpayers. In 1986, 64.6 percent of all adolescent births in New York City were funded by Medicaid.[20] Assuming Medicaid eligibility is a good proxy for AFDC (aid to families with dependent children) eligibility, then applying this proportion to the number of unintended births reported above indicates that 790 White and 1,691 Black teenage mothers and their children will receive AFDC in the two years after the ban. Based on the methodology described by Burt,[21] the present discounted cost in terms of Medicaid, AFDC, and food stamps of supporting a family headed by a teenager over a 20 year period is $5,560 in 1985 dollars above what it would have cost to support the same teenager and her family had she delayed childbearing until after she was 20 years of age.[21] Thus, the total marginal costs of supporting the 790 White and 1,691 Black births described above would be $13.8 million.

The percentage changes in adolescent childbearing among Whites and Blacks following a prohibition of legal abortion are clearly speculative and cannot be applied to other parts of the country because the projections are based on a set of circumstances specific to New York City in 1970. However, the number of unintended pregnancies among US adolescents strongly suggests that areas in which legal abortion is prohibited will experience substantial increases in the number of births to teenagers. The magnitude of the change will vary by area because of differences in the use of abortion prior to a ban, the proximity to areas where abortion remains legal, and the availability of illegal abortions.

**The figures are based on data provided by the New York City Department of Health.

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An Evaluation of External Cause-of-Injury Codes Using Hospital Records from the Indian Health Service, 1985

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Abstract: To evaluate the usefulness of International Classification of Diseases external cause-of-injury and poisoning codes (E codes) for public health surveillance of nonfatal injuries, we analyzed E codes from Indian Health Service (IHS) hospital records. E codes for unknown or unspecified causes were used for 25 percent of records. At two hospitals, 63 percent of E codes assigned by independent coders agreed; another 18 percent matched on general cause-of-injury groups. With uniform guidelines and increased training, E coding could provide a valuable, cost-effective method of quantifying and characterizing severe, nonfatal injuries. (*Am J Public Health* 1990; 80:279–281.)

Introduction

Because external causes of injury and poisoning are routinely coded on death certificates under the International Classification of Diseases (ICD) external cause-of-injury and poisoning coding scheme (E codes),[1] we have learned a great deal about fatal injuries. We know much less, however, about the causes of the many more numerous nonfatal injuries, partially because we lack the appropriate data. Although hospital discharge records provide a potential mechanism for public health surveillance of nonfatal injuries severe enough to require hospitalization, most hospitals do not routinely E code discharge data. Recently, influential health policy and advisory groups, including the National Academy of Sciences and the Council of State and Territorial Epidemiologists, have advocated routine E coding of hospital discharge data to link injury data collection and the development and implementation of intervention strategies.[2–4]

Presently, population-based E coded hospital discharge data are already available in the Indian Health Service (IHS). To examine a practical application of hospital discharge data E coding as well as the strengths and limitations of using E codes for injury surveillance, we analyzed IHS records to determine the level and quality of E coding, the reliability of E codes assigned by different coders, and the feasibility of using E coded hospital discharge data as an injury surveillance tool.

Methods

Discharge records for eligible American Indians and Alaska Natives hospitalized at IHS facilities from October 1, 1984—September 30, 1985, were selected for analysis. We excluded records for medical care provided by outside contractors and at tribal facilities (27.9 percent of admissions[5]) because they are not reliably E coded.

Medical records for all hospitalizations were coded by on-site medical records personnel at each of 46 IHS hospitals using the standard ICD (ninth revision)-Clinical Modification (ICD-9-CM)[6] codes and rules. Records were coded after discharge based on clinical assessment, diagnostic and therapeutic procedures, and other health services information collected during the hospitalization and recorded on the chart. Each IHS record was coded for: 1) up to six clinical codes; and 2) one cause-of-injury code, if the hospitalization was injury-related.

The ICD hospital indexing system includes the Classification of Diseases and Injuries (often called "N codes"); two supplementary classifications: Factors Influencing Health Status and Contact with Health Service (V codes*) and External Causes of Injury and Poisoning (E codes); and four appendices (Morphology of Neoplasms, Glossary of Mental Disorders, Classification of Drugs, and Classification of Industrial Accidents). Diagnostic and therapeutic procedures are coded with yet another classification scheme, the Classification of Procedures in Medicine.

The cause-of-injury/poisoning E code has several components. All E codes have three digits following an "E." Three-digit E codes can be grouped according to general external cause or intent (e.g., E880-E888: accidental** falls, E950–E959: suicide and self-inflicted injury). Most three-digit E codes also require a fourth digit subdivision (EXXX.0–EXXX.9) to identify the role of the injured person or to provide additional cause-specific information.

For this analysis, we selected injury records by two independent methods: 1) records with injury and poisoning diagnosis and health services codes (ICD-9-CM codes 800–999 and V71.3–V71.6); and 2) records receiving cause-of-injury E codes (E800–E999). We then compared the files to measure the level of injury coding. To evaluate the reliability of IHS E codes, we reviewed E coded records for two hospitals, abstracted cause-of-injury information, and recoded the external causes of injury and poisoning. The author (SMS) who recoded records was blinded to the original codes.

Results

Of the 10,921 (13.9 percent) IHS hospital discharge records with at least one injury-related diagnosis or health services code, 99.3 percent were E coded. All records with an injury diagnosis code (800–999) had an associated E code;

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*V codes are used for occasions when a person encounters the health care system for a specific purpose other than illness or injury (e.g., immunization or pregnancy). For injuries, certain V codes may be appropriate for persons hospitalized for observation following a traumatic event.

**Although use of the term "accidental" denotes an unpreventable event and, therefore, is to be avoided, unintentional injuries are labeled "accidental" in the ICD.

52 of 128 records selected by an injury health services codes (V71.3–V71.6) were E coded.

Of the 11,063 E coded records selected independent of diagnosis codes, 10,845 (98.0 percent) had at least one injury or poisoning diagnosis or health services code. For the other 218 E-coded records, the most frequent conditions requiring hospitalization were adverse effects of medical/surgical care (N = 106) and poisoning (N = 9). These 11,063 hospitalizations were required for 9,555 individuals.

More than 75 percent of all injury hospitalizations were in six broad external cause groups: transport-related incidents, falls, acts of violence, adverse effects of medical and surgical care, other accidents (being struck, caught, or cut), and suicides/attempts (Table 1). Transport-related events and falls together accounted for more than a third of all injury hospitalizations.

About 25 percent of IHS injury hospitalization records were coded using three-digit E codes for general or unspecified causes of injury, reflecting a lack of more detailed information about the injury events. Most frequently, records for falls, transport-related injuries, and assaults could be coded with only general E codes. For 540 records, the underlying cause of injury was not available, and coders used two non-specific codes, E887 (fracture, cause unspecified) and E928 (other and unspecified environmental and accidental causes).

Fourth-digit codes were assigned where appropriate. Coders assigned a fourth-digit code to all three-digits E codes requiring the additional character. For those three-digit E codes without an associated fourth-digit, IHS appropriately did not assign a fourth digit. For 22.6 percent of records with E codes requiring a fourth digit, a nonspecific fourth digit code was assigned because sufficient information to code the fourth digit was not provided in the record.

The E coding reliability evaluation was based on injury hospitalization records from two IHS Service Units. Ninety-seven percent of 333 discharge records receiving E codes were available to abstract and recode. We assessed coding agreement on a hierarchical scale. When we compared recoded E codes with those assigned by IHS medical records personnel, 59 percent agreed completely (agreement to the three-digit level and agreement to the four-digit level where appropriate). Comparison of the three-digit agreement hierarchy is shown in Table 2. Overall, in 63 percent of the records, the original and recoded three-digit E codes matched. Eighty-one percent of records had matching three-digit codes or similar codes from an external cause group. An additional 5 percent of records had original and recoded E codes that fell under the same ICD-9-CM rubric of intent (i.e., unintentional, intentional, or unknown intent of injury). Neither three-digit E code, external cause group, nor intent agreed for 14 percent of records. For most of the nonmatching sets of codes, medical care providers recorded different scenarios of the injury event on an individual chart or E codes were selected on the basis of recorded injury event circumstances that did not fit well within the ICD-9-CM classification scheme (e.g., Should a fall out of an automobile be coded as a fall or as a transport-related event?).

TABLE 1—Frequency of Three-Digit External Cause-of-Injury Codes from Hospitalization Records,* by Injury Group, Indian Health Service, Fiscal Year 1985

E Codes	External Cause Group	Frequency	Percent	Cumulative Percent
E800–E848	Transport-related	1896	17.1	17.1
E880–E888	Falls	1871	16.9	34.1
E960–E978,E990–E999	Assaults/legal/war	1606	14.5	48.6
E870–E879,E930–E949	Adverse effects of care	1367	12.4	60.9
E916–E920	Struck/caught/cut	973	8.8	69.7
E950–E959	Suicides/attempts	805	7.3	77.0
E980–E989	Intent unknown	477	4.3	81.3
E900–E909	Environmental	456	4.1	85.4
E928–E929	Other/late effects	455	4.1	89.5
E850–E869	Poisonings	366	3.3	92.9
E927	Overexertion	221	2.0	94.8
All Other E Codes		570	5.2	100.0

*N = 11,063

TABLE 2—Percent Agreement of External Cause-of-Injury Codes for Injury Hospitalization Records, Indian Health Service

Level of Agreement	Percent Agreement*		
	Service Unit A (N = 240)	Service Unit B (N = 83)	Cumulative Percent (N = 323)
Three-digit E code	60	71	63
External cause group	76	91	81
Intent**	83	92	86

*A mutually exclusive hierarchy. For each hospital record, the E code assigned by IHS medical records personnel was compared to the E code assigned by one of the authors. Codes matching on the external cause group are those for which both coders assigned three-digit E codes from the same group of external causes (e.g., transport-related incident [E800–E848], falls [E880–E888], or assaults [E960–E978]) but did not assign identical three-digit E codes.

**Codes matching on intent are those for which both coders assigned E codes of the same intent classification (i.e., unintentional, intentional, or unknown intent) but did not assign identical three-digit E codes or codes from the same external cause group.

Discussion

Because proposals to require universal E coding of hospital discharge data are gaining prominence,[2–4,7,8] this examination of E code use in the IHS is valuable for public health practitioners and other researchers interested in injury surveillance and data collection. Few other opportunities exist for us to test the feasibility of requiring universal E coding and to understand the strengths and limitations of injury cause information obtained from E coded records. As shown in this evaluation, routine E coding of IHS hospital discharge data was a valuable, cost-effective method of quantifying and characterizing severe, nonfatal injuries. E codes provided us with a clear distribution of the underlying causes of injuries requiring hospitalization in this population, thus permitting us to identify priority injury problems. Community-based injury prevention programs, based in part on external cause information extracted from hospital records, are currently being coordinated and conducted by trained injury prevention professionals throughout IHS.[9] The E coded records are also valuable for evaluating the effectiveness of these prevention programs.

E codes were reproducible. Although the specific three- and four-digit codes assigned by independent coders sometimes differed, codes agreed closely according to the external cause group. The extent of E coding agreement compares favorably with previous evaluations of the reliability of death certificate and cancer registry information. In Connecticut, an analysis of autopsy reports and corresponding death certificates found major disagreement leading to reclassification of the underlying cause of death for 29 percent of

deaths.[10] For an additional 26 percent of deaths, the death certificate and autopsy report agreed on the major disease category but attributed the death to a different specific disease. As part of the Third National Cancer Survey, death certificates for 48,826 cases of single primary cancer were compared to hospital diagnoses.[11] The underlying cause of death was found to be accurate in 65 percent of cases. In a comparison of data collected from interviews and those reported to the Missouri cancer registry,[12] 70 percent of occupation, 72 percent of industry, 83 percent of smoking status, and 65 percent of alcohol consumption codes agreed. In spite of these limitations, coded data from death certificates and cancer registries have been used extensively in public health practice. E coded hospital discharge data can be of similar value.

With increased attention to the value of E codes, reliability can be improved. E code users and potential users would benefit from standardized E coding guidelines and definitions, increased availability of E code training, and more rigorous evaluation of the current E code scheme. For example, because only 40.6 percent of hospitalizations for observation following trauma (V71.3–V71.6) received E codes in this analysis, we assume that the use of E codes for records coded with health services codes (V codes), and perhaps other injury codes, was not consistent. Due to reimbursement, hospitals have financial incentives to provide adequate training for coders to ensure accurate diagnosis and procedure coding. As more states require E coding of uniform hospital discharge data,[7] the need for uniform E coding procedures and training will increase.

About a fourth of IHS injury records were coded with E codes for general or unspecified external causes. These general or unspecified codes are much less useful for public health surveillance and the development of injury prevention programs. The nonspecific E codes were used most often because more detailed cause-of-injury information was not recorded in the medical charts. Medical record keeping is, understandably, focused on recording clinical assessment and treatment. Generally, health care providers are not aware of the other ongoing or potential uses for medical record data or of the priority data items to collect for public health surveillance. By presenting studies such as this one, health care providers can be made more aware of the crucial role of the medical record in the flow of data through epidemiologic and statistical processes to health care policy development and evaluation.

Because almost all IHS discharge records with injury diagnosis codes received E codes (including multiple injury-related hospitalizations for individuals in the same fiscal year), these E coded records do not represent injury incidence. We could not determine whether the multiple admissions for individuals were due to multiple injury events or follow-up medical and surgical care for previous injuries. E codes could describe injury incidence, however, if coding rules were standardized, restricting E codes to the initial hospitalization following an injury, or if they were modified to include a separate code identifying the initial hospitalization. We could then code records for subsequent hospitalizations consistently using the existing diagnosis codes available for follow-up treatment and late effects of injuries.

Although E coded hospital discharge data have great value for injury surveillance, they have more limited value for epidemiologic research. E codes can help us meet some of the priority objectives for public health surveillance—ascertaining cases, quantifying and characterizing injuries, and monitoring trends in a defined community.[10] Because the number of E codes for certain specific injury events is restricted or absent, the detail needed for focused epidemiologic research is limited. To obtain more detailed cause-of-injury data or information about specific injury problems of interest (e.g., injuries associated with all-terrain vehicles) we will require other surveillance tools or analytic epidemiologic research.

Information about underlying causes is essential for the development, implementation, and evaluation of injury intervention strategies. E coding of hospital records, as performed in the IHS, provides much of this underlying cause information. By extending hospital record E coding to other health care systems, we can develop a standardized external cause data base for prevention programs.

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The opinions expressed in this paper are those of the above authors and do not necessarily reflect the views of the Indian Health Service.

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Cardiovascular Deaths among Alaskan Natives, 1980–86

JOHN P. MIDDAUGH, MD

Abstract: Average annual, age-adjusted death rates from cardiovascular diseases and atherosclerosis from 1980–86 among Alaskan Natives were lower than rates among other Alaskans (162.0 vs 242.1; RR = 0.67), while death rates from other causes were higher (954.4 vs 618.6; RR = 1.54). These suggest that Alaskan Natives have less cardiovascular disease than other populations. Additional research on the role of marine omega-3 fatty acids is needed. (*Am J Public Health* 1990; 80:282–285.)

Introduction

Numerous reviews on the role of diets rich in omega-3 fatty acids and the incidence of atherosclerosis and coronary artery disease recently have been published, reflecting widespread interest and enthusiasm over the potential beneficial health effects of omega-3 fatty acid consumption.[1–13]

Careful review of published literature reveals that the epidemiologic studies that show a low incidence of atherosclerosis and cardiovascular disease in arctic peoples, particularly, rest on fragile evidence.[6–9,14,15] For example, earlier works in Alaska to investigate causes of deaths were based on a limited number of autopsies conducted by non-pathologists using no standardized protocols without histopathology, and the two studies reached opposite conclusions about incidence of atherosclerosis among Alaskan Natives.[8,9] A study of death records for 1955–65 found lower mortality rates from heart disease among Alaskan Natives than the US population.[15] This study presents additional data based on death certificates on the causes of death among Alaskan Natives.

Methods

All Alaskan death certificates for 1980 through 1986 (N = 13,534) were reviewed. The underlying cause of death and all other conditions that contributed to the death as recorded on the death certificate were coded using the *Ninth International Classification of Diseases—Ninth Revision* (ICD-9). As a validation of the accuracy of coding, for deaths occurring during 1980–82, we manually verified our codes against those furnished by the National Center for Health Statistics (NCHS). Population estimates were obtained from the Alaska Department of Labor and the US Bureau of Census.[16] Alaskan Natives, consistent with designations of the Alaska Department of Labor and the US Bureau of Census, included Eskimo (Inupiaq and Yu'pik), American Indian (Athebaskan, Tlinqit, Haida, Tsimshian and other North American tribes), and Aleut.

We compared average annual death rates between Alaskan Natives and others according to major causes of death, using age-adjusted rates calculated by the direct method using the 1980 US Census as the standard. We compared cause-specific, age-adjusted death rates using the underlying cause of death, only, and by using contributory causes. Groupings of ICD codes are shown in Table 1 and the Appendix. Statistical analyses were done using Epi Info, Version 3, Division of Surveillance and Epidemiologic Studies, Epidemiology Program Office, Centers for Disease Control, Atlanta, Georgia.

Results

Alaskan Natives had a lower age-adjusted mortality rate from atherosclerosis and cardiovascular disease compared to other Alaskans (162.0 vs 242.1; RR = 0.67). Because the differences in death rates between underlying cause and multiple cause were so similar, only data for underlying cause are presented (Table 1). The only major difference in cardiovascular death rates by sex was for the comparison of ICD code 410, acute myocardial infarction, for underlying cause only, where female Natives had a slightly higher average annual, age-adjusted death rate than other females. For all other comparisons of cardiovascular disease-related groups, using underlying cause of death only, multiple causes, male, female, or total population, Alaskan Natives had consistently about 60 percent of the death rate of others.

In contrast, the death rates from infections, respiratory diseases, and neoplasms were all higher among Alaskan Natives than in others. The death rate from cardiovascular disease rose strikingly with age for both Natives and others (Figure 1) and was consistently present among the various cardiovascular-related disease, ICD code groups, by underlying cause and multiple cause, and in both males and females.

Interestingly, there was only one Native death due to aortic aneurysm (ICD code 441) compared to 88 deaths among others (RR = 23.8). The death rate due to all cerebrovascular disease (ICD codes 430–438) was slightly higher among Natives (RR = 1.08) and were primarily related to a higher rate of deaths due to cerebrovascular hemorrhage (ICD codes 430–432; RR = 1.36).

Discussion

This analysis of death certificate data provides additional evidence that arctic indigenous people have strikingly lower death rates from cardiovascular disease than others. The death rate due to cerebrovascular hemorrhage was slightly higher among Alaskan Natives than others, and similar to the overall difference in mortality rates between Natives and others. Other investigators have reported a high rate of cerebrovascular hemorrhage among Eskimos[14,17] and Japanese[18] who consumed high quantities of fish, raising concerns that increased use of dietary consumption of fish oils may lead to the unwanted consequence of cerebral hemorrhage. Our data do not support this concern.

Limitations of death certificates are well known.[19–22] No data exist about their accuracy in Alaska or the correlation of recorded causes of death with autopsy results. Autopsy

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TABLE 1—Causes of Death in Alaskans, 1980–86

Cause of Death ICD-9 Codes	Gender	Number of Deaths		Rate*		RR**	95% Confidence Interval
		Native	Other	Native	Other		
Cardiovascular Diseases and Atherosclerosis							
410	Male	74	323	54.3	60.6	0.90	0.55,1.44
	Female	42	106	43.4	32.8	1.32	0.76,2.35
	Total	**116**	**429**	**50.2**	**49.5**	**1.01**	**0.71,1.46**
410–414	Male	205	1,339	154.1	245.7	0.63	0.47,082
	Female	87	448	90.8	140.0	0.65	0.45,0.94
	Total	**292**	**1,787**	**127.9**	**203.0**	**0.63**	**0.51,0.79**
401–404, 410–414, 429.2, 440	Male	245	1,483	186.2	279.3	0.67	0.52,0.86
	Female	118	568	125.9	182.1	0.69	0.50,0.95
	Total	**363**	**2,051**	**162.0**	**242.1**	**0.67**	**0.55,0.81**
Cerebrovascular Diseases 431	Male	6	24	3.6	3.8	0.95	0.09,5.98
	Female	11	24	6.9	6.2	1.11	0.22,4.38
	Total	**17**	**48**	**5.1**	**4.9**	**1.04**	**0.41,3.51**
430–432	Male	14	46	8.4	5.8	1.45	0.42,5.30
	Female	24	59	16.1	12.5	1.29	0.44,3.06
	Total	**38**	**105**	**12.1**	**8.9**	**1.36**	**0.60,2.79**
430–438	Male	58	180	43.7	38.7	1.13	0.63,1.90
	Female	65	194	60.0	58.0	1.03	0.63,1.63
	Total	**123**	**374**	**52.0**	**48.2**	**1.08**	**0.75,1.53**
Aneurysm 441	Male	1	68	0.8	15.5	0.05	0.02,1.33
	Female	0	20	0	6.5	—	
	Total	**1**	**88**	**0.5**	**11.9**	**0.04**	**0.02,0.87**
Infections 001–139	Male	21	59	12.7	7.0	1.81	0.53,4.81
	Female	18	29	11.9	5.6	2.13	0.67,6.82
	Total	**39**	**88**	**12.5**	**6.5**	**1.92**	**0.92,4.19**
Respiratory 460–519	Male	139	270	97.1	61.9	1.57	1.07,2.27
	Female	100	214	83.9	61.4	1.37	0.92,2.08
	Total	**239**	**484**	**93.2**	**63.3**	**1.47**	**1.13,1.95**
Neoplasms 140–239	Male	300	1,051	223.6	198.4	1.13	0.89,1.44
	Female	243	823	216.3	182.5	1.19	0.92,1.53
	Total	**543**	**1,874**	**224.8**	**195.7**	**1.15**	**0.97,1.36**
Intentional Injuries E950-E969	Male	302	572	124.6	40.8	3.10	2.13,4.40
	Female	86	158	34.5	13.6	2.50	1.29,5.11
	Total	**388**	**730**	**79.6**	**28.5**	**2.80**	**2.02,3.85**
Unintentional Injuries E000-E949, E970-E999	Male	615	1,829	266.3	133.3	2.00	1.58,2.52
	Female	201	400	93.4	43.5	2.10	1.44,3.26
	Total	**816**	**2,229**	**180.3**	**92.7**	**1.90**	**1.59,2.38**
All except Cardiovascular	Male	1,990	5,203	1,071.6	665.4	1.64	1.46,1.83
	Female	1,173	2,684	817.4	553.0	1.48	1.30,1.69
	Total	**3,163**	**7,887**	**954.4**	**618.6**	**1.54**	**1.42,1.68**
All Causes	Male	2,235	6,686	1,257.8	934.7	1.35	1.21,1.49
	Female	1,291	3,252	943.3	735.1	1.28	1.14,1.45
	Total	**3,526**	**9,938**	**1,116.4**	**860.8**	**1.30**	**1.20,1.40**

*Average annual age-adjusted rate per 100,000 by direct method to 1980 U.S. population.
**Native rate/Other rate

results, if available, were not routinely used to determine cause of death that was recorded on the death certificate by the attending physician. ICD coding was done by physician epidemiologists who were not trained nosologists but for another study and prior to involvement with work on diet and atherosclerosis—therefore, potential bias in coding cardiovascular related disease was minimized. We coded all death certificates because previous studies revealed many incorrectly coded certificates and because only the underlying cause of death had been coded. We analyzed our data using our codes; results using multiple cause coding were similar to results using underlying cause only. An analysis of NCHS codes using underlying cause only also showed similar results.

Many recent studies have focused on health effects of fish consumption.[1–5,13,23] Bang emphasized the role of Eskimo diet rich in marine mammal fatty acids, especially seal and whale, and these foods generally were eaten raw.[24] Whether the same health benefits can be anticipated from raw fish and from cooked fish is less clear. Consumption of

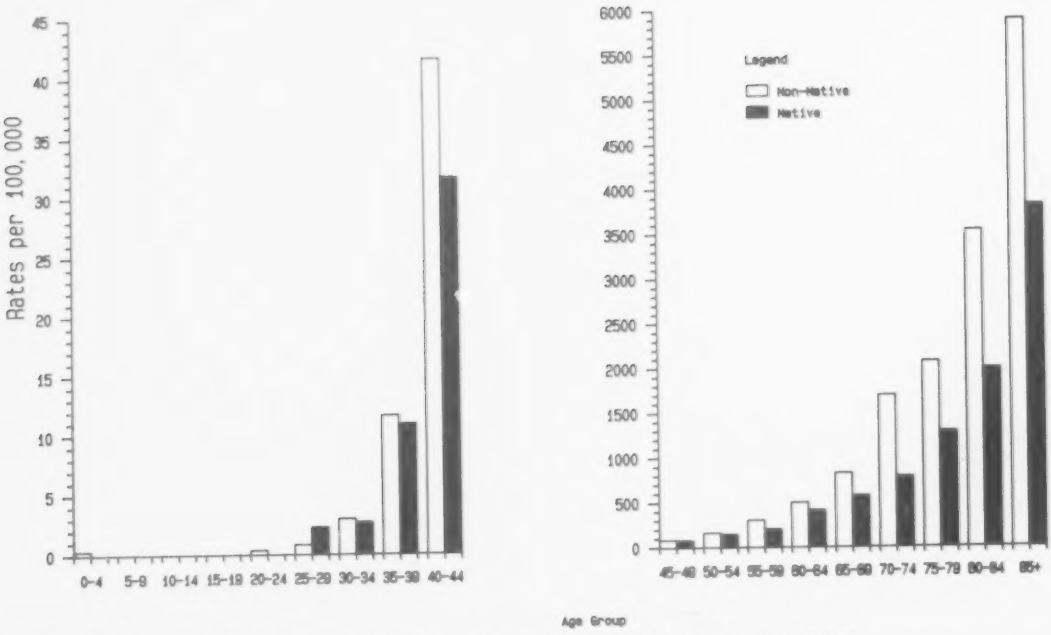

FIGURE 1—Cardiovascular Deaths in Alaska 1980–86 (average annual rate per 100,000 population by age group and race)

salmon remains high and seal, walrus, and whale consumption, both raw and cooked, remains an important part of Alaskan Natives' subsistence diet.

As early as 1900, diet changes among Alaskan Natives were documented as a result of contact with whalers and later with missionaries.[1] Since the 1950s, there has been a dramatic increase in smoking and alcohol consumption. Since the 1960s, western diets increasingly have become available. Since the 1970s, use of snuff has increased.[25] In view of these many life-style changes, it is even more remarkable to find such striking differences in cardiovascular mortality.

The evidence presented here does not establish that the cause of the difference in cardiovascular mortality rates is due to dietary intake of omega-3 fatty acids from marine mammals and fish. It is possible that the differences could be due to genetic factors, or that the observed differences are a result to the effect of competing risks[21,22] acting disproportionately on Natives, or to inaccuracies inherent in death certificate studies. For example, Alaskan Natives may have differences from other Alaskans in the nature and frequency of medical system contacts, the likelihood of receiving care from physicians of similar background and training, availability of diagnostic services, death certification practices in rural compared to urban areas, classification of race on the death certificate, and differences in accuracy of census data. Additional research is needed to pursue accumulating evidence of the beneficial effects that may exist from consumption of omega-3 fatty acids.

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APPENDIX

ICD Code	*Specific Diagnosis*
401	Essential hypertension
402	Hypertensive heart disease
403	Hypertensive renal disease
404	Hypertensive heart and renal disease
410	Acute myocardial infarction
411	Other acute and subacute forms of ischemic heart disease
412	Old myocardial infarction
413	Angina Pectoris
414	Other forms of chronic ischemic heart disease
429.2	Cardiovascular disease, unspecified
440	Atherosclerosis

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ERRATA

In: Wells JK, Williams AF, Fields M: Coverage gaps in seat belt use laws. Am J Public Health 1989; 79:332–333. Table 1 which appeared on page 333 last year is in error. In the listing of all the states, Utah was inadvertently omitted and the wrong data presented for Tennessee and Texas. All other states and data were correct. Printed below are the correct figures for Tennessee, Texas, and Utah. We regret the error.

TABLE 1—Correction

State	Total Deaths	Not Subsequently Covered: Rear Seat Occupants	Not Subsequently Covered: Light Truck Van Occupants	Total Not Covered: N	Total Not Covered: %
Tennessee	3,013	111	0	111	4
Texas	12,564	784	0	784	6
Utah	882	71	0	71	8

* * *

In: Popkin BM, Akin JS, Flieger W, Wong EL: Breastfeeding trends in the Philippines, 1973 and 1983. Am J Public Health 1989; 79(1):32–35. The authors wish to call attention to an error in the coding manual for a data set they used in that paper. Although the error does not change the substantive results, it does change slightly the results presented in their Table 2 (page 34) under the subheading of "Mother's Education (years)," lines 4 and 5 for "7–9" years and "10≅ years". The corrected results show a slightly greater decline among those with 7–10 years of schooling and with more than 11 years of schooling, as shown in the two lines in the table correction below.

TABLE 2—Correction

Characteristics	Average Sample Size	Incidence: 1973	Incidence: 1983	Unadjusted Difference*	95% Confidence Interval of Difference: Lower Limit	95% Confidence Interval of Difference: Upper Limit
7–10	763	86.7	81.2	−5.5	−9.1	−1.9
11≅	1058	66.2	72.6	6.4	1.0	11.8

Salmonella arizona Infections in Latinos Associated with Rattlesnake Folk Medicine

STEPHEN H. WATERMAN, MD, MPH, GREGORY JUAREZ, MPH, SUSAN J. CARR, MPH, AND LIGA KILMAN, BS

Abstract: In 1987 two Los Angeles County (California) hospitals reported four Latino patients with serious *Salmonella arizona* (*Salmonella* subgroup 3) infections who gave a medical history of taking rattlesnake capsules prior to illness. Capsules supplied by the patients or household members grew *Salmonella arizona*. We reviewed surveillance data for this *Salmonella* species and conducted a case-control study to determine the magnitude of this public health problem. Eighteen (82 percent) of the 22 Latino cases in 1986 and 1987 who were questioned reported ingesting snake capsules compared to two (8 percent) of 24 matched Latino controls with non-subgroup 3 salmonellosis or shigellosis (matched pair odds ratio = 18.0, CI = 4.2, 76.3). An average of 18 cases per year of *Salmonella arizona* were reported in the county between 1980 and 1987. In this investigation the majority of *S. arizona* cases reporting snake capsule ingestion had underlying illnesses such as acquired immunodeficiency syndrome (AIDS), diabetes, arthritis, cancer. The capsules were obtained primarily from Tijuana, Mexico and from Los Angeles, California pharmacies in Latino neighborhoods. Despite publicity and attempts to remove the capsules from sale in California, *Salmonella arizona* cases associated with snake-capsule ingestion continue to occur. (*Am J Public Health* 1990; 80:286–289.)

Introduction

In early 1987, two hospitals in Los Angeles County (California) reported four cases of *Salmonella arizona* (*Salmonella* subgroup 3, formerly *Arizona hinshawii*) infection in Latino patients to the Los Angeles County Department of Health Services. All patients gave a history of ingesting rattlesnake capsules prior to the onset of illness; in all four cases, culture of the rattlesnake capsules from the patients' supply or from their household grew *Salmonella arizona* and other enteric bacteria.[1] The patients had underlying chronic medical illnesses including acquired immunodeficiency syndrome (AIDS) and cancer, and the bacteria were isolated from extragastrointestinal sites (blood, pleural fluid, lymph node). We conducted an investigation to define further the epidemiology of rattlesnake-capsule ingestion and *Salmonella arizona* infections in Los Angeles County.

Methods

Surveillance data for *A. hinshawii* and *S. arizona* infections were reviewed for 1980–87. A matched case-control study was performed in late 1987 and early 1988. All Latino cases of *S. arizona* reported to the Los Angeles County Department of Health Services during 1986 and 1987 were eligible. Each case was matched to one or more persons with either *Salmonella typhimurium*, *Salmonella enteritidis*, or *Shigella*. *Salmonella* controls were chosen from 1986 and 1987 while *Shigella* controls were chosen from 1987. Cases were matched on race and age. Controls were at least 17 years old.

All cases of salmonellosis and shigellosis occurring in Los Angeles County are routinely administered a standard two-page questionnaire covering demographics, clinical information, and potential exposures. These data were supplemented by non-blinded administration of a telephone questionnaire which contained information regarding rattlesnake-pill ingestion, underlying illness, and folk medicine usage. Most interviews were conducted in Spanish by bilingual investigators. Serotype data for all *S. arizona* isolates submitted to the Los Angeles County Public Health laboratories during 1986–87 were reviewed.

Time of exposure was defined as the date to the nearest week, month, and/or year in some cases that the patient reported taking rattlesnake capsules. Since many of the patients were interviewed six months or more after their illness, they often had to estimate these dates. Estimated incubation period was defined as the interval between most recent exposure to rattlesnake pills and date of onset of illness or date of first positive culture, if illness onset date was unclear.

Unless otherwise specified, Mantel-Haenszel matched analysis was used to determine odds ratios and 95 percent confidence intervals (CI).

Results

An average of 18 cases a year of *S. arizona* were reported in Los Angeles County during 1980–87 (Figure 1). The number of cases ranged from nine in 1984 to 33 in 1987. In 1986–87, 45 (85 percent) of 53 such cases were Latino, while 208 (31 percent) of 673 cases with other common *Salmonella* serotypes were Latino (95 percent CI of proportion difference = 0.43, 0.65).

Twenty-two cases were interviewed with the supplemental questionnaire. Not surprisingly, we were able to contact more of the 1987 cases (18 of 29) than 1986 cases (four of 16). The age and geographic distributions of *S. arizona* cases not included were similar to those of interviewed cases. Matched controls included 12 *Salmonella* patients from 1986–87 and 12 *Shigella* patients from 1987. The pool of potential *Salmonella* controls in the proper age range was small. Age matching of cases and controls was ±10 years for 96 percent of the matches; the largest age difference was 14 years.

The median age of cases was 44 years with a range of 16 to 61 years. The male-to-female ratio of interviewed cases was 2.7:1 while that for all Latino *S. arizona* cases was 1.5:1. Cases lived throughout the County but were concentrated in neighborhoods within central Los Angeles. The birthplace or national background of rattlesnake capsule-exposed cases was predominantly Mexico (72 percent).

Eighteen (82 percent) of the 22 cases gave a history of rattlesnake-capsule exposure compared to two (8 percent) of

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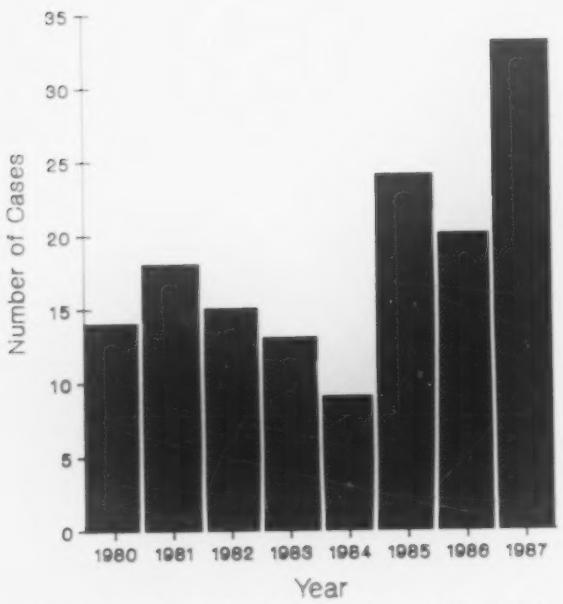

FIGURE 1—Cases of *Salmonella arizona* by Year, Los Angeles County, California 1980–87

24 matched controls (OR = 18.0, CI = 4.2, 76.3). The attributable fraction of *S. arizona* cases due to rattlesnake-capsule exposure in the Latino population is 77 percent.

Ten (56 per cent) of the 18 cases took fewer than 10 capsules over a period of a week or less. Five patients took only two or three pills. One patient ingested capsules daily for four months. The interval between exposure and onset of symptoms or first positive culture ranged from within a week to four years. Eleven (73 percent) of the 15 cases for whom an incubation period could be estimated became ill within three months. Two of the patients with longer estimated incubation periods had perirectal abscesses (Table 1). No correlation was observed between number of pills ingested and incubation period or site of infection.

The reasons patients used rattlesnake capsules included treatments for AIDS, arthritis, blood disorders, cancer, infections, itchy feet, sinus and skin conditions, and diarrhea. The majority of rattlesnake capsules were obtained from Tijuana, Mexico and from Los Angeles, California pharmacies. Patients also reported purchasing rattlesnake capsules in other Mexican border cities, in Central America, from a street vendor, at a local "swap meet" and from a friend. Capsules sold in Los Angeles cost about 20 cents per capsule. Forty-one percent of cases and 54 percent of the controls used other common folk medicines including yerba buena and other herbs and teas.

Feces, blood, and lung or pleural fluid were the most common sites of *S. arizona* isolation (Table 2). All three patients with multiple positive sites of *S. arizona* isolates had AIDS. The antibiogram of one isolate showed sensitivity to ampicillin and all other antibiotics tested.[1]

Fourteen (64 percent) of the 22 cases studied were hospitalized for *S. arizona*-related illness. Two patients had multiple hospitalizations for their infections. One other case was hospitalized for unrelated congestive heart failure. The remaining seven cases were managed as outpatients. The median stay for *S. arizona* cases hospitalized once was 17 days.

Cases with *S. arizona* infection were significantly more likely to have an underlying chronic illness than *Salmonella* controls (OR = 6.7, CI = 1.1, 42.1). The most common chronic conditions among cases included AIDS (five cases), diabetes (four cases), and connective tissue disease (three cases); one case had both diabetes and connective tissue disease, and one case each had heart disease, cancer, and an illness of unknown etiology. Five of six *S. arizona*-infected cases (83 percent) without underlying disease had the bacteria isolated from feces only compared to one of 16 (6 percent) infected chronically ill patients with such an isolation pattern (95 percent CI of proportion difference = 0.33, 1.0). In unmatched analysis, controlling for chronic illness, cases were more likely than *Salmonella* controls to have an extragastrointestinal site of infection (OR = 26.3, CI = 1.4, 500). Three of six *S. arizona* patients without underlying

TABLE 1—Summary of Four Latino *S. arizona* Cases with Estimated Incubation Periods Greater Than Three Months, Los Angeles County, 1986–87

Case No	Date of Exposure to Rattlesnake Capsules	Date of First Positive Culture	Site of Positive Cultures	Chronic Illness	Comment
7	March 1987	8.13.87	Lung, urine	AIDS	Patient diagnosed with AIDS January 1987. Because the patient had chronic fever, the date of onset with regard to *S. arizona* is unclear.
17	1982	3.26.87	Perirectal Abscess	Adult onset diabetes mellitus and rheumatoid arthritis	Perirectal abscess first developed in June 1986 and recurred. Patient gave no history of symptoms which could be related to *S. arizona* infection prior to this time.
20	Early 1986	12.12.86	Perirectal Abscess	None	The date of onset is unclear. Patient took rattlesnake capsules for four months.
21	1985	9.8.86	Feces	None	Patient was hospitalized for 12 days during August 1986 for spinal surgery and received prophylactic antibiotics. Patient developed diarrhea during the hospitalization.

TABLE 2—Sites of Positive Cultures for *Salmonella arizona* by Disease Status in 22 Latino Patients, Los Angles County, 1986–87

Site of Isolate	No. Cases (%)			
	Nomal Host (n = 6)	AIDS (n = 5)*	Other underlying conditions (n = 11)	Total (n = 22)
Feces	5 (83)	2 (40)	1 (9)	8 (36)
Blood		3 (60)	3 (27)	6 (27)
Lung or pleural fluid		2 (40)	2 (18)	4 (18)
Urine		1 (20)	2 (18)	3 (14)
Perirectal abscess	1 (17)		1 (9)	2 (9)
Wound			2 (18)	2 (9)
Lymph node		1 (20)		1 (5)

*One patient had positive blood and feces cultures, a second patient has positive lymph node, pleural fluid and feces cultures, and a third patient had positive lung and urine cultures.

illness (50 percent) were hospitalized with their infection.

Death occurred in six cases and one control. Three of the *S. arizona*-infected patients who died had AIDS. *S. arizona* infection was not listed as the direct cause of death in any of the cases. However, three patients had positive blood cultures for *S. arizona* within two months of their death and the infection appears to have exacerbated their condition.

Eighteen of 49 (37 percent) *S. arizona* isolates from the Latino cases were either serotype 3a 53:z_4,z_{23},z_{32}:-(n = 9) or 3a 21:z_{29}:-(n = 9) compared to none of 10 isolates serotyped from non-Latino patients in 1986 and 1987 (95 percent CI of proportion difference = 0.18, 0.57, Table 3). Both of these serotypes were cultured directly from rattlesnake capsules as were two other serotypes, 3a 61:1, v:1,5,7 and 3b 50:4:z, which were also isolated from both Latino and non-Latino patients. The 10 isolates of the latter two serotypes were all from feces, whereas 17 (94 percent) of the isolates of the former two serotypes were from extragastrointestinal sites.

Discussion

This investigation of Los Angeles County residents shows that human *S. arizona* cases are relatively common and that over three-fourths of recent *S. arizona* infections in Latinos are linked to rattlesnake capsules. Rattlesnake-capsule ingestion by persons with invasive *S. arizona* infection has been previously documented in case reports.[1–4] All of these cases were Latino patients who resided in the Southwest and had underlying disease.

Capsules cultured from *S. arizona* cases or households have been positive for *S. arizona* and other enteric bacteria.[1,2] The capsules are made of dried ground rattlesnake flesh and are known by several names including polvo de vibora, carne de vibora, or vibora de cascabel. The ingestion of snake preparations is an old popular folk remedy of Mexico and Central America and is used for a variety of medical conditions.[1–5] In this investigation, the majority of patients acquired the capsules from the border city of Tijuana, Mexico and from Los Angeles, California pharmacies or "farmacias" and "boticas" which serve the Latino community.

Reptiles constitute the major reservoir of *S. arizona*.[6–9] Other animal sources from which this bacterial species has been isolated are various fowl and rodents as well as opossums, goats, swine, dogs, and minks.[10,11] Foodborne outbreaks of *S. arizona* gastroenteritis have been reported associated with ice cream, cream pie, eggs, and egg powder.[12,13] Extragastrointestinal disease has been reported in patients with liver abscesses, septic arthritis, brain abscesses, pleural fluid infection, osteomyelitis, and cholecystitis.[12,14–16]

This surveillance study and case reports[1–4] indicate that underlying medical conditions are present in the majority of *S. arizona* cases who ingest rattlesnake capsules. Our findings suggest that *S. arizona* infections are at least as likely as other *Salmonella* serotypes to result in septicemia and focal

TABLE 3—*S. Arizona* Subgroup Serotypes by Ethnicity and Source of Isolation Los Angeles County, California 1986–87

Serotype	Number of Isolates				
	Latino		Non-Latino		
	Extragastrointestinal	Intestinal	Extragastrointestinal	Intestinal	Rattlesnake Capsules
G:z_4,z_{23}:-	1				
18:z_4,z_{23}:-	1				
18:z_4,z_{32}:-	1		1		
21:z_{29}:-	8	1			1
40:z_4,z_{23}:-	3				
40:z_4,z_{24}:-		1			
40:g,z_{51}:			2		
43:z_{24},z_{23}:-		1		2	
48:g,z_{51}:-				1	
48:z_{36}:-					1
48:l,z	2	1			
50:z_4,z_{24}:-	1				
50:k,z:z_{53}				1	
50:r,z		2		2	1
53:z_4,z_{23}:-	1				
53:z_4,z_{23},z_{32}:-	9				1
53:z_{29}:-					1
56:z_4,z_{23}:-	3	1			
60:r,e,n,x,z_{15}		3			
61:1,z		1			
61:1,v:1,5,7		5		1	1
65:(k):z	1				
O rough:z_4,z_{23}:-	2				
O rough:z_4,z_{23},z_{32}:-					1
TOTAL	33	16	3	7	7

infections in patients with significant underlying disease.[17–19] The predisposing disease process alone or in association with treatments such as corticosteroids, chemotherapy, radiotherapy and antibiotics can interfere with host-resistance mechanisms. Edwards, *et al*,[12] have suggested, on the basis of isolation site proportions, that *S. arizona* may be more invasive than other *Salmonella* species. Our analysis using *Salmonella* controls and controlling for chronic disease is consistent with this possibility as is the 50 percent hospitalization rate in cases without underlying illness. Further, our data on *S. arizona* serotype distribution by isolation site and that of Weiss, *et al*,[20] suggest that invasiveness may be serotype-specific. In both studies, serotype 61:1,v:1,5,7 stands out as relatively noninvasive.

Most of the patients in this study ingested fewer than 10 rattlesnake capsules. Riley, *et al*,[1] found 10^5 *S. arizona* organisms per gram in one sample. Capsules weigh an average of 700 mg and, thus, the infectious dose of *S. arizona* may be as low as 10^5 or 10^6 organisms. For the many patients with reduced resistance who take rattlesnake capsules, the infectious dose may be even lower.

Although the incubation period for *Salmonella* gastroenteritis is usually 24–36 hours, four study persons apparently were indolently infected for months to years before developing signs of illness or before positive cultures were obtained. Prolonged intestinal carriage of *Salmonella* bacteria is a well recognized phenomenon; chronic illness or other factors such as surgery or disruption of intestinal flora due to antibiotics may have triggered active infection in these patients.[21] Two persons, one with underlying illness and one a normal host, had perirectal abscesses which could have been present for some time but were undetected. While recall and interviewer bias is possible, we believe the histories of rattlesnake capsule ingestion are accurate because interviewers described or denied such exposure without hesitation.

Five cases who were exposed to rattlesnake capsules had an underlying condition of AIDS. Investigators at Los Angeles County/University of Southern California Medical Center have recently reported on four of these patients.[1] Human immunodeficiency virus (HIV) infection has been shown to predispose patients to serious *Salmonella* infections.[22,23] Salmonellosis in persons with AIDS is characterized by widespread infection, bacteremia, and relapse even with standard antibiotic therapy. Through September 1988, over 900 cases of AIDS in Latinos have been reported in Los Angeles County, and the proportion of Latino AIDS cases has increased 133 percent from 9 percent to 19 percent between 1982 and 1988.* Thus, the dangers of rattlesnake-capsule exposure should be emphasized to Latinos due to their increasing pool of AIDS victims and the propensity of some to use folk remedies.

Physicians and health care workers should be aware of the potential for *S. arizona* infection from ingesting rattlesnake capsules and the prevalence of this practice. The State of California Department of Health Services Food and Drug Branch has taken action to prevent further distribution of the products and to remove the capsules from pharmacies and markets known to distribute them. Distributors were also requested to recall the products. Perhaps because of these actions and publicity in both Spanish and English, only 12 *S. arizona* cases linked to rattlesnake capsule ingestion were reported in Los Angeles County in 1988. Continued occurrence of cases is not surprising given the availability of rattlesnake pills in local Latino neighborhoods and along the US-Mexico border. Health agencies and community groups should pursue education of the Latino population about the risks and absence of medical benefit from rattlesnake capsules.

*Lieb L. AIDS Epidemiology Program, Los Angeles County Department of Health Services, personal communication, 1988.

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Use of Medical Care for Chest Pain: Differences between Blacks and Whites

David S. Strogatz, PhD

Abstract: Data from a 1980, community-based survey of adult residents of Edgecombe County, North Carolina were analyzed to examine differences between Blacks and Whites in the reported use of medical care after experiencing chest pain. Of all adults (N=302) with chest pain in the year prior to interview, 49 percent of Blacks and 27 percent of Whites did not see a physician following the chest pain (difference = 22%, 95% CI = 12, 33). A multivariable analysis found that although the association between race and utilization was reduced at poverty levels of income, it was not explained by differences in demographic characteristics, health status or other dimensions of access to care. (*Am J Public Health* 1990;80:290–294.)

Introduction

Coronary heart disease (CHD) mortality rates for Black Americans exceed those of Whites at ages 25–64 and are similar to the rates of Whites for all ages combined.[1–3] In contrast, population rates of CHD occurrence based on cases from hospital discharge surveys are lower for Blacks than for Whites.[4,5] The inconsistency between the mortality and hospitalization data may be due to bias in detection and measurement, but it may also reflect an excess of CHD deaths among Blacks before the hospital is reached. A hospital surveillance study of CHD in Newark determined that Blacks were more likely to be dead on arrival at the hospital.[6] A study in urban and rural areas of South Carolina also reported that Blacks had higher rates of out-of-hospital mortality from acute myocardial infarction.[7] In community-based studies of sudden death, Blacks were found to be at greater risk than Whites in Charleston,[8] New Orleans,[9] and Nashville,[10] although not in Baltimore[11] where there was essentially no Black-White difference in the occurrence of sudden death.

Among those who do not die immediately, Blacks and Whites may differ by how soon medical care is received following the onset of symptoms. The National Ambulatory Medical Care Survey found that Blacks had fewer office visits for CHD and other diseases of the circulatory system.[12] Compared to Whites, Blacks are far less likely to be seen by a cardiologist, to have coronary angiography performed, and to undergo coronary artery bypass surgery.[12–15] These differences persist even among hospitalized patients, controlling for the clinical severity of disease.[14–17]

Less is known about Black-White differences in patients' knowledge of symptoms and decisions to seek care, an increasingly salient issue with the emergence of thrombolytic agents (e.g., streptokinase, tPA) for treatment in the early phase of myocardial infarction. A community survey on CHD conducted among Black residents of a southern US city found low levels of knowledge about CHD, its signs and symptoms.[18] In a comparison of Blacks and Whites living in Minneapolis-St. Paul, Blacks were less informed about the symptoms of myocardial infarction.[19] A study of attitudes about a variety of hypothetical symptoms found that Blacks and Whites had similar perceptions about how urgently to seek care for stable or unstable angina.[20] However, interviews with a consecutive series of Black patients hospitalized with acute myocardial infarction revealed that the average time between onset of symptoms and arrival at the hospital exceeded the times reported in similar investigations of White patients.[21] These preliminary findings indicate that gaps in knowledge and delays in receiving care may contribute to Black-White differences in pre-hospital mortality from CHD.

The purpose of this paper is to compare reported use of medical care by Blacks and Whites who experienced chest pain and to examine the degree to which demographic characteristics, access to medical care, and health status modified or accounted for Black-White differences in care-seeking behavior.

Methods

The data for these analyses were collected as part of the 1980 baseline survey of the Edgecombe County (North Carolina) High Blood Pressure Control Program. One thousand dwellings were randomly chosen, and interviews were sought with all members of the selected households who were 18 years of age or older. Data were collected from 2,029 individuals (1,085 White, 944 Black), representing an overall response of 91 percent. Additional information on the study population and procedures has been published.[22]

History of chest pain and associated use of medical care were measured by answers to a pair of questions from a larger set of similar items used to derive the symptom-response ratio, a need-based measure of utilization of health services.[23] During the interview, each individual was asked if he or she had experienced "repeated pains in or near the heart" at any time during the last 12 months and, if so, whether a doctor had been seen for it, in the past year or some time earlier. From this question, the 319 respondents (147 White, 172 Black) with chest pain in the past year were divided into three groups: 1) those who had seen a doctor for chest pain within the past year; 2) those who had seen a doctor for chest pain at some time, but not in the past year for this most recent episode; and 3) those who had never seen a doctor for their chest pain.

The baseline interview also included a series of questions concerning various barriers to medical care. As described in detail elsewhere,[24] these questions were used to measure four dimensions of access to care identified by Penchansky and Thomas[25]: *accessibility* (convenience of location of health services); *accommodation* (ease in getting an appointment); *affordability* (ability to pay for care); and *acceptability* (how well respondents get along with providers). Each dimension

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of access to care was represented by a structural score, a psychological score, or both. The structural access variables referred to rank ordering on tangible entities (e.g., distance to be traveled, time spent waiting, money or insurance at one's disposal), with a high score denoting good access. Psychological access referred to reported dissatisfaction on each dimension; scores on measures of psychological access represent the percent of individuals expressing dissatisfaction. The questions on access to care were answered by all persons who reported having a regular source of medical care. Fifteen of the 319 individuals with chest pain (3 White, 12 Black) did not have a regular source of care and therefore were not included in analyses involving these variables. Two other individuals had missing data on other variables of interest, leaving a total of 302 in the final analysis.

Other potential predictors of utilization which were measured at baseline and included in the analysis were age, sex, self-perceived health as measured by the Current Health Scale (CHS),[26] and reported history of myocardial infarction ("Have you ever had a heart attack?") and current antihypertensive treatment ("Are you now taking medicine for high blood pressure?")

The effect of race on the use of medical care for chest pain was initially characterized by an observed (unadjusted) difference between proportions with a corresponding confidence interval.[27] This measure conveys the potential impact of strategies to eliminate the association between race and utilization. Further analyses were conducted using linear multiple regression to estimate the Black-White difference adjusted for demographic characteristics, measures of health status, and dimensions of access to care. To highlight the contrast in use of care, these analyses were restricted to the two groups most different in utilization: individuals seen *during the past year* when chest pain occurred (group 1 defined above) vs individuals who were *not seen at all* for their chest pain (group 3). A backward stepwise procedure (with $p = .05$ to remain) was run to evaluate product terms combining race with each covariate.[27] Main effect terms for race and for all covariates were automatically included in the model. With the linear regression analysis, the coefficient for the variable representing race was equal to the adjusted difference between the percent seeking care for Whites and the percent seeking care for Blacks. Race-specific regression models were also computed to assess the predictive importance of the covariates.

Results

The use of medical care by adults after they experienced chest pain is summarized in Table 1. Almost half of all Blacks with repeated pain near the heart never saw or discussed this with a doctor, compared to 27 percent of Whites (difference between percentages = 22%, 95% CI: 12, 33).

TABLE 1—Association between Race and Use of Medical Care by Adults Experiencing Chest Pain in the Past Year: Edgecombe County, NC, 1980

Use of Medical Care for Chest Pain	Black		White	
	N	(%)	N	(%)
MD seen in past year	35	(22)	67	(47)
MD seen, not in past year	45	(28)	38	(26)
MD never seen	78	(49)	39	(27)
Total	158	(100)	144	(100)

Subsequent analyses examined the distribution and influence of the covariates in addition to the Black-White difference in utilization. These analyses were restricted to individuals who either had seen a physician for chest pain in the past year (35 Blacks, 67 Whites—top row of Table 1) or had never seen a physician for chest pain (88 Blacks, 41 Whites). The characteristics of these adults are shown in Table 2. In the overall comparison of Blacks and Whites with chest pain, Blacks were younger, appeared to be healthier (by self-perception and history of myocardial infarction), had worse structural access to their source of medical care, and (except for cost) had somewhat more dissatisfaction with their source of care.

In the unadjusted data of both Blacks and Whites, utilization in response to chest pain was associated with older age, worse perceived health, and a history of myocardial infarction or current antihypertensive therapy. In contrast, crude associations between dimensions of access to care and utilization of care appeared to differ by race. Among Whites with chest pain, those adults who sought care had better structural access and less dissatisfaction with care. Among Blacks, care seeking was not associated with psychological access and had an inverse relationship with two dimensions (affordability, accommodation) of structural access.

The results of the multivariable analyses are given in Table 3. For Black adults, physician visits for chest pain were independently associated with history of myocardial infarction, current antihypertensive treatment, and having experienced delays in the past when trying to make an appointment (low structural accommodation). These variables were related to utilization in the same direction for Whites, but were less pronounced. The only predictor of significance for Whites was worse self-perceived health. Older age and relative satisfaction with the appointment process (psychological accommodation) were also associated with physician visits by Whites.

The predictors from the race-specific models were similarly identified in the model combining Blacks and Whites. The only statistically significant interaction term was the product of race and structural affordability (an index which combines information on income and insurance[24]). The race-specific models show that structural affordability was a weak positive predictor of utilization for Whites and negative for Blacks. The impact of this interaction on the Black-White difference in utilization can be illustrated for selected values of structural affordability (where the difference between percent utilization by Blacks and percent utilization by Whites equals $0.128 - 0.052 \cdot$ structural affordability). Among poor individuals (e.g., annual family income $\leq$ $5,000, no health insurance: structural affordability = 4), Whites were more likely than Blacks to have seen a physician but the difference between the percentages was relatively small (8 percent). This difference between percentages of Whites and Blacks who saw a physician was 18 percent at the median value (6) for structural affordability in this sample. At a high level of resources for this community (e.g., annual family income = $20,000, insurance pays for a portion of office visit and medication costs: structural affordability = 10) the difference was 39 percent.

Discussion

These data showed the expected Black-White difference in the use of medical care for chest pain. This difference increased with greater structural affordability (i.e., greater

TABLE 2—Characteristics of Adults Experiencing Chest Pain in the Past Year*: Edgecombe County, NC 1980

Characteristics**	Black			White		
	Total (N = 113)	Care (N = 35)	No Care (N = 78)	Total (N = 106)	Care (N = 67)	No Care (N = 39)
Sex (% male)	38	37	38	43	42	44
Mean Age (years)	42 (1.8)	50 (3.2)	38 (2.0)	51 (1.5)	55 (1.7)	43 (2.4)
Health						
Mean CHS	26.2 (1.0)	22.5 (1.6)	27.9 (1.2)	24.7 (1.0)	22.2 (1.3)	29.1 (1.6)
History of MI (% MI)	10	29	1	20	28	5
Treatment for HBP (% RX)	21	40	13	24	31	10
Mean Structural Access						
Affordability	6.1 (0.2)	5.7 (0.3)	6.2 (0.3)	7.7 (0.3)	8.0 (0.3)	7.3 (0.4)
Accommodation	7.3 (0.2)	6.9 (0.3)	7.5 (0.2)	7.5 (0.2)	7.7 (0.2)	7.1 (0.3)
Accessibility	10.8 (0.2)	10.9 (0.3)	10.7 (0.2)	11.5 (0.1)	11.6 (0.2)	11.3 (0.3)
Psychological Access (% dissatisfied)						
Affordability	53	51	54	58	51	72
Accommodation	48	46	49	41	30	59
Acceptability	51	49	49	41	36	49

*Restricted to (1) adults seen by a physician for chest pain during the past year and (2) adults never seen for chest pain.
**Mean and standard error reported for continuous variables, percent reported for dichotomous variables.

TABLE 3—Linear Regression Coefficients for the Multivariable Analysis of Race and Use of Medical Care by Adults Experiencing Chest Pain in the Past Year*: Edgecombe County, NC, 1980

Variables	Black		White		Overall	
	β	SE	β	SE	β	SE
Sex	0.006	0.085	0.044	0.088	0.034	0.060
Age	0.001	0.003	0.006	0.003	0.003	0.002
Health						
CHS	−0.004	0.004	−0.009	0.005	−0.006	0.003
History of MI	0.585	0.143	0.148	0.125	0.333	0.091
Treatment for HBP	0.253	0.106	0.145	0.107	0.195	0.074
Structural Access						
Affordability	−0.026	0.019	0.024	0.018	0.080	0.038
Accommodation	−0.048	0.024	−0.017	0.027	−0.037	0.017
Accessibility	0.013	0.023	0.013	0.032	0.015	0.018
Psychological Access						
Affordability	−0.022	0.081	−0.104	0.094	−0.067	0.060
Accommodation	−0.074	0.085	−0.179	0.110	−0.139	0.067
Acceptability	0.045	0.083	−0.028	0.092	0.021	0.059
Race	—	—	—	—	0.128	0.184
Race × Structural Accessibility	—	—	—	—	−0.052	0.025

*Restricted to (1) adults seen by a physician for chest pain during past year and (2) adults never seen for chest pain.

family income and health insurance). Interpretation of these results requires recognition that measures of socioeconomic status may not have the same meaning and implications for Blacks and Whites.[28] For example, Blacks generally have had less opportunity than Whites to translate educational achievement into better jobs, more stable employment, and higher income,[29] so comparisons of Blacks and Whites with the same years of education can be misleading. Similar problems arise when comparing Blacks and Whites at the same level of income without adjustment for differences in family size, purchasing power, and property, all of which would tend to accentuate racial inequality in income.[30] The Black-White gap in utilization at higher levels of structural affordability in part may reflect differences in economic resources which may not be reflected by family income. These problems do not explain the inverse relationship between structural affordability and utilization among symptomatic Black adults. It may be a chance association in these data. Alternatively, social support is directly related to economic resources in this population[31]; perhaps the more economically advantaged Blacks rely more on social support networks for advice on health. Untapped dissatisfaction with past medical visits may also be reflected in lower utilization by those individuals with somewhat more income or insurance. As others have reported, the elimination of racial differences in the use of medical care will require more than just modifying the ability to pay for services.[32,33]

Other possible explanations for the Black-White difference in utilization include differences in perceptions about the seriousness of the symptom and the potential efficacy of medical care. Compared to Whites, Blacks may be less likely to perceive chest pain as serious enough to warrant immediate medical attention. Berkanovic and Telesky[34] suggest that the pronounced association between severity of symptoms and utilization for Blacks may be a vestige of past circumstances in which only the most disabling conditions could serve as justification for interruption of one's duties in order to seek care. This habit may persist today even for individuals who have more resources or autonomy over their personal lives. Studies of Whites with chest pain suggest that

both the severity of the symptom and an individual's flexibility to respond at the time of symptom onset will contribute to delay in receiving care.[35,36]

Blacks and Whites also may view the likely efficacy of visiting the doctor differently. Perceived efficacy, an important predictor of care-seeking by symptomatic individuals,[37,38] was different in this population when treatment for hypertension was assessed.[24] Hypertensive Black adults were found to have less confidence in the net benefit of anti-hypertensive medication, and this attitude was associated with a greater likelihood of being untreated for diagnosed hypertension.

There are several limitations in these data. First, the question used to signify chest pain may not be measuring ischemic pain, and the meaning may vary by race. In Blacks and Whites, self-reported chest pain was associated with history of myocardial infarction and with established predictors of CHD (age, hypertension, and cigarette smoking), suggesting some common features of the measurement of the symptom. The instrument for the symptom-response ratio also included separate questions on indigestion and abdominal pain to avoid misclassification of chest pain in respondents with gastrointestinal symptoms.

Another problem is that the perceptions about health or access to health care may have been a product, as well as a determinant, of use of health services. Any resultant overestimation of the association between covariates and utilization (either because of selective recall or the influence of utilization on perceptions) would probably lead to an underestimate of the residual association between race and utilization. Despite this possibility, substantial Black-White differences were found in the multivariable analysis.

These findings require confirmation, e.g., in individuals from a defined population who present to the emergency room with acute myocardial infarction. The sequence of perceptions and behaviors from initial symptom to arrival at the hospital should be characterized. To the extent possible, similar data should be collected from witnessed instances of fatal CHD. Such a study was conducted 20 years ago in a White population[39]; replication in a Black or biracial community would be informative. The knowledge of symptoms of CHD also should be measured in community-based surveys related to cardiovascular disease.

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A special award created by friends of Fred L. Soper, Director of the Pan American Health Organization (the World Health Organization Regional Office for the Americas) from 1947 to 1958, has been established in recognition of his outstanding contributions to health in the Americas.

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The award will be presented annually to the author or authors of an original scientific contribution comprising new information on, or new insights into, the broad field of public health, with special relevance to Latin America or the Caribbean or both. This may consist of a report, an analysis of new data, experimental or observational, or a new approach to analyzing available data. Preference will be given to studies involving more than one discipline and to papers related to infectious disease, a life-long concern of Dr. Soper. Review papers are eligible if their conclusions deal directly with policy primarily affecting Latin America or the Caribbean.

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Beliefs about AIDS, Use of Alcohol and Drugs, and Unprotected Sex among Massachusetts Adolescents

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Abstract: In August 1988, 1,773 Massachusetts 16–19-year-olds were surveyed by telephone using anonymous random digit dialing; response rate 82 percent. Logistic regression tested whether alcohol and drug use, perceived susceptibility to human immunodeficiency virus (HIV), severity of HIV if infected, effectiveness of condoms in preventing infection, barriers to condom use, and behavioral cues such as exposure to media or personal communication about acquired immunodeficiency syndrome (AIDS) were independently related to condom use. Among sexually active respondents, (61 percent of those interviewed) 31 percent reported always using condoms. Respondents who believed condoms are effective in preventing HIV transmission and worried they can get AIDS were 3.1 and 1.8 times, respectively, more likely to use condoms all the time. Respondents who carried condoms and who had discussed AIDS with a physician were 2.7 and 1.7 times, respectively, more likely to use them. Those who believed condoms do not reduce sexual pleasure and would not be embarrassed if asked to use them were 3.1 and 2.4 times, respectively, more likely to use condoms. Teens who averaged five or more drinks daily or used marijuana in the previous month were 2.8 and 1.9 times, respectively, less likely to use condoms. Among respondents who drink and use drugs, 16 percent used condoms less often after drinking and 25 percent after drug use. Those counseling adolescents about HIV should assess and discuss beliefs outlined in the Health Belief Model, as well as their alcohol and drug use. (*Am J Public Health* 1990; 80:295–299.)

Introduction

One-fifth of reported AIDS (acquired immunodeficiency syndrome) cases in the United States have been in the 20–29 year age group.[1] Given the incubation period, many of those persons were infected during their high school years or shortly thereafter.

Knowledge of behavioral risks for AIDS does not prompt all at risk to change their behavior.[2] Most older teens in Massachusetts have learned the principle modes of human immunodeficiency virus (HIV) transmission, and adolescent condom use has increased since 1986.[3,4] However, two-thirds of sexually active 16–19 year olds surveyed there in the fall of 1988 reported sexual intercourse in the past year without the use of condoms.[4] Nationally, teenagers account for one-fourth of sexually transmitted disease cases.[5]

Educators need to move beyond imparting knowledge about modes of HIV transmission to change adolescent behavior. A specific set of beliefs described in the Health Belief Model has been found to predict behaviors to avoid other health risks,[6] and has been hypothesized to also predict whether someone will adopt behavior to avoid HIV transmission.[7] Persons who believe they are personally susceptible to HIV exposure, the consequences of exposure are severe, protective measures such as condom use are effective in preventing transmission, and perceive few barriers to the adoption of condom use may be more likely to adopt such behavior to avoid HIV transmission. According to this model, people make a rational cost-benefit analysis when trying to decide whether to adopt preventive behavior. Actual changes in behavior may then be stimulated by cues to action such as educational messages or learning that someone they know has AIDS.

However, among adolescents, sexual encounters may be unplanned and also often occur after drinking or drug use. Exploratory studies in San Francisco indicate gay men are more likely to have unprotected intercourse after alcohol and drug use.[8] Whether this applies to heterosexuals warrants exploration. Because these and other factors may mitigate against adolescents making a cost-benefit analysis about HIV and condom use before each sexual encounter, it is important to assess whether the beliefs described in the model do indeed predict which adolescents will use condoms and, if so, which beliefs are the strongest independent predictors of use. It is also important to assess whether alcohol and drug use are negatively related to condom use and, if so, why.

This Massachusetts statewide study of adolescents assessed:

- whether beliefs outlined in the Health Belief Model are significantly associated with condom use among sexually active adolescents;
- which beliefs are the strongest independent predictors of condom use after controlling for social and demographic characteristics of respondents;
- whether condom use is less likely after drinking and drug use; and
- whether those who use condoms less often after drinking and drug use have different characteristics and beliefs about HIV transmission.

Methods

In September 1988, a statewide anonymous random digit dial telephone survey of 16-to-19-year-olds was conducted in Massachusetts using methods developed by Waksberg.[9] Within each household, one adolescent was randomly selected using methods developed by Kish.[10] The survey of 1,773 adolescents (response rate 82 percent) was conducted in English and Spanish and explored respondent knowledge and beliefs about HIV transmission; alcohol, marijuana, and other illegal drug use; and behavior change in response to the AIDS epidemic. The study specifically asked respondents beliefs that are identified in the Health Belief Model (see Appendix for details).

In addition, respondents were asked their age, level of education, gender, whether their sexual activity was with males, females or both, and the number of sexual partners

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they had in the past year. Chi square analyses tested whether individual beliefs outlined in the Health Belief Model and alcohol and drug use were associated with condom use. Condom use was classified as: never used condoms, use condoms some of the time, always used condoms. Stepwise logistic regression analysis[11] tested which beliefs were the strongest independent predictors of condom use after considering demographic characteristics, alcohol and drug use, and numbers of sexual partners, and whether alcohol and drug use were independently related to condom use. Condom use was dichotomized as always used condoms versus sometimes or never used. Odds ratios and confidence intervals were calculated. The chi square test for symmetry[12] assessed whether individuals were less likely to use condoms after drinking and drug use. Finally, chi square analyses tested for differences in characteristics and beliefs of adolescents who used condoms less often after drinking or drug use.

Results

Bivariate Analysis

Condom use according to respondent characteristics—Among the 1,773 adolescents interviewed, 61 percent (n = 1,050) reported having had intercourse in the previous year. Two percent of males reported homosexual behavior. Among sexually active teens, 31 percent (n = 320) reported always using condoms, 32 percent (n = 345) sometimes, and 37 percent (n = 385) never used condoms. Males were more likely than females to report always using (34 percent vs 26 percent). Thirty-nine percent of 19-year-olds compared to 29 percent of 16-year-olds were more likely to always use condoms. Thirty-one percent of Whites, 28 percent of Blacks, and 39 percent of Hispanics, but only 7 percent of persons from other racial backgrounds, always used condoms. Condom use varied by previous numbers of sexual partners (Table 1). Respondents who reported only one partner in the last year were much more likely to never use condoms than those who had more partners. However, respondents who reported 10 or more partners were the least likely to always use condoms.

TABLE 1—Condom Use Among Sexually Active Massachusetts Teenagers According to Numbers of Sexual Partners

Number of Sexual Partners (past year)	% Never Use Condoms N = 380	% Sometimes Use Condoms N = 337	% Always Use Condoms N = 316	
1 (N = 549)	46	26	29	
2–4 (N = 370)	29	35	36	(p<.0001)
5–9 (N = 85)	22	52	26	
10+ (N = 28)	8	75	17	

Adolescents who averaged five or more drinks daily were somewhat less likely to always use condoms than abstainers (29 vs 35 percent) as were persons who smoked marijuana at least weekly (22 vs 32 percent of abstainers). Persons who used other psychoactive drugs in the past month were also less likely to always use condoms than those who did not (20 vs 32 percent).

Condom use according to respondents' health beliefs—On most dimensions of the Health Belief Model, bivariate associations with condom use were observed. Respondents were more likely to always use condoms if they felt susceptible to AIDS; believed condoms are effective; perceived few barriers to condom use; and were exposed to more cues to action (Tables 2–4).

Multivariate Analysis

Health beliefs—We used stepwise logistic regression analysis to assess which beliefs were the strongest independent predictors of whether sexually active adolescents always used condoms (Table 5). Variables under barriers to use were the strongest predictors of condom use. Perceived

TABLE 2—Proportion of Massachusetts Adolescents Who Always Use Condoms According to Their Perceived Susceptibility to HIV and AIDS (N = 1,050)

Perceived Susceptibility	N	% Always Use Condoms	Relative Risk*	(95% C.I.)*
How likely is a female to acquire the AIDS virus during intercourse with an infected male?				
Very likely	(749)	30	9.44	(1.37,65.09)
Somewhat likely	(270)	34		
Not likely/a little likely	(31)	3		
How likely is a male to acquire the AIDS virus during intercourse with an infected female?				
Very likely	(688)	30	1.70	(1.01,2.88)
Somewhat likely	(293)	35		
Not likely/a little likely	(68)	18		
How much do you worry you could get AIDS?				
A great deal	(161)	47	1.81	(1.46,2.26)
Somewhat	(357)	31		
A little/not at all	(532)	26		
Perceived Severity				
Likelihood an infected person will die of AIDS?				
Very	(868)	31	53	(.32,.86)
Somewhat	(170)	27		
Not at all/a little	(12)	58		
Is there a cure for AIDS?				
No	(1,035)	31	4.62	(.69,30.78)
Yes	(14)	7		

*When a variable has more than two categories the extreme categories were compared.

TABLE 3—Proportion of Sexually Active Massachusetts Adolescents Who Always Use Condoms According to Their Perceived Effectiveness and Barriers to Use (N = 1,050)

Effectiveness	N	% Always Use Condoms	Relative Risk*	(95% C.I.)*
Perceived Effectiveness of Condoms in Reducing HIV Transmission				
Very extremely	(693)	31	5.50	(1.43,21.24)
Somewhat	(321)	31		
Not at all/a little	(35)	6		
Barriers to use				
Carry condoms				
Yes	(267)	49	2.06	(1.73,2.45)
No	(783)	24		
Condoms reduce pleasure				
Not at all true/a little	(484)	40	3.19	(2.16,4.71)
Somewhat true	(367)	27		
Very true	(189)	13		
Condoms are difficult and embarrassing to obtain				
Not at all true	(384)	31	1.29	(.92,1.82)
Somewhat true/a little	(537)	31		
Very true	(128)	24		
Would be embarrassed if a partner asked condoms to be used				
Not at all true	(798)	33	2.06	(.98,4.31)
Somewhat true/a little	(208)	23		
Very true	(37)	15		

*When a variable had more than two categories the extremes were compared.

effectiveness of condoms, and how much respondents worried about acquiring AIDS also independently predicted use. Also, adolescents who spoke to physicians about AIDS were more likely to use condoms.

Of the demographic and behavioral variables entered into the same regression analysis, age predicted always using condoms, with younger persons more likely to use condoms. Race was also predictive of use, with Whites more likely than Blacks or other non-Whites to use condoms. Independent of beliefs, number of sexual partners, education, and gender were not related to condom use.

Drinking and drug use—Adolescents who drank five or more drinks daily (5 percent of sexually active adolescents)

TABLE 4—Proportion of Sexually Active Massachusetts Adolescents Who Always Use Condoms According to Their Exposure to Cues to Action (N = 1,050)

Cues to Action	N	% Use Condoms	Relative Risk	(95% CI)
Know someone with AIDS				
Yes	(57)	16	.50	(.27,.92)
No	(993)	31		
Discussed AIDS with friends				
Yes	(920)	32	1.48	(1.05,2.08)
No	(130)	21		
Discussed AIDS with Physician				
Yes	(161)	46	1.66	(1.36,2.02)
No	(889)	28		
Media Exposure to AIDS information				
Yes	(701)	32	1.21	(.99,1.49)
No	(349)	27		
Teacher discussed about AIDS				
Yes	(778)	31	1.10	(.86,1.40)
No	(197)	28		

TABLE 5—Logistic Regression of Behaviors and Beliefs Associated with Consistent Condom Use among Sexually Active Massachusetts Adolescents

Health Belief Variable	Odds Ratio	(95% CI)
Susceptibility-Severity		
Worry can get AIDS (great deal vs not at all)	1.82	(1.13,2.92)
Effectiveness		
Condoms effective in preventing HIV transmission (extremely vs not)	3.12	(1.37,7.14)
Barriers to Use		
Condoms reduce pleasure (not true vs very true)	3.13	(1.94,5.04)
Carry condoms (yes vs no)	2.73	(1.92,3.89)
Embarrassed if asked to use condom (not vs very)	2.36	(1.16,4.79)
Cues to Action		
Discussed AIDS with Physician	1.67	(1.13,2.47)
Other Characteristics and Behaviors		
Average daily drinks (0 vs 5+)	2.77	(1.42,5.41)
Days smoked marijuana (0 vs 5+ past month)	1.86	(1.06,3.25)
Age (16 vs 19 years)	2.48	(1.56,3.94)
Race		
White vs Black	2.13	(1.25,3.64)
White vs Other	2.91	(1.02,8.27)

and those who used marijuana in the past month (29 percent of sexually active adolescents) were 2.8 and 1.9 times, respectively, less likely to use condoms. To further assess whether drinking and drug use independently influenced condom use, we asked respondents if they had intercourse in the past six months after drinking and after drug use. Those who had intercourse were then asked how often they used condoms when having sex after drinking and after drug use, and how often they used condoms when they had not been drinking or taking drugs.

Sixteen percent of adolescents who said they had sex after drinking reported using condoms less often after drinking than when not drinking (73/465) (Table 6) and 25 percent who said they had sex after drug use reported using condoms less often after using drugs (40/160) (Table 7).

Compared to other sexually active adolescents who drink, those who use condoms less often after drinking were significantly more likely to believe HIV sexual transmission, if one partner is infected, is only a little likely from males to females (10 vs 2 percent), females to males (19 vs 2 percent), or between males (5 vs .1 percent). They were also more likely to believe it is very true that condoms reduce pleasure (27 vs 15 percent), to have sex after drug use (45 vs 28 percent), and to have had 10 or more sexual partners in the past year (11 vs 4 percent).

Compared to other sexually active adolescents, those

TABLE 6—Condom Use When Drinking and not Drinking among Sexually Active Massachusetts Teenage Drinkers

	Condom Use When not Drinking					
	Always		Sometimes		Never	
Condon Use When Drinking	N	(%)	N	(%)	N	(%)
Always	194	(42)	17	(4)	8	(2)
Sometimes	40	(9)	140	(30)	11	(2)
Never	4	(1)	29	(6)	22	(5)

Use condoms less often after drinking: 73/465, 16%

TABLE 7—Condom Use When Taking Drugs and not Taking Drugs among Massachusetts Teenagers Who Use Drugs

Condom Use When Taking Drugs	Condom Use When not Taking Drugs					
	Always		Sometimes		Never	
	N	(%)	N	(%)	N	(%)
Always	43	(27)	1	(1)	—	—
Sometimes	25	(15)	61	(37)	15	(9)
Never	—		15	(9)	—	—

Use condoms less often after taking drugs 40/160, 25%

who used condoms less often after other psychoactive drug use also more often believed it very true that condoms reduce pleasure (34 vs 16 percent). They tended to be younger (45 vs 21 percent age 16), more likely to have dropped out of school (25 vs 8 percent), and much more likely to believe HIV is transmitted in ways it is not, e.g., by sharing eating and drinking utensils, and shaking hands (both 9 vs 1 percent).

Discussion

Telephones are owned by over 95 percent of households in Massachusetts, but low-income racial/ethnic minority households are the least likely to own phones. Because low-income households are at the highest risk of AIDS, this survey may slightly overestimate respondents' knowledge about AIDS and underestimate prevalence of behaviors that pose risk of HIV transmission.

A study of adults in Montreal found beliefs in the Health Belief Model predicted safer sex practices among adults.[3] This study suggests that some beliefs about HIV transmission and AIDS identified in the Health Belief Model predict whether adolescents will use condoms. Concern about acquiring AIDS, beliefs that condoms are effective in preventing HIV transmission, and speaking to a physician about AIDS predicted use. Barriers to use such—i.e., beliefs that condoms reduce pleasure, are embarrassing to use, or that respondents do not routinely carry condoms—were also highly predictive of not using condoms.

Variables concerning perceived severity of HIV infection were not, however, significant independent predictors of use. Although bivariate analyses revealed these variables were related to condom use, the overwhelming majority of respondents believed there is no cure for AIDS, and that infected persons will die from AIDS. Consequently, those variables did not explain much variation in condom use.

Beliefs about AIDS accounted for approximately one-tenth of variation in use. While a sizable fraction, this means that numerous other issues relate to use. In this study, use was less among heavy drinkers and frequent drug users suggesting the limits of rational cost-benefit beliefs in determining condom use when people are intoxicated.

A study of adolescents in Scotland found that respondents' first sexual intercourse was more likely to have occurred without the use of a condom if they had been drinking.[14] In this Massachusetts study, even among the same individuals, use of condoms were less likely after drinking and drug use suggesting that concerns about condom use may seem less pressing when people are intoxicated.

Many other factors not explored in this survey and not directly related to health beliefs about condoms and AIDS or HIV may also influence whether adolescents use, or will use, condoms. These include the ability to discuss contraception with sexual partners; perceptions of which partner's responsibility it is to purchase and use contraceptives; beliefs about abortion; concerns about other sexually transmitted diseases; beliefs and desires for pregnancy; beliefs about the efficacy of condoms relative to other contraceptives; issues of ambivalence about being prepared for sexual intercourse; influence of peers and perceptions about whether condom use is increasing among peers; advice received about family planning; perceptions of medical care providers and the adequacy or sensitivity of their advice; and cultural and religious beliefs. Beliefs and preferences of partners on all the issues cited above could also influence condom use. Also, although 77 percent of adolescents reported that they would not be embarrassed if a partner asked that condoms be used, this was an abstract response to a question most had never been asked. If one sex partner asks another to use a condom, questions of trust and fidelity could easily surface for both partners.

Nonetheless, this study suggests that the Health Belief Model provides a useful set of beliefs about AIDS to explore among adolescents and to target in efforts to increase condom use. Not only should educators try to change beliefs through education about personal susceptibility to the virus,[10] they should try to heighten perceived effectiveness of condoms by teaching adolescents which types of condoms offer the most protection, and how to avoid mistakes in using condoms that could reduce their effectiveness. Issues of embarrassment should be addressed possibly by role playing of discussion with sexual partners about AIDS and condoms, and efforts could be undertaken to make condoms more easily accessible to both male and female adolescents.

Finally, alcohol and drug use may reduce the likelihood that adolescents will use condoms. The array of educational, counseling, and legal approaches to reduce adolescent alcohol and drug consumption may thus have a particularly important influence on HIV transmission.

APPENDIX

Questions Used in Massachusetts Survey of Beliefs, Knowledge about AIDS HIV and Behavior

1) *Susceptibility*
- How much do you worry that you could get AIDS in your lifetime? (A great deal, Some, A little, Not at all.)
- How likely is it that if a man and woman have sex, and the man is infected with HIV, that the woman will become infected? (Very, Somewhat, A little, Not at all.)
- How likely is it, if the woman is infected, the man will become infected? (Very, Somewhat, A little, Not at all.)
- If two men have sex and one is infected how likely is it the other will become infected? (Very, Somewhat, A little, No at all.)

2) *Severity*
- Is there a cure for AIDS? (Yes, No)
- How likely is it that someone with AIDS will die from the disease? (Very, Somewhat, A little, Not at all.)

3) *Effectiveness*
- How effective are condoms in preventing transmission of AIDS virus? (Extremely, Very, A little, No at all effective.)

4) *Barriers to Action*
- How true do you think it is that:
  - condoms are difficult and embarrassing to obtain (Very, Somewhat, A little, Not at all)
  - condoms reduce pleasure (Very, Somewhat, A little, Not at all)
- If your partner wanted a condom to be used during intercourse, how likely is it that:
  - you would be embarrassed (Very, Somewhat, A little, Not at all)
  - you would be upset with your partner (Very, Somewhat, A little, Not at all)

- Do you usually carry condoms with you? (Yes, No)

5) *Cues to Action*

- Did you read or hear anything about AIDS, in the past week, in the newspaper, magazine, radio, or T.V.? (Yes, No)
- Have you ever discussed AIDS with a parent or other adult member of your family? (Yes, No)
- Have any of your teachers in school every talked about AIDS? (Yes, No)
- Have you ever discussed AIDS with a physician (Yes, No)
- Do you know anyone with AIDS? (Yes, No)

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AAFP and CDC Recommend Second MMR Vaccination at Preschool Age

The American Academy of Family Physicians (AAFP) recommends routine second measles, mumps and rubella (MMR) immunizations for children four to six years of age, or just prior to entering school. The national association representing more than 68,000 family doctors, residents, and medical students also said that children in areas where there is a high risk of contracting measles should receive their first MMR at 12 months, instead of 15 months of age. The revised guidelines calling for two childhood measles vaccinations concur with the Immunizations Practice Advisory Committee's report: *Measles Prevention*, issued recently by the federal Centers for Disease Control (CDC). The AAFP revised its schedule for MMR vaccinations because of the increasing number of infants, children and adolescents who have contracted measles. About five out of every 100 children previously immunized failed to develop protection against measles.

The new guidelines define a high-risk area as a county or portion of a county with:

- more than five measles cases among preschool-aged children during each of the last five years;
- a recent outbreak among unvaccinated preschool-aged children; or
- a large urban population.

CDC also recommends a second MMR shot or a measles immunity test for everyone who is going to college or other educational institutions beyond college, as well as medical personnel beginning employment who will have direct patient care contact.

Persons who are *not recommended* to receive a measles vaccination generally include pregnant women, persons with active tuberculosis, or those who have allergies to eggs.

The Growing Proportion of Female Physicians: Implications for US Physician Supply

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Abstract: This study analyzes how the growing proportion of women in the United States physician population will affect the amount and type of physician services available to the US population. Female physicians work fewer hours per week, are slightly less likely to be in patient care, and tend to enter different specialties than male physicians. Female physicians also have higher retirement rates than male physicians, but due to their lower mortality rates, have work lives nearly as long as male physicians. We examined how the changing composition of the physician population will affect the availability of physician services by comparing historical and projected trends for the number of active post-residency physicians with comparable trends for a full-time-equivalent measure of physician supply. The full-time-equivalent measure takes into account the different labor supply behavior of key subpopulations (e.g., women and graduates of US versus foreign medical schools). The results suggest that the changing composition of the physician population will reduce the growth of effective physician supply between 1986 and 2010 but only by four percentage points. (*Am J Public Health* 1990; 80:300–304.)

Introduction

The proportion of United States physicians who are women has grown rapidly since 1970 and, according to recent projections,[1] it will continue to rise until the year 2010. Since the practice characteristics of female physicians differ from those of male physicians, the growing representation of women in the medical profession is likely to have significant effects on the future supply of physicians.

In general, women physicians have been disproportionately represented in pediatrics and psychiatry and have been underrepresented in the surgical specialties.[2,3] Analyses of the most recent cohorts of new physicians, however, show that the specialty distributions of male and female physicians appear to be converging.[4] This convergence is much more pronounced among graduates of US medical schools (USMGs) than graduates of foreign medical schools (FMGs).[5] Nevertheless, there are still significant differences in the specialty choices of male and female US medical students.[6]

Numerous studies have documented that female physicians work fewer hours per week than male physicians,[7–11] and several analyses have explored the extent to which this difference is attributable to marital status and household responsibilities.[12,13] Longitudinal data show that the differences in the number of hours male and female physicians work may be decreasing, although significant differences still remain.[14] Because women physicians work fewer hours per week than male physicians and are comprising an increasing proportion of the physician population, some analysts have concluded that current projections of the physician population overstate effective future physician supply.[15,16] Partly in response to this issue, a recent report of the US Council on Graduate Medical Education[17] indicated that the practice patterns of male and female physicians deserve greater research. This paper measures current differences between male and female physicians and traces their consequences for future physician supply.

Methods

We used the Physician Masterfile[2] of the American Medical Association (AMA) for both current and historical data on all US physicians (both AMA members and nonmembers). We used the Demographic Model of the Physician Population[1] to project the size and composition of the future physician population.

Our projection of the physician population assumed that:

- the proportion of women among graduates of US medical schools will increase 1 percent per year from 32 percent in 1986 until it reaches 40 percent and will remain constant thereafter;
- male and female physicians will choose specialties following patterns identical to the most recent cohorts of medical school graduates; and
- retirement and mortality rates for male and female physicians will remain constant at their current levels.

We analyzed data from the AMA Socioeconomic Monitoring System[11] to examine practice differences of male and female physicians. These data come from a series of periodic telephone surveys of random samples of non-federal, postresident, patient care physicians since 1982. We used data from the 1987 core survey, which has data on approximately 4,000 physicians.

Results

Demographic Characteristics

Table 1 shows that the proportion of women physicians increased from 7.1 percent to 15.3 percent between 1970 and 1986, and is projected to reach almost 30 percent by the year 2010. Consequently, women physicians are concentrated in the younger age categories. In 1970 and 1986, female physicians were disproportionately represented among foreign medical graduates not born in the US, but the relationship between sex and country of graduation reverses during the projection period due to the growing number of females graduating from US medical schools. Female physicians are concentrated in pediatrics and psychiatry and are underrepresented in general surgery and the surgical subspecial-

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TABLE 1—Proportion of Active Physicians who are Female According to Age, Specialty, and Country of Graduation for Years 1970, 1986, 2000, and 2010

	Historical		Projected	
	1970	1986	2000	2010
Total	7.1%	15.3%	24.1%	29.4%
Country of Graduation				
USMG	5.4	14.0	24.7	30.7
FMG	14.6	20.0	21.7	23.0
USFMG	4.2	10.7	13.0	14.0
Foreign-born FMG	16.0	21.7	24.0	25.9
Age (years)				
< 36	9.5	24.5	36.5	37.3
36–45	6.4	15.6	30.3	37.1
46–55	6.3	9.5	20.5	30.9
56–65	5.0	7.1	12.8	21.4
> 65	5.4	5.9	6.8	13.8
Specialty				
General/Family Practice	4.4	12.9	22.9	28.8
General Internal Medicine	5.8	17.4	26.5	31.5
Medical Subspecialties	5.0	10.2	18.4	22.8
General Surgery	1.1	5.8	10.6	14.0
Surgical Subspecialties	1.3	3.9	6.8	8.9
Pediatrics	21.4	35.3	47.5	54.1
Obstetrics/Gynecology	7.2	19.6	35.9	45.6
Radiology	5.3	12.1	19.7	24.4
Psychiatry	13.2	22.4	34.0	41.8
Anesthesiology	14.3	17.4	23.2	26.6
Pathology	12.9	21.8	29.2	34.7
Emergency Medicine	—*	12.9	20.5	24.3
Other Specialties	9.6	18.1	26.4	31.7

SOURCE: AMA Physician Masterfile, 1970–86; Department of Data Release Services, Division of Survey and Data Resources, AMA. Demographic Model of the Physician Population, AMA Center for Health Policy Research, 1988.

ties. These patterns appear in both the 1970 and 1986 data as well as the projection estimates for 2000 and 2010.

Differences in the specialty distributions of male and female physicians persist but are gradually weakening, however. The index of dissimilarity for the specialty distributions of male and female post-resident physicians (i.e., the percent of female physicians who would have to change specialties so that their specialty distribution would match that of male physicians) dropped from 31.2 to 23.7 between 1970 and 1986, indicating that the concentration of female physicians in certain specialties has decreased. The index calculated only for physicians less than 35 years of age dropped from 28.5 to 18.4 during the same period.

The mortality, retirement, and accession (resumption of activity) rates for the projection model were estimated from an analysis of Masterfile data for years 1978 to 1986 from a set of logistic regression equations, based on the physician's age, sex, specialty, and country of graduation. (Analyses available on request to author.) Female physicians had higher retirement rates and lower mortality rates than male physicians. By assuming that the schedules of transition rates remain constant, we estimated how much longer physicians of a given age, sex, specialty, and country of graduation are, on average, likely to live, and how much longer they are likely to be professionally active.

Table 2 presents the expected years of remaining life and the expected years of remaining work life for active male and female USMG physicians at ages 35, 50, and 65 in 13 different specialty groups. Physicians on average, live and work longer than the rest of the US population. Female physicians can expect to live approximately four to five years longer than male physicians, but their higher retirement rates mean that

TABLE 2—Expected Life and Work Life of Active USMG Physicians at Ages 35, 40, and 65 by Specialty and Sex

	Age (years)					
	35		50		65	
	Male	Female	Male	Female	Male	Female
Expected Life						
Specialty General/Family Practice	42.4	47.2	28.4	33.1	16.3	20.8
General Internal Medicine	43.8	48.4	29.7	34.2	17.3	21.3
Medical Subspecialties	45.0	49.6	30.8	35.3	18.2	22.2
General Surgery	43.7	48.4	29.6	34.1	17.2	21.2
Surgical Subspecialties	44.5	49.2	30.4	34.9	17.9	21.9
Pediatrics	44.9	49.6	30.8	35.3	18.2	22.2
Obstetrics/Gynecology	43.1	47.7	29.0	33.6	16.8	20.7
Radiology	44.4	49.2	30.2	34.9	17.8	21.9
Psychiatry	43.4	48.2	29.3	34.0	17.1	21.2
Anesthesiology	43.9	48.6	29.8	34.4	17.4	21.4
Pathology	43.6	48.6	29.6	34.4	17.3	21.5
Emergency Medicine	42.7	47.9	28.8	33.8	16.8	21.0
Other Specialties	43.7	48.6	29.6	34.4	17.4	21.5
Expected Work Life						
Specialty						
General/Family Practice	36.5	36.8	22.7	23.5	11.2	12.3
General Internal Medicine	37.6	37.8	23.6	24.3	11.8	13.0
Medical Subspecialties	39.1	39.7	25.1	26.1	13.0	14.3
General Surgery	37.1	37.0	23.1	23.7	11.5	12.5
Surgical Subspecialties	37.7	37.5	23.6	24.1	11.8	12.8
Pediatrics	37.8	37.6	23.7	24.2	11.9	12.9
Obstetrics/Gynecology	37.0	37.0	23.0	23.6	11.4	12.4
Radiology	36.5	36.0	22.6	22.8	11.0	11.9
Psychiatry	37.5	37.9	23.5	24.5	11.8	13.1
Anesthsiology	35.8	34.9	21.9	21.9	10.5	11.2
Pathology	36.3	35.9	22.4	22.8	10.9	11.9
Emergency Medicine	34.9	34.2	21.1	21.3	10.0	10.8
Other Specialties	35.8	35.0	22.0	22.1	10.6	11.4

SOURCE: Marder, *et al. Physician Supply and Utilization by Specialty: Trends and Projections.* Table 3.1, p 42.

active female physicians at age 35 have approximately the same expected work life as male physicians at 35.

Physician Time in Patient Care

Among the entire post-residency physician population, 89.7 percent of male physicians and 88.0 percent of female physicians are classified as being in direct patient care (i.e., spending the plurality of their professional working hours treating patients in either an office-based or hospital-based practice). The proportion decreases somewhat for older physicians, and both USFMGs and foreign-born FMGs are slightly more likely than USMGs to be in patient care. (Data available upon request to author.) The probability of being in patient care was highest in general/family practice, the surgical subspecialties, and obstetrics/gynecology, and lowest in the medical subspecialties, pathology, and in the residual "other specialties" category. There are few differences by gender.

Table 3 shows that post-residency patient care physicians worked an average of 58.3 hours per week in 1987. On average, female physicians, older physicians and USMGs worked fewer hours than their counterparts. Physicians in general internal medicine, the medical subspecialties, pediatrics, obstetrics/gynecology, and emergency medicine worked several more hours per week than average, while physicians in psychiatry, pathology, and the other specialties worked fewer hours than average.

TABLE 3—Average Hours Worked per Week by Male and Female Non-Federal Post-residency Patient Care Physicians According to Age, Specialty, and Country of Graduation, 1987

	Total	Male	Female
Total	58.3	59.0	52.3
Country of Graduation			
USMG	58.1	58.6	52.3
FMG	59.3	60.6	52.2
USFMG	59.1	59.3	55.0
Foreign-born FMG	59.3	60.9	52.1
Age (years)			
< 36	60.3	61.7	53.9
36–45	60.2	61.4	50.6
46–55	58.8	59.2	54.3
56–65	55.6	55.8	51.9
> 65	49.0	49.1	45.7
Specialty			
General/Family Practice	58.2	59.3	48.0
General Internal Medicine	60.6	62.0	50.5
Medicine Subspecialties	62.1	62.4	56.1
General Surgery	59.7	59.7	53.7
Surgical Subspecialties	57.8	57.9	52.5
Pediatrics	59.8	62.2	53.3
Obstetrics/Gynecology	60.9	61.7	56.2
Radiology	57.2	57.3	54.8
Psychiatry	50.4	51.1	46.5
Anesthesiology	58.8	60.3	49.7
Pathology	52.9	52.5	56.1
Emergency Medicine	60.7	60.2	64.3
Other Specialties	54.6	54.7	54.4

SOURCE: Marder, *et al. Physician Supply and Utilization by Specialty: Trends and Projections.* Table 2.6, p.18.

The findings reported for the proportion of physicians in patient care and their average hours of work per week were confirmed in a logistic regression analysis of whether a physician was in patient care and an ordinary least squares regression analysis of the log of hours worked per week by patient care physicians. (Analysis available on request to author.) Independent variables were: the physician's sex, country of graduation, specialty, physician's age and age-squared (since the relationships with the dependent variables did not appear to be linear with respect to age), and interaction terms for sex with age and age-squared.

In each analysis, the coefficients for sex and the age-sex interactions were jointly significant at the .001 level. Plots of the age-specific regression estimates for male and female physicians indicated that female physicians are slightly less likely to be in patient care than male physicians for all ages. The hours worked per week by patient care physicians peak at age 37 for male physicians and at age 43 for female physicians. Male physicians generally work more hours than female physicians, but the differential decreases with age.

Alternate Measures of Physician Supply

Some researchers have speculated that because female physicians work fewer hours per week than male physicians and because the proportion of female physicians is increasing, projections of the total physician population may overestimate effective physician supply. We investigated this issue by estimating the *aggregate time worked by patient care physicians*, measured in terms of full-time-equivalent physicians. This measure includes only physicians in direct patient care and weights each physician with the amount of time spent in professional activities. This adjusted measure of physician supply takes into account the changing composition of the physician population. We compared this measure of the effective physician supply with the simple number of physicians.

We considered the population of post-resident physicians—physicians who have completed specialty training—because we wanted to analyze how the changing composition of physician supply will affect the specialty distribution of physician services. Our projection estimates did not indicate how many physicians have completed residency training, so we estimated the future number of post-resident physicians based on the available information—the projected age, sex, country of graduation, and specialty distributions of the future physician population. We used a logistic regression analysis of the 1986 AMA Physician Masterfile data to estimate $q_{i,j,k,l}$, the proportion of physicians of age i, sex j, specialty k, and country of graduation l who have completed residency training. (Analyses available upon request to author.) Assuming that these proportions remain constant throughout the projection, we estimated P, the projected size of the active post-resident physician population, as follows:

$$P = \sum P_{i,j,k,l}$$

$$= \sum [q_{i,j,k,l} A_{i,j,k,l}]$$

where $A_{i,j,k,l}$ represents the projected number of active physicians in the corresponding age-sex-specialty-country of graduation category.

We estimated F, the future number of full-time equivalent patient care physicians, as follows:

$$F = \sum F_{i,j,k,l}$$

$$= \sum \left[c_{i,j,k,l} h_{i,j,k,l} P_{i,j,k,l} \frac{1}{58.0} \right]$$

where $c_{i,j,k,l}$ is the estimated proportion of physicians in each sex-age-specialty-country of graduation category who are in patient care, derived from our logistic regression analysis of the 1986 Masterfile data; $h_{i,j,k,l}$ is the estimated number of hours worked by patient care physicians, derived from the ordinary least squares analysis of the SMS data; and 58.0 is the average hours per week worked by patient care physicians in 1987.

Table 4 presents historical and projected data for the number of active post-residency physicians, and the number of full-time-equivalent post-residency patient care physicians. Both measures show that physician supply is growing rapidly. Projected growth is slightly slower for the full-time-equivalent measure.

The bottom line of Table 5 presents the number of full-time-equivalent patient care post-residency physicians divided by the total number of post-residency physicians. This ratio describes how effectively the projected physician population will provide time to patient care in terms of 1986 full-time-equivalent patient care physicians. During the 1970–86 historical period, the ratio increased slightly from .888 to .896. Despite the growing proportion of female physicians throughout this time period, the average physician provided more patient care because: the proportion of patient care physicians increased; and a greater proportion of physicians were entering the middle parts of their careers, during which they provide the most patient care.

Between 1986 and 2010, the ratio is projected to decrease from .896 to .854, representing more than a 4 percent

TABLE 4—The Number of Active Post-residency Physicians and the Number of Full-Time-Equivalent Post-residency Patient Care Physicians by Age, Specialty, and Country of Graduation, for Years 1970, 1986, 2000, and 2010

	Historical		Projected	
	1970	1986	2000	2010
Active Post-residency Physicians	262,694	438,935	567,300	611,100
Full-Time-Equivalent Post-residency Patient Care Physicians	233,400	393,500	495,700	522,100
Ratio of Full-Time-Equivalent Post-residency Patient Care Physicians to Active Post-residency Physicians	888	896	873	854

SOURCE: AMA Physician Masterfile, 1970–86, Department of Data Release Services, Division of Survey and Data Resources, AMA Demographic Model of the Physician Population, AMA Center for Health Policy Research, 1988.

decrease in the amount of patient care provided by the average physician. This finding is attributable to the growing proportion of women physicians who, on average, work fewer hours per week and are somewhat less likely to be in patient care; and to the growing proportion of older physicians, who work fewer hours per week.

Table 5 shows the percent growth in the two supply measures for the various components of the physician population in 1970–86 and 1986–2010. Both measures indicate that the physician population is growing rapidly, although the growth rate is gradually diminishing. Both indicate that: the percent growth is substantially larger for female physicians than for male physicians; growth rates for emergency medicine, general internal medicine, and pediatrics are greater than for other specialties; and the number of physicians over age 65 will grow faster than the physician population as a whole.

More important for this analysis are the subtle differences between the two measures attributable to changes in the composition of the physician population. In both time periods, the percent increase of active post-residency physicians is several percentage points greater than their full-time equivalents, i.e., the growth of the physician population is slightly faster than the growth of effective physician supply.

For almost all the components of the physician population, the percent increase in active physicians exceeds the percent increase in full-time-equivalent patient care physicians. Female physicians are an important exception. Between 1986 and 2000, disproportionate numbers of female physicians will enter the middle age categories when they spend the most time in patient care. On the other hand, male physicians will be entering the older age categories and thus are expected to work fewer hours per week. Due to this difference, the sex differential in the rate at which physicians provide patient care will decrease in the future.

Discussion

If current patterns persist, the rapidly growing proportion of female physicians will affect both the amount and type of health care available in the future. We have presented data

TABLE 5—Percent Change in the Number of Active Post-residency Physicians and the Number of Full-Time-Equivalent Post-residency Patient Care Physicians, 1986–2000 and 1986–2010

	Percent Change in Post-residency Population			
	1986–2000		1986–2010	
	All Active	Full-Time-Equivalent Patient Care	All Active	Full-Time-Equivalent Patient Care
Total	29.2	26.0	39.2	32.7
Sex				
Male	15.6	13.8	15.1	11.9
Female	120.0	120.7	199.3	197.4
Country of Graduation				
USMG	34.7	32.0	49.5	43.6
FMG	11.6	6.8	5.9	−2.1
USFMG	64.3	62.1	92.4	85.5
Foreign-born FMG	3.7	−1.7	−7.1	−15.7
Age (years)				
< 36	−19.5	−21.3	−21.1	−23.0
36–45	16.0	13.8	2.6	−1.3
46–55	76.0	73.0	71.8	67.1
56–65	30.6	30.0	100.2	97.2
> 65	71.2	67.9	121.1	115.5
Specialty				
General/Family Practice	11.8	10.7	17.0	14.4
General Internal Medicine	54.1	51.1	72.5	64.6
Medical Subspecialties	38.4	32.7	58.7	47.4
General Surgery	25.7	24.0	25.5	21.5
Surgical Subspecialties	18.4	15.2	21.2	15.8
Pediatrics	47.1	41.3	67.9	56.5
Obstetrics/Gynecology	26.6	21.2	37.6	28.4
Radiology	30.9	26.3	41.5	33.9
Psychiatry	13.8	8.9	15.7	7.8
Anesthesiology	41.5	37.9	58.9	51.9
Pathology	14.4	8.5	12.0	3.2
Emergency Medicine	57.6	53.0	87.4	77.4
Other Specialties	20.0	15.8	22.4	14.4

SOURCE: AMA Physician Masterfile, 1970–86, Department of Data Release Services, Division of Survey and Data Resources, AMA Demographic Model of the Physician Population, AMA Center for Health Policy Research, 1988.

on several important past differences between male and female physicians. The projected growth of the physician population slightly overstates projected growth in the effective supply of physician hours worked. However, the actual magnitude of this difference is small, especially when compared to the rapid growth projected for the physician population.

It is quite likely that, as the number of female physicians increases over time, their practices will more closely resemble those of male physicians. Several factors suggest that some of the differences between male and female physicians may be declining. The specialty distributions of male and female physicians appear to be becoming more similar, especially among the younger age groups. Female physicians are no longer disproportionately represented among foreign-born FMGs. Even though female physicians have higher retirement rates than male physicians, their expected work lives are just as long as male physicians due to their lower mortality rates. As career patterns of male and female physicians become more similar, the classic measures of physician supply will be more reliable indicators of long-term growth.

ACKNOWLEDGMENTS

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New Quarterly Periodical: *Accountability in Research*

A new quarterly journal, entitled *Accountability in Research: Policies and Quality Assurance*, was launched in September 1989 to serve as a forum for critical examination of issues involving the integrity of scientific data. The papers appearing in the first issue are based, in part, on presentations from the First International Conference on Scientific Data Audit, Policies, and Quality Assurance held May 8–10, 1988 at the University of Maryland, College Park. Adil E. Shamoo, University of Maryland School of Medicine, has been named Editor-in-Chief of the new journal.

According to "A Note from the Editor" in the inaugural issue, "Researchers today perceive a growing problem of error and fraud in the vast research and development enterprise. The growing complexity of research, the increased distance between those who gather, manipulate, and evaluate data, and the mounting financial and professional pressures on researchers all contribute to this problem. In recent years, the need for a broad-based consensus on ways of ensuring the integrity of scientific data has become increasingly clear," Shamoo wrote.

The journal will speak to a broad, interdisciplinary range of scientists and administrators in academia, industry, and government. "While relevant to many disciplines," he said, "it will focus in particular on biotechnology, chemistry, medicine, pharmacology, toxicology, pathology, and environmental science. Topics to be addressed include: historical perspectives on data integrity; auditing data as a management tool; research policies and their impact on science and the public; ethical and legal issues; relevant data analysis methods and technology; and legislative and regulatory aspects."

The journal welcomes divergent views on these and related topics from research scientists as well as those in the fields of accounting, law, economics, statistics, management studies, public policy, and information science.

Submissions to the journal should be sent in triplicate to the Editor-in-Chief, Adil E. Shamoo, Department of Biological Chemistry, University of Maryland School of Medicine, 660 West Redwood Street, Baltimore, MD 21201.

Subscription rates and other information or **a sample copy** may be obtained from the publisher: Gordon and Breach, Science Publishers, Marketing Department, P.O. Box 786 Cooper Station, New York, NY 10276, USA; or P.O. Box 197, London WC2E 9PX, UK.

Cryptosporidium Infection in Oregon Public Health Clinic Patients 1985–88: The Value of Statewide Laboratory Surveillance

MICHAEL R. SKEELS, PHD, MPH, ROBERT SOKOLOW, BS, MBA, C. VANCE HUBBARD, BS, M(ASCP), JON K. ANDRUS, MD, AND JOANNE BAISCH, RN

Abstract: To evaluate the utility of statewide laboratory *Cryptosporidium* surveillance, we screened stools from all 5,256 patients evaluated at local health departments for parasitic disease from January 1985 through June 1988. Fifty-seven patients (1.1 percent) were found to have *Cryptosporidium*. Seasonal peaks in positivity were observed in the spring, summer, and early autumn months. In children, younger age was associated with higher positivity rate of cryptosporidiosis. As a result of these surveillance efforts, Oregon's first known outbreak of cryptosporidiosis was detected and investigated during 1988. Twenty-five persons were infected, including children, parents, and staff associated with two day care centers. The cost of routine screening for *Cryptosporidium* was $1.13 per specimen in our laboratory, and we consider it useful. (*Am J Public Health* 1990; 80:305–308.)

Introduction

With reports from at least 26 countries,[1–4] *Cryptosporidium* is now well recognized as a pathogen in immunocompetent patients seen for diarrheal disease in a variety of clinical settings. In the United States, most cases are derived from a continuous human reservoir sustained by fecal-oral transmission, with occasional zoonotic infections acquired from cattle, goats, lambs, and domestic animals.[5,6] Surface water may also be an important source of infection.[7,8] Several outbreaks have been reported in day care centers.[9–11] The prevalence of human cryptosporidiosis differs greatly between geographic areas, even within the United States,[12,13] and there is also some evidence for seasonal fluctuation.[14–16]

Many laboratories are currently considering the addition of *Cryptosporidium* screening to the battery of routine tests used for all stools submitted for parasitology. Although *Cryptosporidium* screening is relatively simple, it requires unique staining and examination apart from the usual methods, and its implementation raises issues of cost and work flow which must be addressed. Its usefulness depends upon several factors, including the local prevalence of the agent, the potential for transmission in the population screened, and the cost of screening. There is little information available to assist laboratories and disease control programs in deciding whether to adopt *Cryptosporidium* testing as a part of their routine parasitology protocol.

We have been screening for *Cryptosporidium* infection in immunocompetent public health clinic patients since 1984, and have previously reported our findings in 1,710 patients from our initial year of study.[12] In that report, we noted that although Oregon had not yet experienced the cryptosporidial positivity rate observed in some other areas, we did plan to continue active surveillance. Our ongoing screening during the ensuing three-and-one-half years has allowed us to define further the positivity rate in a selected population, and to detect the first known outbreak of cryptosporidiosis in Oregon. In addition, we have continued to examine the putative association between *Giardia* and *Cryptosporidium* which has been reported by others.[17,18]

Methods

From January 1, 1985 through June 30, 1988, all stool specimens submitted to the Oregon State Public Health Laboratory for parasitological examination from local health department clinics were screened for *Cryptosporidium* in addition to *Giardia* and other parasites. These specimens, which were collected from persons evaluated for parasitic gastroenteritis, were submitted from all of Oregon's local health department outpatient clinics.

All stools were received in 10 percent formalin and concentrated by the formalin-ethyl acetate method.[19] Iodine stained and unstained preparations were examined at both low (100×) and high (400×) magnification for parasitic forms other than *Cryptosporidium*. A smear from each sample was then stained and examined for *Cryptosporidium* in the manner which we have described previously.[12] Briefly, each smear was stained by the auramine-O fluorochrome method and scanned at 400× magnification using a fluorescence microscope with a 390-490 nm light source. Smears were considered presumptively positive if at least one fluorescing object of correct size (and any morphology) was observed per field, or if fluorescing objects morphologically typical of *Cryptosporidium* were observed in any number. *Cryptosporidium* oocysts were seen as typical rings or crescent forms with a mean diameter of 4.5 μm and a resemblance to crenated erythrocytes. Each presumptively positive smear was then overstained with our modification of the Ziehl-Neelsen (Z-N) method for confirmation. The combination of formalin-ethyl acetate concentration plus acid-fast staining has been found to be equivalent to the Sheather sucrose method for recovery of cryptosporidial oocysts.[20] After Z-N staining, the sample was examined at 1,000× magnification and considered confirmed positive if acid-fast oocysts with typical morphology were observed. Positive and negative controls were included in each run and examined in the same manner as clinical specimens. Proper control reactions were a prerequisite for accepting and reporting the concurrent patient results; these controls were correct in 100 percent of the test runs.

Results

Laboratory Surveillance

A total of 5,256 patients (8,396 specimens) were screened for *Cryptosporidium* and other parasites; the percentage who

From the Oregon State Public Health Laboratory, Portland (Skeels, Sokolow, Hubbard), the Epidemiology Program Office, Division of Field Services, CDC, Atlanta (Andrus), and the Marion County Health Department, Salem, Oregon (Baisch). Address reprint requests to Michael R. Skeels, PhD, MPH, Oregon State Public Health Laboratory, P.O. Box 275, Portland, OR 97207-0275. This paper, submitted to the Journal May 18, 1989, was revised and accepted for publication August 8, 1989.

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had diarrhea was unknown to us. The patients were drawn from a variety of ethnic backgrounds and locales throughout Oregon. Fifteen percent (786) had Southeast Asian surnames; none of these patients were positive for *Cryptosporidium*. Only one of the positive patients was known to have human immunodeficiency virus infection. The age distribution of patients is shown in Table 1.

Fifty-seven of the patients screened (1.1 percent) had stools positive for *Cryptosporidium*, although 25 of these patients were related to the day care-associated outbreak in 1988. Eight of 666 patients with *Giardia* (1.2 percent) had *Cryptosporidium*, compared with 49 of 4,590 patients (1.1 percent) who did not have *Giardia*.

Excluding specimens from the patients who were tested as part of our 1988 outbreak investigation (25 positive and eight negative), the three-and-one-half year statewide "background" *Cryptosporidium* positivity rate among those persons screened was 32 of 5,223, or 0.61 percent. The proportion positive for each age group is given in Table 1. When we compared proportion positive by one-year age increments, we found that among patients less than 18 years old who were tested, younger children were more likely to be infected with *Cryptosporidium*; however, the numbers were small and this difference did not reach statistical significance ($p = .07$, chi-square analysis for trend).

Cryptosporidiosis Outbreak, 1988

As a result of laboratory surveillance, Oregon's first known community outbreak of cryptosporidiosis that occurred during a 10-week period from mid-April through June 1988 was discovered and investigated. A total of 24 cases and one asymptomatic infection were identified in two day care centers in one community in western Oregon. Cases were defined as having a diarrheal illness with laboratory confirmation of the presence of *Cryptosporidium* oocysts in stool specimens. The epidemic curves by day care center for this outbreak are consistent with initial person-to-person transmission of infection, perhaps followed by a common source outbreak (Figure 1).

The outbreak was brought to the attention of the local health department in the following manner. A child attending Day Care B was found to be positive for *Giardia* and negative for *Cryptosporidium*; however, an investigation revealed that the child had two infant siblings attending Day Care A. One of the two siblings had a stool sample positive for both *Cryptosporidium* and *Giardia*, and the stool sample from the other was negative for both agents. The finding by the laboratory of *Cryptosporidium*, which had not been suspected in this outbreak, alerted the local health department to the need to conduct an epidemiologic investigation centered on this organism.

TABLE 1—*Cryptosporidium* Positivity Rate in Oregon Public Health Patients by Age, 1985–88

Age (years)	No. Patients Examined	% Positive		
		Day Care Outbreak*	Other	Total
<1	188	66.7 (2/3)	1.1 (2/185)	2.1
1–4	1,248	76.5 (13/17)	1.3 (16/1,231)	2.3
5–9	671	100.0 (2/2)	0.6 (4/669)	0.9
10–14	273	100.0 (2/2)	1.1 (3/271)	1.8
15–17	139	0 (0/0)	0 (0/139)	0
≥18	2,540	66.7 (6/9)	0.2 (6/2,531)	0.5
Unknown	197	0 (0/0)	0.5 (1/197)	0.5
Total	5,256	75.8 (25/33)	0.6 (32/5,223)	1.1

*Outbreak April–June, 1988.

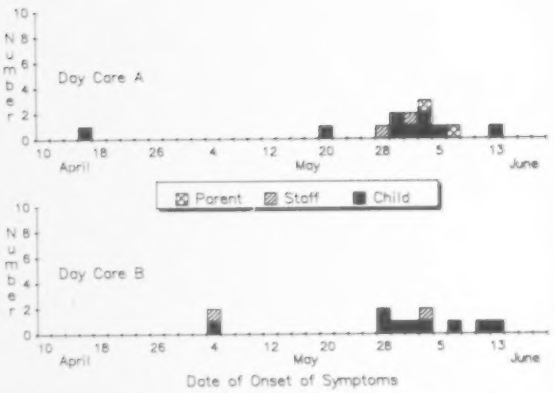


FIGURE 1—Epidemic Curves for Oregon Cryptosporidiosis Outbreak, 1988

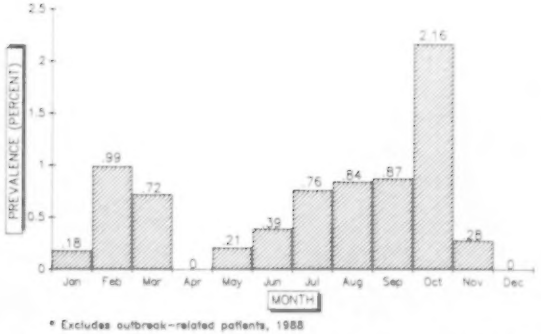
The cryptosporidiosis attack rate for attendees at Day Care A was 90 percent (9/10 children attendees). The onset of the initial case occurred in mid-April. The average age of the children-cases was 1.8 years with a range from 0.8 to 6.0 years. One of the nine case-children also had *Giardia* on stool examination. Both adult staff at Day Care A were infected with *Cryptosporidium*. In addition, two parents of attendees were reported as cases.

At Day Care B the cryptosporidiosis attack rate for attendees was 60 percent (9/15 children). The date of onset of the initial case was May 5, 1988. One attendee had a positive stool specimen, but was asymptomatic, and therefore was not counted as an outbreak-related case. The average age of the case-children was 3.7 years with a range of 2.5 to 6.0 years. Two of the nine case-children also had *Giardia* on stool examination, as did one of the six children who were negative for *Cryptosporidium*. This child was also infected with *Entamoeba histolytica* and *Blastocystis hominis*. In addition to these attendee cases, two of six adult staff were cases. None of the parents were reported as cases.

Excluding the three cases with concurrent giardiasis, who received antimicrobic therapy, all other cases had self-limiting illnesses without treatment; however, the clinical course of one adult case was complicated by a 12-pound weight loss. Among the 10 cases for whom information was available, the average duration of symptoms was eight days with a range of one to 28 days. No one required hospitalization. Control measures included strict handwashing technique and environmental sanitation measures such as cleaning of toys; however, cohorting was not implemented. During the first week of July 1988, repeat stool examinations of all children attendees and infected adults were negative for cryptosporidiosis.

Seasonal Fluctuations in Positivity Rate

When our data were aggregated by month for January 1985 through June 1988, with the 1988 outbreak patients excluded, peak positivity rates were observed in February/March and in summer and early autumn (Figure 2). Given the small number of positive stools in each year, it was necessary to combine the data in this way to identify seasonal patterns.



* Excludes outbreak-related patients, 1988

FIGURE 2—*Cryptosporidium* Prevalence in 6,223 Oregon Public Health Patients Screened for Ova and Parasites by Month, January 1985–June 1988*

Discussion

In Oregon, our finding of 0.61 percent "background" positivity rate in patients screened at all local health department clinics during 1985–88 is consistent with our previous report of 0.35 percent for the same population during 1984.[12] Combining all data for both time periods, including the 1988 outbreak, we have screened 6,966 patients and found 63 (0.90 percent) to be positive for *Cryptosporidium* during four-and-one-half years. Although positivity rates of *Cryptosporidium* vary widely by geography, setting, and population, our results are similar to those reported from Colorado, Texas, Ohio, and British Columbia.[6,13,16,21]

Young children had the highest positivity rate of cryptosporidial infection in our study, even when patients from the 1988 day care-associated outbreak were excluded from the analysis. This finding is consistent with those of other workers and is generally attributed to either greater susceptibility in children or to more efficient fecal-oral transmission among infants and toddlers.[5,16] Less obvious is the source of cryptosporidiosis in older children and adults, many of whom present as sporadic cases without a known source of infection. Many may acquire their infection through contact with young children, as has been suggested in other outbreak situations involving both adults and children.[5,7,11] However, the importance of adult-to-adult transmission (in the immunocompetent) and environmental acquisition is unknown.

Although our findings did not reveal a "statistically significant" association between *Giardia* and *Cryptosporidium* in the population screened or among the day care outbreak cases, co-transmission of these two agents and others may be common during outbreaks. Information is lacking regarding the sensitivity of a single stool examination for the detection of coinfection by these agents. Thus, because of intermittent cyst shedding, differences in incubation period between the two protozoa, the timing of any given sample, and differences in the sensitivity of the laboratory methods, it is possible for one of the pathogens to go undetected, even in a dual infection. For example, some patients excrete atypical *Cryptosporidium* oocysts which are quite fragile and sometimes cannot be stained by the usual methods.[22] Seasonal fluctuations in cryptosporidiosis have also been reported from other countries. Peak prevalences during the late summer and early autumn months have been reported from England[23,24] and Australia;[25] two other studies from the United Kingdom showed peaks during the spring months.[14,15] Among all the seasonal cryptosporidiosis data which are available, those from British Columbia may be the most useful for comparison to our results in Oregon, given the environmental, geographic, and demographic similarities between the two areas. In a one-year study of 7,300 patients with diarrhea in the Vancouver area, the peak incidence was seen in August and September, with over half of the cases occurring in those two months.[16] Our seasonal findings are consistent with those from the Untied Kingdom, Australia, and British Columbia. The interpretation of this seasonality is unclear, but could reflect changing opportunities for acquisition of infection from the environment (e.g., travel, contact with surface water or animals), or for person-to-person transmission.

The relative importance of environmental versus person-to-person transmission of *Cryptosporidium* is still poorly defined, and a better understanding is central to the development of control measures for this agent. Using our experience with *Giardia* as a model, we know that over one-fourth of Oregon giardiasis cases are attributed to drinking surface water, and that there have been several documented community drinking water supply outbreaks of giardiasis in Oregon during the past decade.* There may exist a background of ongoing person-to-person *Giardia* transmission, especially among young children, perhaps augmented by frequent acquisition of the organism from contaminated surface water. Such a pattern may also exist for cryptosporidiosis, but until recently the available technology has limited our ability to examine water for cryptosporidial oocysts.

At least two US waterborne community outbreaks of cryptosporidiosis have been documented, in Texas and Georgia,[26,27] and very recently, the drinking of surface water was identified as an important risk factor in *Cryptosporidium* infections in New Mexico.[7] The Georgia outbreak arose from a sand-filtered and chlorinated community water supply which met all regulatory standards. The presence of cryptosporidial cysts was attributed to infected cattle in the watershed; inadequate flocculation filtration may have allowed the organism to contaminate the drinking water system. Within the past four years, methods have become available for identifying cryptosporidial oocysts in water.[8,28] In Arizona, a study of surface waters during summer 1986 revealed widespread stream contamination with *Cryptosporidium*.[29] A midsummer study of four rivers in Washington and two in northern California showed all six to be contaminated with cryptosporidial oocysts.[8] Similar contamination in nearby watersheds in Oregon seems plausible, especially given the similarities in geography and potential for contamination by dairy cattle and other animals. Comparable studies of Oregon surface water are needed.

An important question about routine cryptosporidial screening of stool samples is whether the cost is justified by the yield. In our laboratory, the direct materials and labor cost for the inclusion of *Cryptosporidium* screening into the usual ova and parasite examination is an additional $1.13 per specimen ($1.03 for personnel, $.10 in materials). The cost-effectiveness of this screening in a given locale will depend

*Oregon disease surveillance reporting data, unpublished. Portland: Oregon Health Division, 1989.

upon the individual laboratory cost, the local prevalence of *Cryptosporidium* and the capacity for preventing and controlling outbreaks once infected persons have been identified.

We conclude that laboratory-based surveillance is useful in defining the *Cryptosporidium* positivity rate and patterns of transmission for a given locale, and in detecting outbreaks when they occur. In areas where *Cryptosporidium* screening is not routinely performed, it is possible that the etiology of diarrheal outbreaks, particularly among children, may remain undiscovered or may be incorrectly attributed to other agents. Even in low prevalence populations, laboratory-based surveillance may be justified due to the relatively low cost of screening and the potential for early intervention in outbreaks.

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Call for Abstracts on Dental Public Health Topics

The American Association of Public Health Dentistry has issued a call for abstracts of papers on a broad range of dental public health topics for presentation at its 53rd annual meeting, to be held in Boston, Massachusetts October 10–13, 1990. The theme for the meeting is "Health for All: Opportunities and Responsibilities for Dental Public Health."

Major topics being sought include: access to services, trends in oral diseases or conditions, preventive and educational measures, national/state/local dental programs, delivery systems, infection control, ethics, and impact of state practice acts.

The *deadline for submission of abstracts is April 1, 1990*. An abstract form and information can be found in the November 1989 issue of *Communique* (AAPHD's newsletter) and the Winter 1990 issue of the *Journal of Public Health Dentistry*. Send abstracts to: Alice M. Horowitz, RDH, MA, AAPHD Vice President, 6307 Herkos Court, Bethesda, MD 20817.

For further information, contact AAPHD National Office, 10619 Jousting Lane, Richmond, VA 23235. Tel: 804/272-8344.

A Comparison of Alcohol Sales Data with Survey Data on Self-Reported Alcohol Use in 21 States

PERRY F. SMITH, MD, PATRICK L. REMINGTON, MD, MPH, DAVID F. WILLIAMSON, PHD, AND ROBERT F. ANDA, MD

Abstract: We used data from 21 states that participated in the 1985 Behavioral Risk Factor Surveillance System to compare state-specific per capita self-reported alcohol consumption and the prevalence of three drinking behaviors with state-specific per capita sales. The correlation coefficient for per capita sales and per capita self-reported consumption for the 21 states was 0.81. Per capita sales were also significantly correlated with the prevalence of self-reported heavier drinking, binge drinking, and drinking and driving; the corresponding correlation coefficients were 0.74, 0.59, and 0.51. These findings suggest that states with higher per capita sales of alcohol also have higher rates of self-reported consumption and drinking patterns suggestive of high-risk behavior. (*Am J Public Health* 1990; 80:309–312.)

Introduction

The social, medical, and economic costs of alcohol abuse are a major public health problem in the United States.[1] At the state level, public health officials are paying increased attention to the consequences of problem drinking. Recently, investigators estimated that problem drinking in Minnesota accounted for 1,110 deaths and for $1.4 to $2.1 billion in medical and social expenditures during 1983.[2]

Because direct measures of problem drinking are not always available, indirect measures, such as total sales, are often used to estimate the total alcohol exposure for state populations. For example, one of the 1990 health objectives for the nation is to limit per capita consumption of alcohol to 2.71 gallons per year, an amount estimated from alcohol sales.[3] Sales data at the state level may mismeasure actual per capita consumption because of out-of-state visitors' purchase of alcohol and wastage or stockpiling of alcohol.[4] More importantly, per capita sales may not reflect rates of problem drinking in a population.[5]

A somewhat more direct method for estimating alcohol exposure at the state level is the use of survey data of self-reported alcohol consumption by individuals. However, survey data may not accurately reflect rates of problem drinking for various reasons, including biased self-reporting and problems with sampling.[4,6–8]

Although public health officials use alcohol sales as a measure of typical alcohol consumption and of the frequency of problem drinking, it has not been shown that states with higher alcohol sales have higher levels of per capita consumption and/or higher frequency of drinking behaviors that increase a person's risk for alcohol-related morbidity and mortality. A strong correlation between sales and survey data at the state level would support the use of sales information for monitoring alcohol consumption. To determine the strength of this correlation, we compared state-specific per capita alcohol sales with rates of per capita consumption and three drinking behaviors as reported in surveys administered in 21 states in 1985.

Methods

We obtained state-specific estimates of per capita alcohol sales for 1985 from the Alcohol Epidemiologic Data System of the National Institute on Alcohol Abuse and Alcoholism (NIAAA).[9] These data are based on beverage sales and tax receipt figures for states in which these data are available (including 13 of the 21 states in our study). For the remaining states, data are used on product shipments from major alcoholic beverage industry sources (the Distilled Spirits Council of the United States, United States Brewers Association, and the Wine Institute). For this report, we refer to both sources of data as "alcohol sales." Per capita alcohol sales are determined by dividing each state's total sales in gallons of absolute alcohol for beer, wine, and liquor by the number of state residents aged 14 years and older.

To estimate state-specific rates of alcohol consumption and drinking behaviors based on self-reports, we used 1985 data from the Behavioral Risk Factor Surveillance System. This system uses random-digit telephone surveys for the state-level surveillance of behaviors related to the leading causes of death among adults in the United States. Described in detail elsewhere,[10] the methodology is briefly summarized here.

In 1985, 21 states participated in the surveillance system. Adult respondents were randomly selected from civilian residents, aged 18 years or older, who had telephones. The sample was chosen by a multistage cluster design procedure based on the Waksberg method.[11] Trained interviewers in each state conducted interviews by telephone; calls were made during the day, at night, and on weekends and lasted about 15 minutes each.

In each state, about 100 interviews were completed each month; the annual total per state ranged from 628 to 2,386 (median = 1,177) (Table 1). Response rates were calculated as the percentage of eligible respondents contacted who completed interviews, and ranged from 61–92 percent (median = 83 percent), among the 19 states for which this information was available.

Respondents were asked to report their beer, wine, and liquor consumption, including the number of days in the prior month that they had consumed each type of beverage and the average number of drinks they had consumed per day. They were also asked two questions: "Considering all types of alcoholic beverages, that is, beer, wine, and liquor, as drinks, how many times during the past month did you have five or more drinks on an occasion?" and "During the past month, how many times have you driven when you've had *perhaps* too much to drink?" Those who did not report consuming any alcohol in the month before the interview were considered nondrinkers. For the 21 states, the percentage of nondrinkers ranged from twenty-six to sixty-nine percent (median = forty-one percent) (Table 1).

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TABLE 1—Number of Completed Interviews, Response Rates, and Percentage Nondrinkers, by State, Behavioral Risk Factor Surveillance System, 1985

State	No. of Completed Interviews	% Response Rate*	Percentage Nondrinkers**
Arizona	1,175	76.5	41.0
California	1,372	82.6	37.3
Connecticut	983	***	30.3
Florida	818	61.2	40.4
Georgia	818	81.8	53.8
Idaho	1,179	79.3	47.8
Illinois	1,148	72.8	41.1
Indiana	1,182	90.9	49.2
Kentucky	803	86.6	62.0
Minnesota	2,386	75.2	36.3
Montana	1,183	***	35.6
New York	1,183	79.1	32.8
North Carolina	1,528	89.9	56.7
North Dakota	628	90.4	37.6
Ohio	1,156	75.7	43.8
Rhode Island	1,277	75.4	38.1
South Carolina	1,216	83.7	58.2
Tennessee	1,207	83.8	66.2
Utah	1,162	92.3	69.3
West Virginia	1,177	89.8	63.5
Wisconsin	965	91.4	25.3

*Percentage of eligible respondents contacted who completed interviews.
**Percentage of respondents who did not report consuming any alcohol in the month before the interview. These percentages are adjusted to compensate for the multistage cluster design of the survey and to be representative of each state's population (based on the age and sex distribution of each state's population in 1985).
***Data necessary for computing response rates not available.

To calculate per capita consumption based on self-reports, we assumed that one beer contained 12 oz of 4.5 percent alcohol; one glass of wine 4 oz of 12.9 percent alcohol; and one liquor drink 1 oz of 41.1 percent alcohol. These alcohol percentages are the same coefficients used by the NIAAA to convert the volume of beer, wine and liquor into the volume of absolute alcohol.[9] We added each respondent's estimate of beer, wine, and liquor consumption and multiplied the sum by 12 to determine the total alcohol consumption for each respondent per year. To calculate the mean alcohol consumption by state, we weighted the value of each respondent's total consumption according to his or her probability of selection as a result of the multistage cluster design of the survey.[12] This weighting compensated for the number of persons 18 years of age and older in each household, the number of telephone numbers reaching each household, and variation in cluster size. We also weighted each respondent's total consumption according to the age and sex distribution of the 1985 adult population for each participating state. This procedure provided population-based estimates of per capita consumption that are representative of each participating state.

Using the survey data, we also determined the state-specific prevalence rates of three drinking behaviors: *heavier drinking* (consuming 60 or more drinks during the prior month), *binge drinking* (consuming five or more drinks on at least one occasion during the prior month), and *drinking and driving* (driving at least once during the prior month after having had *perhaps* too much to drink). As was done with the rates of per capita consumption, the state-specific prevalence rates of these behaviors were adjusted to the age and sex distribution of the 1985 adult population for each state, after weighting the responses to account for the multistage cluster design.

We used linear regression to assess the strength of the association between the state-specific per capita alcohol sales and the measures of self-reported alcohol use; the dependent variables were the measures of self-reported alcohol use and the independent variable was per capita sales.

We used computer software from SAS Institute Inc,[13] and Lotus Development Corporation[14] to perform all calculations.

Results

Among the 21 states, per capita alcohol sales varied twofold, from 1.58 gallons per year in Utah to 3.19 gallons per year in Wisconsin (median = 2.49 gallons) (Table 2). Per capita alcohol consumption varied two and one half-fold, from 0.40 gallons per year in Utah to 1.06 gallons per year in New York (median = 0.72 gallons). Self-reported per capita consumption, when expressed as a percentage of per capita sales, ranged from 21.6 percent in Tennessee to 40.5 percent in New York (median = 28.2 percent) (data not shown).

States that ranked higher in alcohol sales also tended to rank higher in self-reported consumption (Table 2). For example, of the four highest-ranking states in sales (Wisconsin, Arizona, California, and Florida), Wisconsin and Florida both ranked second, Arizona ranked sixth, and California ranked seventh in self-reported consumption. The four states that ranked lowest in sales (Utah, West Virginia, Kentucky, and Tennessee) also ranked lowest in self-reported consumption.

Per capita sales showed a strong linear relation with self-reported consumption among the 21 states (Figure 1). The correlation between sales and consumption was 0.81, with a β coefficient of 0.34 (95 percent confidence interval = 0.23, 0.45) (Table 3). The β coefficient indicates that the average per capita increase for this group of states was 0.34 gallons of self-reported alcohol consumption for each gallon increase in per capita sales of alcohol.

We also assessed the strength of association between sales and self-reported consumption among states in two subgroups: the 13 states for which beverage sales and tax receipt figures were used to determine alcohol sales; and the remaining eight states for which alcohol shipments records were used to determine alcohol sales. The correlation between sales and consumption for states in the first group ($r = 0.70$) was similar to the correlation for states in the second group ($r = 0.74$).

The rates of the three drinking behaviors varied from state to state (Table 2). The prevalence of heavier drinking varied threefold, from 3.8 percent in Utah to 11.5 percent in Illinois (median = 7.1 percent). The prevalence of binge drinking varied almost fivefold, from 5.7 percent in Tennessee to 27.8 percent in Wisconsin (median = 16.6 percent). The prevalence of drinking and driving varied sixfold, from 1.5 percent in Kentucky and Tennessee to 9.4 percent in Wisconsin (median = 3.5 percent).

As was found for self-reported alcohol consumption, the prevalence estimates for each of the three drinking behaviors were correlated with per capita sales (Table 3): heavier drinking $r = 0.74$, binge drinking $r = 0.59$, and drinking and driving $r = 0.51$.

Discussion

Our analysis found that alcohol sales and self-reported drinking behaviors are strongly correlated and provide com-

TABLE 2—Annual per capita Alcohol Sales, Self-Reported Consumption of Alcohol, and Prevalence of Self-Reported Drinking Behaviors, by State, 1985

State	Per Capita Sales (gallons)	Self-Reports			
		Per Capita Consumption (gallons)	Rate of Heavier Drinking %	Rate of Binge Drinking %	Rate of Drinking & Driving %
Wisconsin	3.19 (1)*	1.02 (2)	10.4 (5)	27.8 (1)	9.4 (1)
Arizona	3.12 (2)	0.88 (6)	10.7 (4)	17.9 (10)	5.3 (7)
California	3.12 (2)	0.86 (7)	9.3 (7)	16.6 (11)	3.0 (14)
Florida	3.04 (4)	1.02 (2)	10.8 (3)	16.5 (12)	3.0 (14)
Rhode Island	2.94 (5)	0.78 (9)	7.4 (10)	12.3 (16)	2.8 (16)
Connecticut	2.84 (6)	0.99 (4)	11.2 (2)	19.9 (6)	5.7 (5)
Montana	2.81 (7)	0.76 (10)	6.4 (12)	22.3 (4)	5.4 (6)
Illinois	2.72 (8)	0.99 (4)	11.5 (1)	20.8 (5)	6.2 (4)
Minnesota	2.68 (9)	0.86 (7)	7.6 (9)	23.1 (3)	6.8 (3)
New York	2.62 (10)	1.06 (1)	9.5 (6)	18.5 (9)	4.1 (9)
South Carolina	2.49 (11)	0.57 (16)	6.0 (15)	8.2 (19)	2.3 (19)
North Dakota	2.48 (12)	0.61 (14)	4.6 (20)	23.4 (2)	9.3 (2)
Georgia	2.43 (13)	0.57 (16)	6.1 (14)	14.3 (14)	3.5 (11)
Idaho	2.42 (14)	0.67 (13)	7.1 (11)	15.3 (13)	3.3 (13)
Ohio	2.20 (15)	0.72 (11)	8.6 (8)	19.2 (7)	4.3 (8)
Indiana	2.19 (16)	0.71 (12)	5.8 (16)	19.2 (7)	3.8 (10)
North Carolina	2.13 (17)	0.59 (15)	5.6 (18)	11.6 (18)	3.4 (12)
Tennessee	1.94 (18)	0.42 (20)	5.8 (16)	5.7 (21)	1.5 (20)
Kentucky	1.86 (19)	0.56 (18)	6.3 (13)	7.8 (20)	1.5 (20)
West Virginia	1.66 (20)	0.54 (19)	4.9 (19)	13.2 (15)	2.8 (16)
Utah	1.58 (21)	0.40 (21)	3.8 (21)	11.8 (17)	2.5 (18)

*Numbers in parentheses indicate the rank order (high to low) of the 21 states for each measure of alcohol use. The same ranking number was assigned to states with identical values.

FIGURE 1—Regression Analysis of per capita Sales and per capita Consumption of Alcohol for 21 States, 1985
NOTE: Data points for the 21 states are shown with the fitted regression line, its equation, and the correlation coefficient. Per capita consumption was based on self-reports.

TABLE 3—Summary of Simple Linear Regression Analyses: Annual per capita Alcohol Sales by Four Measures of Alcohol Use from Self-Reports in 21 States, 1985

Measures of Alcohol Use from Self-Reports	β Coefficient*	r	R^2
Per Capita Alcohol Consumption in Gallons	.34 (.23,.45)**	.81	.66
Prevalence of Heavier Drinking	.03 (.02,.05)	.74	.55
Prevalence of Binge Drinking	.07 (.03,.11)	.59	.35
Prevalence of Drinking and Driving	.02 (.01,.04)	.51	.26

*The β coefficient represents the amount of increase in each measure of self-reported alcohol use for each gallon increase in per capita sales of alcohol.
**Numbers in parentheses indicate the 95 percent confidence intervals for the β coefficients.

plementary sources of information. However, each measure has its limitations.

State-specific sales may overestimate consumption in states where alcohol is often purchased by out-of-state visitors. An example is the District of Columbia (DC), which participated in the 1985 Behavioral Risk Factor Surveillance System but was excluded from our analysis because DC is more comparable to a large metropolitan area than to a state. Compared with the results for the 21 states, alcohol sales were disproportionately higher in DC relative to self-reported consumption. This finding is due in part to the purchase of alcohol by tourists and residents from nearby states.

An additional limitation to using alcohol sales to monitor alcohol use is that alcohol that is purchased may not be consumed, either because of wastage (unfinished drinks, spillage) or because of stockpiling at the wholesale, retail, and consumer levels. Although the problem of wastage may be negligible,[4] stockpiling can greatly bias estimates of alcohol use.[15]

In contrast to sales data which provide information only about total alcohol consumption, population surveys can be used to assess both consumption and self-reported drinking behaviors. However, these surveys also have well-recognized problems.[4,6,16] Surveys of self-reported alcohol use uniformly produce lower estimates of total alcohol consumption than do analyses of alcohol sales data.[4,7,8] In previous surveys, self-reported consumption has accounted for only 40–60 percent of alcohol sales, with some reports as low as 26–30 percent of sales.[4] In our study, self-reported consumption accounted for 22–41 percent of alcohol sales. Most telephone surveys are unlikely to include certain segments of the population—such as college students and young men living on military bases—who often are drinkers. Also, population surveys generally undersample heavy drinkers because many of these persons refuse to participate or cannot be located. An additional problem is reporting bias: respondents who are concerned about the social acceptability of their alcohol use may give lower estimates of consumption.

Estimates of self-reported consumption also suffer from the approximations involved in calculating the volume of alcohol consumed per drink. The considerable variation in the size and concentration of alcohol per drink makes estimates of total consumption inexact. This problem has increased with the recent introduction of low-alcohol-content drinks, such as "light" beers and wine "coolers."

Despite all these limitations of sales data and of surveys of self-reported alcohol use, we found a strong correlation between per capita sales and per capita consumption based on self-reports. Per capita sales were also strongly correlated with self-reported heavier drinking, binge drinking, and drinking and driving for the 21 states. These three behaviors do not necessarily identify individual alcohol abusers, nor do they provide accurate measures of problem drinking in a population. However, the prevalence of these behaviors may represent indirect measures of problem drinking by indicating the percentage of a population at increased risk for alcohol-related problems, especially injuries.[17,18]

The results of this study may have been influenced by age considerations. The per capita consumption estimates from sales data were based on each state's number of residents aged 14 and older, as an estimate of the drinking population.[9] Changing the assumed size of the drinking population would affect the calculated rates of per capita consumption from sales data. Furthermore, the surveys included persons only 18 years and older and thus did not reflect consumption and drinking behaviors among younger persons.

Because our study examined ecological associations between alcohol sales and patterns of alcohol use, it was not possible to determine the reasons for the observed correlations between per capita sales and the three drinking behaviors. However, our results are consistent with several theories of alcohol use, including the distribution of consumption model.[19] This theory states that there is a constant relation between per capita alcohol consumption and the prevalence of heavy drinking and suggests that the average alcohol consumption in a population is positively correlated with the prevalence of problem drinking.

We conclude that per capita sales of alcohol generally parallel self-reported consumption and drinking patterns suggestive of high-risk behavior. Although public health officials need to be aware of the limitations of both sales data and self-reports, the high degree of correlation between these two measures of alcohol use suggests that it is reasonable to use alcohol sales for monitoring alcohol consumption when other sources of information are not available.

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Physician Supply Projections to be Re-Examined

Projected manpower needs through the year 2010 in six medical specialties will be re-examined and revised as indicated under a contract awarded to Abt Associates Inc., of Cambridge, Massachusetts. The 13-month, $279,463 project, funded by the Health Resources and Services Administration (HRSA), Division of Medicine, will update projections made in 1980 by the Graduate Medical Education National Advisory Committee (GMENAC) for the fields of family practice, general pediatrics, obstetrics-gynecology, general internal medicine, general surgery, and general and child psychiatry.

The project will involve professional organizations representing these six specialties, and will use Bureau of Health Professions physician supply projections through 2010 adjusted to incorporate the supply of osteopathic physicians. The activity will also include a qualitative assessment of the impact that various economic, social, and behavioral constraints, such as problems of the underserved population, would have on the balance between physician supply and national needs.

For further information, contact Blake Crawford, HRSA, USPHS, DHHS, 5600 Fishers Lane, Room 14-43, Rockville, MD 20857. Tel: 301/443-3376.

Cesarean Section Use and Source of Payment: An Analysis of California Hospital Discharge Abstracts

RANDALL S. STAFFORD, PHD, MS

Abstract: This study assessed the relation between payment source and cesarean section use by analyzing California data on hospital deliveries. Of 461,066 deliveries in 1986, cesarean sections were performed in 24.4 percent. Women with private insurance had the highest cesarean section rates (29.1 percent). Successively lower rates were observed for women covered by non-Kaiser health maintenance organizations (26.8 percent), Medi-Cal (22.9 percent), Kaiser (19.7 percent), self-pay (19.3 percent), and Indigent Services (15.6 percent). Vaginal birth after cesarean (VBAC) occurred more than twice as frequently in women covered by Kaiser (19.9 percent) and Indigent Services (24.8 percent), compared to those with private insurance (8.1 percent). Sizable, although less pronounced, associations between payment source and cesarean section use were noted for the indications of breech presentation, dystocia, and fetal distress. Accounting for maternal age and race/ethnicity did not alter these findings. Variations in the use of cesarean section have a substantial financial impact on health care payors. (*Am J Public Health* 1990; 80:313–315.)

Introduction

Medical care decisions are influenced by non-clinical factors, such as health care financing.[1–3] Cesarean section is the most common hospital surgical procedure in the United States,[4] accounting for 24.4 percent of all deliveries in 1987.* The quadrupling of US cesarean section rates in the past 15 years has led to concerns regarding maternal and perinatal outcome, as well as health care costs.[5]

Past studies suggest that women covered by private insurance have the highest cesarean section rates.[6–8] Women covered by Medicaid, those covered by health maintenance organizations (HMOs) and those without a source of payment (self-pay) have lower C-section rates. Lower C-section rates also are observed for teaching hospitals,[9] public hospitals,[6,8] and salaried physicians.[10] Unfortunately, past research has not controlled adequately for the population characteristics (case-mix) of different payors or organizational settings.

This report investigates the relationship between hospital payment source and the use of cesarean section by employing 1986 discharge abstract data from California hospital deliveries.

Methods

Hospital Discharge Data

The Office of Statewide Health Planning and Development collects information on every non-military hospital discharge in California;[11] data for 1986 included 461,066 hospital deliveries.

Cesarean section use was determined via ICD-9-CM procedure codes. Initial categories for expected source of payment for hospitalization were combined into seven distinct payment methods: Private Insurance (Blue Cross/Blue Shield and other private insurance), Kaiser Permanente, Other (non-Kaiser) HMOs, Medi-Cal (the California Medicaid program), Self-Pay, Medically Indigent Services, and Other Payors. Women with HMO coverage delivering in Kaiser Hospitals were assigned to Kaiser Permanente, all other HMO deliveries were assigned to Other HMOs.

Diagnostic information was available as ICD-9-CM codes. A standard and widely employed hierarchy of mutually exclusive diagnoses which accompany cesarean section was adopted for this study.[12,13] From highest to lowest priority, these were: previous cesarean section (ICD-9-CM 654.2), breech presentation (652.2 and 669.6), dystocia (653 and 660-662, except 661.3), fetal distress (656.3), and all other diagnoses (remaining codes in 642-663, and 669, except 669.1-669.5).[12,13] When more than one diagnosis was present for a delivery, the highest priority diagnoses was assigned regardless of its order on the discharge abstract. Maternal age was defined in five-year categories (<20, 20–24, 25–29, 30–34, 35+). Five race/ethnicity categories were employed: White, Black, Hispanic, Asian and Others/Unknown.

Statistical Methods

Cesarean section rates by payment source for individual primary indications were calculated. Confidence intervals were calculated for the cesarean section rates of each payor. Payors whose confidence intervals do not overlap can be considered different at a level of $p < 0.05$, after accounting for multiple comparisons between payors.[14]

To assess the constancy of the relation between payment source and cesarean section rates, the sample was simultaneously stratified by accompanying diagnoses, maternal age, and race/ethnicity. To further validate the findings derived from the above methods, logistic regression analysis was employed to simultaneously estimate the independent effects of payment source, accompanying diagnoses, maternal age, and race/ethnicity. The results of this analysis closely agreed with those from the tabular analysis (details available upon request to author).

Results

In 1986, 112,730 cesarean sections were performed in California hospitals, a cesarean section rate of 24.4 percent. Private Insurance (35 percent) and Medi-Cal (27 percent) were the dominant payors for hospital deliveries (Table 1).

Cesarean section rates varied widely by payment source. Women covered by Private Insurance had the highest cesarean section rates (29.1 percent) (Table 1). Indigent women had the lowest cesarean section rates, with only 15.6 percent being delivered abdominally.

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TABLE 1—Cesarean Section Rates by Payment Source, Hospital Deliveries, California, 1986

Payment Source	Total Deliveries	Percentage of all Deliveries	C-Section Rate (95% CI)
Private Insurance*	161,847	35.1	29.1 (28.8, 29.4)
Other HMOs	46,034	10.0	26.8 (26.3, 27.4)
Medi-Cal	122,792	26.6	22.9 (22.6, 23.2)
Kaiser Permanente	51,589	11.1	19.7 (19.2, 20.1)
Self-Pay	49,292	10.7	19.3 (18.8, 19.8)
Indigent Services	10,322	2.2	15.6 (14.6, 16.5)
Other Payors**	19,190	4.2	20.2 (19.5, 21.0)
Total	461,066	100.0	24.4 (24.3, 24.6)

*Includes Blue Cross/Blue Shield and Other Private Insurance.
**Includes Medicare (658 deliveries), Workers' Compensation (104), Title V (208), Other Governmental Payors (13,220), Other Non-governmental Payors (3,709), and No Charge (1,292).

Repeat cesarean sections accounted for 36 percent of all cesareans (Table 2). Large payor differences were observed in women with previous cesarean sections, with an ordering of payors similar to that for all indications (Table 3). Repeat cesarean section rates varied from 91.9 percent for Private Insurance to 75.2 percent for Indigent Services, the rate of vaginal birth after cesarean (VBAC) in indigent women (24.8 percent) was three times that in women with Private Insurance (8.1 percent).

Cesarean section for breech presentation exhibited a distinct pattern by payor. Kaiser had a relatively high breech cesarean rate (88.3 percent); indigent women the lowest (78.5 percent) (Table 4).

Cesareans for dystocia accounted for 32 percent of all cesareans. For this accompanying diagnosis, relatively high cesarean rates were experienced by Private Insurance (65.9 percent, 65.1–66.7), Other HMOs (65.3 percent, 63.7–67.0), Medi-Cal (64.6 percent, 63.5–65.7), and Self-Pay (61.4 percent, 59.7–63.1). Women covered by Indigent Services (56.9 percent, 52.3–61.5) and Kaiser (58.8 percent, 55.4–62.3) had significantly lower rates.

Deliveries with the diagnosis of fetal distress showed a pattern similar to that for all deliveries. Other HMOs (33.0 percent, 30.9–35.0) and Private Insurance (31.5 percent, 30.4–32.6) had the highest rates, while indigent women had the lowest (21.6 percent, 18.4–24.9).

TABLE 2—Cesarean Section Rates by Primary Accompanying Diagnosis, Hospital Deliveries, California, 1986

Primary Accompanying Diagnosis	Deliveries	Percentage of all Deliveries	Cesarean Sections	Percentage of all Cesareans	Cesarean Section Rate
Previous Cesarean	45,188	9.8	40,274	35.7	89.1
Breech	13,926	3.0	12,112	10.7	87.0
Dystocia	55,895	12.1	35,896	31.9	64.2
Fetal Distress	39,058	8.5	11,134	9.9	28.5
Other Diagnoses*	111,156	24.1	12,926	11.5	11.6
None**	195,843	42.5	388	0.3	0.2
Total	461,066	100.0	112,730	100.0	24.4

*Includes the following diagnoses occurring without previous cesarean section, breech presentation, fetal distress, or dystocia: antepartum hemorrhage, hypertension, excessive vomiting, pre-term labor, prolonged pregnancy, other pregnancy complications, maternal infections, other medical conditions in pregnancy, multiple gestation, other malpresentation, anatomical abnormalities of pelvis, fetal abnormalities, placental problems, polyhydramnios, other amniotic complications, other indications for care, umbilical cord complications, and other complications of labor and delivery.
**Includes deliveries lacking any diagnosis classifiable to any of the above categories.

TABLE 3—Cesarean Section Rates for Women with Previous C-Sections, by Payment Source, Hospital Deliveries, California, 1986

Payment Source	Total Deliveries	Percentage of Payor's Deliveries	C-Section Rate (95% CI)
Private Insurance	18,837	11.6	91.9 (91.4, 92.4)
Other HMOs	5,064	11.0	91.7 (90.6, 92.7)
Medi-Cal	11,444	9.3	90.6 (89.9, 91.4)
Kaiser Permanente	4,385	8.5	80.1 (78.5, 81.7)
Self-Pay	3,353	6.8	81.9 (80.1, 83.7)
Indigent Services	660	6.4	75.2 (70.7, 79.6)
Other Payors	1,445	7.5	82.9 (80.3, 89.5)
Total	45,188	9.8	89.1 (88.7, 89.5)

TABLE 4—Cesarean Section Rates for Breech Presentation, by Payment Source, Hospital Deliveries, California, 1986

Payment Source	Total Deliveries	Percentage of Payor's Deliveries	C-Section Rate (95% CI)
Private Insurance	5,510	3.4	89.7 (88.6, 90.8)
Other HMOs	1,381	3.0	89.9 (87.7, 92.1)
Medi-Cal	3,400	2.7	83.9 (82.2, 85.6)
Kaiser Permanente	1,424	2.8	88.3 (86.0, 90.6)
Self-Pay	1,416	2.9	81.6 (78.8, 84.4)
Indigent Services	247	2.4	78.5 (71.4, 85.6)
Other Payors	548	2.9	85.0 (80.9, 89.1)
Total	13,926	3.0	87.0 (86.2, 87.8)

For deliveries with other accompanying diagnoses, women with Private Insurance had cesarean section rates (14.4 percent, 13.9–14.9) nearly double those of indigent women (7.7 percent, 6.3–9.1).

Of the 125 subpopulations formed from all combinations of age-group (five categories), race/ethnicity (five categories), and accompanying diagnoses (five categories), 105 had three or more payors with more than 20 deliveries. Among these subpopulations, Private Insurance had the highest cesarean rates in 42 percent and non-Kaiser HMOs in 30 percent. Medi-Cal (8 percent), Other Payors (8 percent), Self-Pay (6 percent), Kaiser (6 percent), and Indigent Services (1 percent) had the highest cesarean rates less frequently (detailed tables available on request to author).

Discussion

This study indicates that cesarean section use is strongly associated with the source of payment for obstetrical care, after stratifying for available patient characteristics. Differences between payors were particularly large for women with previous cesarean sections, currently a controversial area of obstetrical practice.[15,16]

Because payment source is not randomly assigned to women, stratification by patient characteristics is an important aspect of this study. Two potential limitations in this process must be noted. First, hospital discharge abstracts have a limited number of data elements available, and do not include information on parity, gravidity, and birthweight. Second, potential problems in data accuracy and completeness may diminish the discharge data's ability to reflect

clinical differences between patients. In particular, dystocia and fetal distress may be subject to biased recording and are more likely to be recorded if a cesarean section is performed.

Direct or indirect financial incentives, as well as additional factors such as access to technology,[8] physician work schedules,[17] hospital teaching status,[8] hospital ownership status,[6,9] patient socioeconomic status,[18] and medical malpractice concerns may contribute to the observed payor differences. In addition, it is possible that the underlying delivery preferences of women or their physicians may vary systematically by source of hospital payment.

The high cesarean rate for Private Insurance is consistent with the financial incentives of fee-for-service practice that favor higher cesarean section rates. The existence of incentives does not necessarily imply that physicians consciously seek greater income. Such incentives may function via implicit clinical standards.

The moderate Medi-Cal cesarean rates are consistent with incentives to perform cesarean section that may be tempered by the reduced physician and hospital reimbursements provided by Medi-Cal.[19] Women with Medi-Cal also tend to deliver in hospitals where there may be other constraints on cesarean section use.

While HMOs have been shown to have lower cesarean section rates than private insurance,[7,20] the current findings suggest this is the case only in large, hospital-based HMOs. The data indicate that the cesarean section experience of HMOs other than Kaiser closely resembles that of private insurance. Besides Kaiser's incentive to avoid excessive cesarean section use, the prominence of peer review and fixed work schedules also may contribute to the lower Kaiser cesarean rates.

The desire of Self-Pay women to avoid the higher cost of cesarean section may discourage cesarean section use. In addition, Self-Pay women make disproportionate use of public hospitals, where additional institutional constraints may reduce cesarean use.

The low rates of cesarean section in women covered by Indigent Services is consistent with the economic constraints of the county hospitals where these women deliver. At the same time, many of these hospitals have teaching programs which, like Kaiser, are more likely to have fixed work schedules and emphasize peer review.

The potential costs savings to payors of fewer cesarean sections may be gauged using 1986 estimates of physician and hospital costs, and Kaiser's moderate cesarean section rate of 19.7 percent. Insurance claims data for the western US indicate that in 1986 cesarean sections were 84 percent more costly than vaginal deliveries ($5,000 versus $2,720).[21] Based on these figures, if the cesarean section patterns of Kaiser were adopted, private insurance plans, non-Kaiser HMOs and Medi-Cal potentially would save $51 million annually by avoiding 22,500 cesarean sections. This may be an underestimate of cost savings, however, because Kaiser's rate is nearly twice that suggested as an optimal cesarean section rate.[22,23]

ACKNOWLEDGMENTS

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Public Health Briefs

The Effects of Case Definition in Maternal Screening and Reporting Criteria on Rates of Congenital Syphilis

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Abstract: Reports of congenital syphilis in 1987 were reviewed to determine how new national guidelines for defining congenital syphilis would influence reported rates in Los Angeles County. After reviewing all reported reactive serologic tests for syphilis, we found 166 additional cases, resulting in a 426 percent increase in the 1987 reported rate of congenital syphilis. Rates of congenital syphilis are dependent upon surveillance practices, screening policies, and case definition. (*Am J Public Health* 1990; 80:316–317.)

Introduction

Until January 1988, the Centers for Disease Control (CDC) had asked health departments to report cases of congenital syphilis based upon Kaufman's criteria.[1] This complicated system classified infants as "definite," "probable," "possible," and "unlikely" based upon symptoms and laboratory findings at birth and serological tests collected over time. A major shortcoming is the variability with which follow-up data might be available, making the accurate reporting of cases difficult. A second problem was that Kaufman's criteria excluded stillborn infants of mothers with untreated syphilis.[2]

In January 1988, the CDC issued new guidelines for classifying and reporting cases of congenital syphilis.[3] The new guidelines broadened the case definition to include all liveborn and stillborn infants with reactive serologic tests for syphilis (STS) delivered by women with a history of untreated syphilis. Such infants are now considered to be "compatible" with a diagnosis of congenital syphilis, regardless of symptoms. This category essentially combined and simplified the "probable" and "possible" classifications of Kaufman's criteria, as well as broadening it to include fetal deaths.

In order to determine how adoption of the new CDC criteria would affect the reported rates of congenital syphilis, the Los Angeles County (California) Sexually Transmitted Disease Program reviewed its 1987 congenital syphilis caseload.

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Methods

In Los Angeles County, all cases of syphilis and all laboratory findings consistent with syphilis must be reported to the Sexually Transmitted Disease (STD) Program of the Department of Health Services.[4] Because California reporting requirements only include symptomatic cases of "probable" and "possible" congenital syphilis, asymptomatic seropositive infants with no other laboratory findings have not been routinely counted in Los Angeles morbidity reports. Instead, these infants have been classified as "epidemiologically treated" or non-cases, because they received treatment for syphilis without supportive evidence that they were truly infected. All reports of "epidemiologically treated" infants were reviewed to distinguish those infants whose mothers had a documented history of adequate treatment prior to delivery ("unlikely" cases) from those infants whose mothers were not treated for syphilis prior to delivery ("compatible" cases). Unreported infants with negative STS born to untreated syphilitic mothers were not included in the review.

To determine the number of fetal deaths in 1987 that occurred among the women with untreated syphilis, we matched all fetal death records (n = 1133) with all STD records by last name and address. As matching in this way might not identify all such outcomes, we reviewed fetal death and syphilis serology records from three public and two private hospitals reporting the largest numbers of congenital syphilis cases in 1987.

Results

Using California's congenital syphilis reporting criteria, Los Angeles County reported 39 cases of congenital syphilis in 1987. An additional 189 infants received presumptive treatment in 1987. Of these, 115 could be classified as "compatible" with a diagnosis of congenital syphilis under the new CDC criteria. In addition, the review of fetal death

TABLE 1—Race-Specific Rates* of Congenital Syphilis in Los Angeles County (California) for 1987

Race	Reportable by Existing Criteria		Reportable by New CDC Criteria		Proportionate Increase
	NUMBER	RATE	NUMBER	RATE	
White	1	2.3	11	25.7	11.1
Black	26	115.3	117	519.0	4.5
Hispanic	12	15.0	59	73.9	4.9
Asian-American	0	0.0	2	16.1	
Unknown	0	0.0	16	391.8	
Total	39	26.0	205	136.7	4.9

*Cases/100,000 total births

TABLE 2—Compatible Congenital Syphilis by Institution of Birth, Los Angeles County, 1987

Race	Public hospitals		Private hospitals			
	n	Rate per 100,000*	n	Rate per 100,000*	Relative Risk	95% CI
White	3/959	312.8	8/41,875	19.1	16.37**	(6.17, 43.5)
Black	51/2,919	1,747.2	66/19,624	336.3	5.19*	(3.75, 7.20)
Hispanic	39/26,681	146.2	20/53,149	37.6	3.88*	(2.36, 6.40)
Asian	2/495	404.0	0/11,904	0		
Unknown	4/331	1,208.5	12/3,751	319.9	3.78**	(1.32, 10.80)
TOTAL	99/31,385	315.4	106/130,303	813.5	3.88*	(3.01, 5.00)

* per 100,000 births (liveborn and stillborn)
*calculated using Yates Corrected Chi-Square
**calculated using Fischer's Exact Chi-Square

records identified 42 among women with untreated syphilis, four among women with no documentation of treatment for syphilis, and five among women who had been treated within the week preceding the stillbirth. The total of 205 cases represent nearly a five-fold increase in the risk of congenital syphilis in Los Angeles County.

When stratified by race, the 39 officially reported cases in 1987 included only one White and no Asian-Americans (Table 1). When all 205 cases were included all race groups were represented. The new rates of congenital syphilis were highest among Blacks, 7.0 times and 20.2 times higher than Hispanics and Whites, respectively.

When cases were divided by ethnicity and source of report, infants born at public hospitals were more likely to be diagnosed as being "compatible" with congenital syphilis than infants born at private hospitals for all race groups (Table 2).

Discussion

Using the expanded case definition, cases of congenital syphilis increased nearly five-fold in 1987, and even this increase is probably an underestimate. Laboratory reporting of reactive STS results is often variable and, particularly in the private sector, pregnancy status may not be reported with a reactive serology. STS is not mandatory at delivery in California; 48 percent (19/39) of the symptomatic reported cases in 1987, diagnosed only when they developed the florid symptoms of congenital syphilis, were missed at delivery. It is probable that many more at-risk infants were not identified due to lack of screening.

The high rate of congenital syphilis among Blacks is consistent with the rates of reported infectious syphilis in Los Angeles (Blacks 300.6/100,000, Whites 10.3/100,000, Hispanics 42.5/100,000).[5] In our series Black infants were more likely to be among those who were not diagnosed until symptoms developed many weeks after delivery (18/19). They were less likely to have prenatal care and more likely than Hispanics to deliver in private sector hospitals where delivery screening is not routine.

Public hospitals in Los Angeles County routinely perform the STS in the first and third trimesters of pregnancy and at delivery. While women who receive care there may be at greater risk for syphilis, they are more likely to be diagnosed with syphilis than in private hospitals where screening is at physician discretion. Since the distribution of livebirths and stillbirths in public hospitals differs from the distribution countywide, overrepresenting Hispanics and underrepresenting all other race groups, these racial-ethnic comparisons may be inaccurate.

The 205 cases of congenital syphilis defined by the new CDC criteria are a better reflection of the extent of the local congenital syphilis problem than the 39 defined by a modified Kaufman's criteria because more infants at risk are included—not just those few who develop classic symptoms of congenital syphilis. While the new CDC criteria overestimate the cases by including uninfected infants who may have a reactive STS due to passive transfer of maternal antibody, many syphilis-related stillbirths are uncounted because the mother is not screened at delivery and the association is never made. Because there is no way to differentiate whether an asymptomatic infant born to a mother with untreated syphilis is healthy or infected, all at-risk infants are treated and should be counted, regardless of whether they have reactive STS. In Los Angeles County, because four infants born with non-reactive STS in 1988 were not appropriately treated at delivery (their mothers' sera were reactive), they subsequently developed florid symptoms of congenital syphilis.

These data demonstrate that reported cases of syphilis are greatly affected by both definition, reporting criteria, and maternal delivery screening practices. For rates to be comparable between reporting jurisdictions, a clear case definition and universal delivery screening for mothers of both stillbirths and livebirths are necessary.

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End State Renal Disease among Native Americans, 1983–86

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Abstract: We used data reported to Medicare from 1983 through 1986 to determine the incidence of end-stage renal disease (ESRD) among Native Americans and Whites in the United States. The 1,075 Native American cases represented an annual incidence, age-adjusted to the White population, of 269 per million, 2.8 times the rate for Whites. Fifty-six percent of Native American cases and 27 percent of the White cases were attributed to diabetes, indicating that ESRD is a major problem. Diabetes control provides the greatest opportunity for prevention. (*Am J Public Health* 1990; 80:318–319.)

Introduction

Chronic diseases, including end-stage renal disease (ESRD) and diabetes, are major public health problems for Native Americans; in the period 1981–83, their age-adjusted mortality rate due to renal disease was 11.7 per 100,000, while the 1982 rate for all US races was 4.5 (National Center for Health Statistics data provided by Thomas Welty, MD, MPH). Although the incidence of ESRD among all racial groups appears to have risen dramatically since the mid-1970s, when federal funding for ESRD treatment was initiated, the increase has been more rapid among the minority populations than among the White population.[1,2]

To compare the distribution of ESRD treatment onset among Native Americans and Whites, we examined the Health Care Financing Administration's ESRD Medical Information System. This system, designed for fiscal management, includes data on over 90 percent of ESRD treatment in the United States.[1]

Methods

The HCFA Medical Evidence Report form is completed for every patient receiving dialysis or renal transplantation reimbursed by HCFA by the medical care provider. Race categories are "Native American" (American Indian or Alaskan Native), Asian or Pacific Islander, Black, White, and unknown. (Ethnicity has not been uniformly recorded.) We reviewed data for all ESRD referrals who were identified as "Native American/American Indian" and as "White" for the period 1983–86: age, sex, year of treatment initiation, state of residence at the time of initiation, and primary diagnosis.

To determine the extent of one form of race misclassification, the case reports designated as American Indian for the period 1982–86 were examined for any names that suggested apparent origin from India or other Asian or Pacific Island countries.

The estimated number of diabetic Native Americans in 1987 was 72,000 in a total population of 1.6 million (diabetes prevalence from Indian Health Service ambulatory care data, provided by Sarah Valway, DMD, MPH); and the national figure for diabetic Whites in 1985 was 4,892,000 out of a total population of 197,576,000.[3] We used these national estimates to calculate the incidence of ESRD among Native Americans and Whites and to determine the association between ESRD and diabetes. Population data from the 1980 US Census were used to calculate overall, age-specific, and sex-specific rates in Native Americans and Whites. We used the direct method to age-adjust Native American ESRD rates to the 1980 White population.

Results

Of cases of ESRD treatment initiation reported to Medicare from 1983 to 1986, 1,075 were categorized as American Indian and 75,291 as White. Fifty-eight percent of the Native American cases resided in the five states with the largest Native American population, in which the crude incidence was 219 per million, compared to 140 per million in 42 other states reporting 1 to 50 American Indian cases.

The age-adjusted incidence of ESRD for Native Americans was 269 per million, 2.8 times that for Whites (Table 1). The age-specific rate ratios for Native Americans were highest in the 45–64 age group.

The proportion of ESRD attributed to diabetes was greater for Native Americans than for Whites (55.5 percent vs. 26.9 percent, $p < 0.01$) (Figure 1). In the Native American total population, the age-adjusted incidence of ESRD attributed to diabetes was 149 per million, 5.8 times the rate for Whites.

In the estimated Native American diabetic population, the incidence of ESRD attributed to diabetes was 2,073 per million, 2.0 times the rate for Whites. The incidence of ESRD associated with other causes in the non-diabetic population was 78 per million for American Indians, 1.1 times the rate for

TABLE 1—Annual Incidence of ESRD Treatment Reported to Medicare among American Indians and Whites, United States, 1983–86

Age (years)	Incidence[a]		Rate Ratio (95% CI)
	American Indian	White (referent)	
0–14	9	8	1.1 (0.4,3.0)
15–24	31	24	1.3 (0.7,2.4)
25–34	93	58	1.6 (1.0,2.4)
35–44	229	94	2.4 (1.8,3.4)
45–54	546	124	4.4 (3.4,5.7)
55–64	889	220	4.0 (3.2,5.1)
65–74	1042	340	3.1 (2.3,4.0)
75+	657	274	2.4 (1.5,3.8)
Total			
Crude	168	95.3	1.8 (1.6,2.0)
Age-adjusted	269[b]	95.3	2.8 (2.6,3.1)

[a]Rates per million population; based on the 1980 US Census.
[b]Direct age-adjustment using 1980 US Census estimate.

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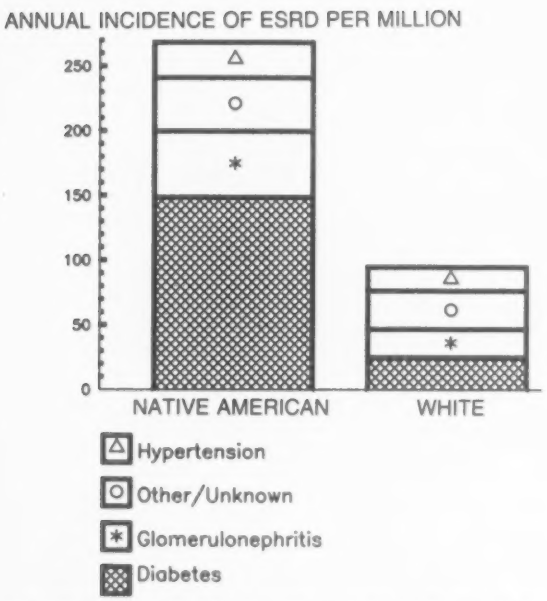

FIGURE 1—Incidence of End-Stage Renal Disease, by Primary Diagnosis, Native Americans and Whites, 1983–86*
*Native American Rate Age-Adjusted to White Population
SOURCE: ESRD data from Medicare, Health Care Financing Administration

Whites. The relative risk for ESRD associated with diabetes is 27 for Native Americans, compared with 14 for Whites.

Review of the ESRD cases from 1982–86 classified as American Indian revealed that the names in 7.4 percent of the cases (93/1265) were highly suggestive of origin from India or other Asian or Pacific Island countries. This apparent race misclassification was less prevalent in cases attributed to diabetes (3.8 percent or 26/678) than in those attributed to other causes (11.4 percent or 67/587), and in the reports submitted from the five states with more than 50 Native American ESRD cases (4.7 percent or 35/739) than from the other states (11.0 percent or 58/526). There was no association between apparent race misclassification and the sex of the referred patient, the age of the referred patient, the year of referral, the incidence rate of ESRD, or the size of the Native American population within the state of referral.

Discussion

For Native Americans, the age-adjusted incidence of ESRD treatment reported to Medicare from 1983 through 1986 was 2.8 times that for Whites. The majority of Native American ESRD cases were attributed to diabetes; the diabetes-attributed incidence was 5.8 times that for Whites. These data are consistent with other measures of the burden of diabetes for Native Americans.[4] While the prevalence of diabetes is higher in Native Americans, the higher incidence of diabetes-attributed ESRD in its diabetic population than among Whites with diabetes suggests that severity of diabetes and other factors among Native Americans are also involved. However, this finding may also be due to an underestimate of the prevalence of diabetes in Native Americans.

Our review of names of cases designated as American Indian indicates that the observed incidence of ESRD is not substantially inflated by one type of race misclassification, that of apparent Asians as American Indian. Because of mixed heritage, there may also be some misclassification between Whites and American Indians. It is likely that misclassification of American Indians as White exceeds misclassification of Whites as American Indian, suggesting that the observed American Indian incidence may be an underestimate. Further study of race misclassification may add to the utility of the ESRD data set for surveillance by the Indian Health Service.

Although the type of diabetes was not designated, Native Americans have a high prevalence of non-insulin dependent diabetes mellitus and a low prevalence of insulin-dependent diabetes, which is consistent with their older age of onset of ESRD. A recent study of the Pima Indians demonstrated a high incidence of ESRD attributed to non-insulin dependent diabetes, and a strong association with its duration.[5]

These findings indicate the need for prevention of ESRD in Native Americans. Potential interventions include the primary prevention of non-insulin dependent diabetes[6] and early identification of renal disease in persons with diabetes, to intensify diet and blood pressure control.[7]

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Mortality from Infectious Diseases among New Mexico's American Indians, Hispanic Whites, and Other Whites, 1958–87

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Abstract: To examine ethnic differences in infectious disease-related mortality in New Mexico's American Indian, Hispanic White and other White populations, we analyzed vital records data from 1958 to 1987. We found that for most infectious causes, American Indians had the highest mortality rates, followed by Hispanics. The state's minority populations remain at increased risk for infectious disease mortality. (*Am J Public Health* 1990; 80:320–323.)

Introduction

Trends in infectious disease mortality among minority groups in the United States have not been adequately examined. New Mexico has three major ethnic populations: American Indians, Hispanic Whites and other Whites. The cultural and genetic backgrounds of these populations are distinct, and the three ethnic groups have differing levels of education and income, with over 40 percent of American Indians and 23 percent of Hispanics living below the poverty level.[1] Many American Indians in New Mexico live traditional life-styles, often in crowded homes with no running water.[2] Despite gradual economic improvement in the state, many disadvantaged minority persons still live in conditions similar to those in developing countries,[3–5] with the attendant risks of acquiring many types of infectious diseases. To examine ethnic differences in infectious disease-related mortality in New Mexico, we analyzed vital records data collected from 1958 to 1987 for American Indians, Hispanic Whites, and other Whites in the state.

Methods

We obtained coded death certificate data for New Mexico residents for the years 1958–87 from the New Mexico Bureau of Vital Statistics. Cause of death on the certificates was coded according to seventh, eighth, and ninth revisions of the International Classification of Diseases (ICD) for the years 1958–68, 1969–78, and 1978–87.[6–8] Conforming to ICD categories, we examined data for: tuberculosis, other (non-tuberculous) infections and parasitic diseases, influenza, pneumonia, kidney infections, and meningitis. Due to ICD classification coding changes, we did not examine diarrheal disease.

The Bureau of Vital Statistics assigned ethnicity according to information on the death certificates. Hispanic ethnicity was determined by surname of the decedent and the decedent's parents, and specific statements on the death certificates; other White ethnicity by coding as "White" on the certificate, and neither a Spanish surname nor other information to indicate otherwise; and American Indian ethnicity by information on the death certificate. American Indian cases were primarily of the Navajo, Pueblo, Zuni, and Apache tribes. Small numbers of cases prevented inclusion of other racial/ethnic groups in the study. The Bureau of Vital Statistics' assignment of Hispanic and other White ethnicity correlated highly with self-reported ethnic status.[9,10] Among Hispanic cases, no attempt was made to distinguish between recent immigrants to the state and the descendants of early settlers to New Mexico.

Denominators for rate calculations were derived from the censuses of 1960,[11–13] 1970,[14,15] and 1980.[1] Since different techniques were used in each census by the US Census Bureau to identify ethnicity, we adjusted estimates of the Hispanic population from 1960 to 1980 to account for the different enumeration procedures.[9,10] Because of coding changes among the ICD revisions, we standardized case counts to conform to the coding scheme of the eighth revision with the National Center for Health Statistics' comparability ratios.[16,17]

Age-specific and age-adjusted mortality rates were calculated by infectious disease category for the five-year periods, 1958–62, 1963–67, 1968–72, 1973–77, 1978–82, and 1983–87. Age-adjusted rates were standardized to the 1970 US standard million population. We used the Statistical Analysis System[18] for our analyses.

Results

The data show substantial differences in infectious disease mortality among New Mexico's three major ethnic populations during the 30-year study period (Tables 1, 2). Age-adjusted mortality rates for most categories of infectious diseases were generally highest among American Indians, followed by Hispanics, for each time period of the study. The greatest rate differences were evident in most periods for tuberculosis, pneumonia, meningitis, other infections, and parasites, with American Indian rates far exceeding rates for Hispanics and other Whites (Tables 1, 2). Ethnic differences for influenza and kidney infections were less striking (Tables 1, 2).

Dramatic declines in mortality rates were observed for American Indians for tuberculosis and for other infections and parasites (Tables 1, 2). However, mortality for tuberculosis, pneumonia, other infections and parasites tended to be higher for American Indians in the latter years of the study

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TABLE 1—Infection-related Mortality among New Mexico Males, by Ethnic Group, 1958–87 (age-adjusted rates per 100,000)

	1958–62	1963–67	1968–72	1973–77	1978–82	1983–1987	% change in rates from 1958 to 1987
Tuberculosis							
Hispanic	13.4	9.6	5.1	3.8	3.0	1.1	−91.8
American Indian	40.5	40.5	27.3	23.5	10.7	11.7	−71.1
Other White	9.4	6.5	3.6	2.0	1.0	1.1	−88.3
US White*	7.8	5.3	3.3	1.2	1.2	0.9	−88.5
US Black*	22.6	16.9	12.1	7.1	6.1	4.9	−78.3
Other Infections and Parasites							
Hispanics	17.6	13.8	9.6	6.7	5.8	10.7	−39.2
American Indian	39.7	44.6	28.0	20.8	15.4	18.3	−53.9
Other White	5.7	4.7	4.9	4.5	6.0	8.4	+47.4**
US White	5.5	4.9	5.6	5.6	7.9	12.4	+125.5**
US Black	16.4	13.2	13.9	14.3	18.8	28.9	+76.2**
Influenza							
Hispanic	4.5	2.5	3.0	4.9	1.1	0.1	−97.8
American Indian	8.1	2.2	0.9	5.9	0.0	0.0	−100.0
Other White	2.9	2.3	3.9	4.4	0.9	0.5	−82.3
US White	4.6	1.2	2.1	2.3	1.1	0.7	−84.8
US Black	11.2	1.9	3.3	1.7	0.4	0.5	−95.5
Pneumonia							
Hispanic	50.7	44.5	56.9	52.2	36.3	33.9	−33.1
American Indian	88.5	64.5	81.2	89.4	48.3	51.6	−41.7
Other White	34.8	34.3	41.0	42.3	24.8	34.0	−2.3
US White	38.3	36.6	35.1	30.1	28.0	31.0	−19.1
US Black	72.6	63.8	61.6	44.3	40.6	39.9	−45.4
Kidney Infection							
Hispanic	4.5	5.2	4.6	1.6	1.0	0.1	−97.8
American Indian	10.6	8.6	4.5	1.0	2.5	1.5	−85.8
Other White	3.5	4.0	3.0	1.9	0.7	0.6	−82.3
US White	4.6	5.3	3.8	1.9	0.9	0.6	−87.0
US Black	11.0	12.6	9.1	3.8	1.7	1.1	−90.0
Meningitis							
Hispanic	1.2	1.2	0.8	2.1	1.0	0.4	−66.7
American Indian	7.0	10.8	8.2	1.6	2.5	0.5	−92.9
Other White	1.4	1.6	0.5	1.4	0.6	0.3	−78.6
US White	0.7	0.7	0.8	0.8	0.7	0.5	−28.6
US Black	2.3	3.2	2.3	2.2	1.8	1.3	−43.5

*Age-adjusted rates calculated at the midpoints of the time intervals.
**Rate increases primarily reflect increases in coding of deaths from zoonotic and other bacterial diseases, septicemia, and mycoses.

than for other Whites in the period 1958–1962, 30 years earlier.

Because of elevated mortality rates for infectious causes among young children in New Mexico, we examined age-specific mortality for pneumonia, meningitis, other infections, and parasites by sex and ethnic group among children ages 0–4 years. These data show trends similar to the age-adjusted data for Hispanics, American Indians, and other Whites in the state, with the highest mortality rates for the American Indian children (Table 3).

Discussion

Our data show that New Mexico's three major ethnic populations followed the national trend[19] of decreasing infectious disease-related mortality, but also indicate substantial ethnic differences in these mortality rates. These differences in mortality rates may be influenced by the disparate cultural beliefs and medical practices in these ethnic groups. Many Navajo, Pueblo, Zuni, and Apache people prefer traditional medicine men to modern medical treatment,[3,4] and some Hispanics use traditional curanderos.[20,21] Any consequent delay in seeking modern medical attention may increase mortality rates. Lack of health care availability can also affect mortality rates. In New Mexico, approximately one-third of Hispanics are medically uninsured, and many of the predominantly Hispanic rural counties are medically underserved.[22] Although the Indian Health Service provides care at no cost to the American Indians, long distances and unimproved roads also lessen health care availability, and have been demonstrated to be a risk factor for Navajo children presenting with infectious diseases.[23]

Our analyses of vital data have certain limitations; these potential sources of bias have been discussed in detail in prior publications.[9,10] In addition, these analyses cannot determine whether the incidence of infections is higher in the minority than in the White majority populations, or whether the incidence of infections is comparable, but the case-fatality ratios are higher in minority populations. Few published studies have directly addressed these issues from a population-based perspective on infectious disease incidence and mortality in the United States. The data from this study emphasize the importance of targeting the state's high-risk minority populations in provision of timely and appropriate medical care to reduce the high rates of mortality from infectious causes in New Mexico.

TABLE 2—Infection-related Mortality among New Mexico Females, by Ethnic Group, 1958–87 (age-adjusted rates per 100,000)

	1958–62	1963–67	1968–72	1973–77	1978–82	1983–1987	% change in rates from 1958 to 1987
Tuberculosis							
Hispanic	9.8	4.1	2.2	2.4	1.5	1.0	−89.8
American Indian	38.0	30.1	15.8	12.8	11.6	10.2	−73.2
Other White	3.7	1.7	0.5	0.7	0.8	0.6	−83.8
US White*	2.4	1.5	1.0	0.7	0.4	0.4	−83.3
US Black*	8.8	6.1	4.4	2.7	2.2	1.7	−80.7
Other Infections and Parasites							
Hispanics	11.9	7.4	7.6	6.4	6.7	6.9	−42.0
American Indian	37.0	38.3	22.5	20.9	12.2	4.5	−87.8
Other White	5.7	4.7	4.9	4.5	6.0	5.7	0
US White	3.5	3.2	4.3	3.9	5.6	8.1	+130.4**
US Black	9.3	8.5	10.0	10.7	13.6	19.1	+105.4**
Influenza							
Hispanic	2.8	3.1	4.7	4.6	0.6	0.2	−92.9
American Indian	4.9	1.5	2.5	3.1	0.7	0.8	−85.4
Other White	2.8	1.2	1.8	3.6	0.7	0.6	−78.6
US White	3.5	1.0	1.5	1.9	1.1	0.8	−77.1
US Black	9.0	2.1	2.4	1.2	0.4	0.3	−96.7
Pneumonia							
Hispanic	38.2	32.5	39.9	33.8	22.4	21.9	−42.7
American Indian	65.8	55.0	61.8	47.2	36.9	28.0	−57.4
Other White	19.2	18.7	25.4	26.0	17.0	21.3	+10.9
US White	21.3	22.4	20.8	16.9	17.3	19.8	−7.0
US Black	43.7	44.0	40.2	20.9	19.2	19.9	−54.5
Kidney Infection							
Hispanic	2.9	6.0	3.8	1.0	0.4	0.7	−75.9
American Indian	12.6	15.4	7.7	4.0	0.0	2.1	−83.3
Other White	2.6	3.6	3.7	1.6	0.7	0.6	−76.9
US White	3.7	4.6	3.5	1.9	1.1	0.7	−81.1
US Black	10.1	10.9	7.9	3.3	1.7	1.0	−90.1
Meningitis							
Hispanic	1.1	1.0	1.3	0.7	0.8	0.3	−72.7
American Indian	7.6	8.7	4.4	5.4	3.5	0.3	−96.1
Other White	0.5	1.1	1.0	0.8	0.3	1.2	+140.0
US White	1.0	1.0	0.6	0.6	0.5	0.4	−60.0
US Black	3.1	2.4	1.8	1.5	1.3	1.0	−67.7

*Age-adjusted rates calculated at the midpoints of the time intervals.
**Rate increases primarily reflect increases in coding of deaths from zoonotic and other bacterial diseases, septicemia, and mycoses.

TABLE 3—Infection-related Mortality among Children Ages 0–4 Years in New Mexico, 1958–87, by Cause and Ethnic Group (age-specific rates per 100,000)

	1958–62	1963–67	1968–72	1973–77	1978–82	1983–1987	% change in rates from 1958 to 1987
Pneumonia							
Males							
Hispanic	131.3	71.0	52.8	23.9	10.5	5.4	−95.9
American Indian	299.8	226.1	150.7	50.0	15.4	25.5	−91.5
Other White	46.0	36.8	30.7	22.8	6.7	4.0	−91.3
Females							
Hispanic	104.2	66.4	46.1	20.4	4.3	7.1	−93.2
American Indian	297.2	151.4	96.4	30.9	10.8	5.7	−98.1
Other White	48.1	35.2	25.6	21.0	6.6	6.1	−87.3
Meningitis							
Males							
Hispanic	5.8	6.4	3.6	10.0	6.5	0.8	−86.2
American Indian	67.3	77.7	59.9	6.7	12.9	5.5	−91.8
Other White	10.7	16.2	3.0	7.3	4.3	0.8	−92.5
Females							
Hispanic	5.1	8.5	9.4	1.7	4.1	2.3	−54.5
American Indian	50.5	65.8	29.6	29.6	9.2	0.0	−100.0
Other White	3.2	7.4	3.2	5.7	2.8	0.0	−100.0
Other Infections and Parasites							
Males							
Hispanic	110.8	60.7	26.8	16.5	16.0	7.6	−93.1
American Indian	323.8	290.6	166.8	80.0	30.5	21.7	−93.3
Other White	22.1	18.5	13.2	11.0	4.6	2.6	−88.2
Females							
Hispanic	98.5	39.9	30.7	18.7	11.6	2.6	−97.4
American Indian	246.9	197.0	98.0	72.4	16.1	3.1	−98.7
Other White	23.1	11.2	12.7	11.5	11.0	5.7	−75.3

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Feasibility of a Telephone Survey to Study a Minority Community: Hispanics in San Francisco

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Abstract: In two random digit dialing surveys conducted among Hispanics using a modified Mitofsky-Waksberg procedure, we found low refusal rates (4.7% and 3.1%), low assumed noncontact rate (14.0% and 18.3%), and high response rates (88.6% and 88.4%) with limited investment in time (1.58 hours and 1.66 hours per completed interview). These results suggest that Hispanics are willing to participate in telephone surveys and that this method may be feasible and useful for research and evaluation purposes. (*Am J Public Health* 1990; 80:323–326.)

Introduction

Surveys conducted over the telephone can be expected to be particularly useful to study minorities since they provide researchers with an efficient method to collect data with acceptable response rates and low interviewer bias.[1–4] Telephone surveys also allow the use of moderately long interview schedules regardless of respondents' reading and writing abilities. In addition, telephone surveys generally cost less than personal interviews. On the other hand, several investigators have suggested there are problems in identifying and sampling Hispanics; obtaining respondent cooperation; designing valid interview schedules; and controlling biases that may be introduced due to the ethnicity of the interviewer and the language used in the interview.[5–7]

The absence of a telephone at home among a significant proportion of potential respondents can be expected to decrease the representativeness of a sample and limit the generalizability of the results of a survey. Most individuals in the United States have a telephone in their household.[4] In San Francisco, for example, the 1980 Census found that 92 percent of self-identified Hispanic households in those census tracts with 400 or more Hispanics had at least one telephone compared with 94 percent for non-Hispanics. In other major cities telephone ownership among Hispanics averages 85 percent.[8]

We used the random digit dialing procedure for telephone surveys suggested by Mitofsky and Waksberg[9] which eliminates the use of nonworking banks (prefixes) and avoids the use of directories (eliminating the problem of excluding

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unlisted numbers) while guaranteeing equal probability of selection for all residential telephone numbers.[1] A recent study with Blacks[10] found this approach particularly efficient in obtaining samples from highly clustered subgroups as is the case of many ethnic/racial minorities living in urban areas of the United States. This paper describes two surveys in which the Mitofsky-Waksberg approach was used to sample Hispanics.

Methods

The surveys were designed to obtain estimates of the prevalence of cigarette smoking[11] and to serve as baselines for the evaluation of a community intervention designed to lower cigarette smoking prevalence among Hispanics in San Francisco. Each interview lasted approximately 15 minutes and included approximately 60 questions dealing with smoking behavior, standard socio-demographic variables, and an acculturation scale.[12] The interviews were conducted in the language of choice of the respondents; 67 percent and 69 percent preferred to use Spanish in the first and second studies, respectively. Interviewers were bilingual (English and Spanish) men and women who received at least 12 hours of training, including role playing and supervised interviewing, before they participated in the study. Training of interviewers was based on procedures for telephone interviewing developed by the Minnesota Heart Health Program and on materials from the Survey Research Center of the University of Michigan.[13]

A household was judged to be eligible when the respondent identified himself/herself as Hispanic, or identified the majority of the residents in the household as "Hispanics." Hispanic ethnicity was ascertained by the respondents' answer to the question "Do you or most of the people in your household consider yourselves to be Hispanics, Blacks, Asians, or Caucasians?" The response to this question was later checked with the respondents' and their parents' reported place of birth and with the respondents' identification with one of the Hispanic subgroups.

Prefix Selection

We selected prefixes that covered census tracts with the largest concentration of Hispanics, according to 1980 census data. This identified 27 tracts with 10 percent or more Hispanic residents. These tracts represented 66.6 percent (N = 55,541) of all Hispanics (N = 83,373) in the city of San Francisco.

In each census tract with high concentrations of Hispanics, three city streets were randomly selected to locate telephone prefixes assigned to residences. This process produced 49 three-digit prefixes out of the 121 prefixes in use in San Francisco. These procedures were conducted separately for each study and in both cases the same 49 three-digit prefixes were identified and used.

Stage One Sampling

A random list of 2,000 different four-digit numbers was generated by a computer and the numbers were randomly assigned to the 49 prefixes. These seven-digit telephone numbers were then assigned to interviewers. When a Hispanic household was reached, the first five digits of that telephone number became a Primary Sampling Unit (PSU). During Study I, a total of 4,326 working numbers were called as part of this first stage in the sampling procedure yielding 259 PSUs. Study II produced 355 PSUs out of the 4,680 working numbers called. Tables 1 and 2 present a summary of the outcome of the calls made in this stage during both studies.

TABLE 1—Summary of Telephone Calls for Study I

Outcome	Stage I N (%)	Stage II N (%)	Total N (%)
Total working numbers called	4,326 (100)	12,107 (100)	16,433 (100)
Eligible			
Completed	286 (6.6)	1,383 (11.4)	1,669 (10.2)
Refusals	20 (.5)	63 (.5)	83 (.5)
Ineligible			
Business	852 (19.7)	1,704 (14.1)	2,556 (15.6)
Ethnicity	2,232 (51.6)	8,605 (71.1)	10,837 (65.9)
Age	35 (1.0)	114 (1.0)	149 (1.0)
Lost to survey	901 (20.1)	238 (2.0)	1,139 (6.9)
Total screened	3,425 (79.9)	11,869 (98.0)	15,294 (93.1)
Response rate	(74.1)	(93.8)	88.6
Refusal rate	(6.5)	(4.3)	(4.7)
Assumed noncontact rate	(42.4)	(4.0)	(14.0)

TABLE 2—Summary of Telephone Calls for Study II

Outcome	Stage I N (%)	Stage II N (%)	Total N (%)
Total working numbers called	4,680 (100)	16,823 (100)	21,503 (100)
Eligible			
Completed	365 (7.8)	1,694 (10.1)	2,059 (9.6)
Refusals	11 (.2)	55 (.3)	66 (.3)
Ineligible			
Business	1,245 (26.6)	3,347 (19.9)	4,592 (21.4)
Ethnicity	2,735 (58.4)	9,942 (59.1)	12,677 (59.0)
Age	40 (.9)	167 (1.0)	207 (1.0)
Lost to survey	284 (6.1)	1,618 (9.6)	1,902 (8.8)
Total screened	4,396 (93.9)	15,205 (90.4)	19,601 (91.2)
Response rate	(91.3)	(87.5)	(88.4)
Refusal rate	(2.9)	(3.1)	(3.1)
Assumed noncontact rate	(15.9)	(18.8)	(18.3)

Stage Two Sampling

A computer-generated list of 99 random two-digit numbers was used to produce the suffixes for each of the PSUs. Each interviewer continued using a given PSU until eight Hispanic households were reached including completed interviews or refusals, or until the list was exhausted including four follow-up calls made within a four-week period to every unanswered number. Tables 1 and 2 present a summary of the outcome of the numbers called in this stage during both studies. A total of 92 PSUs (36 percent) were exhausted before reaching eight Hispanic households during the first study. The corresponding figure for the second study was 95 (25 percent).

Interviewee Eligibility

Within a Hispanic household, the respondent was selected by asking for the resident who had most recently celebrated a birthday and who was between 18 and 65 years of age. Approximately 8 percent of otherwise eligible households failed to meet this criterion (Tables 1 and 2).

A total of 1,669 respondents were interviewed during Study I (Fall 1986). Of these, 44 percent were males and the majority of the respondents (69 percent) were foreign-born with 60 percent of them reporting being born in Central America, 29 percent in Mexico, and the remainder in South America, Cuba, or Puerto Rico. The respondents' mean age was 35.5 years and they reported a mean number of years of

education of 11.6. In Study II (Summer 1987), 2,059 Hispanics were interviewed (42 percent men). The respondents' mean age was 35.5 years while the mean number of years of education was 11.6. The majority (70 percent) were born outside the United States, 60 percent of them in Central America, 28 percent in Mexico, and the remainder in either Cuba, Puerto Rico, or South America.

Results

Response/Refusal Rates

Tables 1 and 2 show the response and refusal rates for both studies. We have adapted Kviz's[14] definition of response rate which is the proportion of completed interviews in terms of the number of eligible respondents (defined here as Hispanics between 18 and 65 years of age). This calculation assumes that those not contacted after four follow-up calls ("lost to survey") were eligibles.[15] This proportion was assumed to be the same as the proportion of eligibles found among telephone numbers that were fully screened. The Stage I response rate, assumed that the proportion of eligibles found among the screened calls 306/3425 (8.9 percent) would be similar to that among the 901 numbers that were "lost to survey." Adding the 80 assumed eligible numbers yielded a response rate of 286/386 (74.1 percent). Response rates for the other stages of both studies were calculated in the same manner noted here and show an overall response rate of 88.6 percent for Study I and 88.4 percent for Study II.

The refusal rates were defined[1] as the proportion of eligible respondents (Hispanics between 18 and 65 years of age) who were contacted but declined to be interviewed. Refusal rates were only 4.7 percent for Study I and 3.1 percent for Study II.

The Assumed Noncontact Rate[1] is an index of the accessibility of respondents or the probability of researchers locating respondents. This index is made up of the proportion of respondents not contacted after four follow-up calls ("lost to survey" in both tables) in relation to the total number of possible eligible respondents. Given that a significant proportion of telephone numbers in San Francisco are unlisted (38 percent according to Pacific Bell), it is not appropriate to estimate the true number of eligible households from an alphabetic telephone directory. Thus, in computing the assumed noncontact rate we are assuming that 25 percent of those respondents "lost to survey" were eligible Hispanics. This assumption reflects twice the number of Hispanics reported to be living in San Francisco in 1985 and therefore produces a very conservative computation of nonresponse rates. In Study I for example, 25 percent of the 1,139 "lost to survey" (N = 285) represent 14 percent of the total number of assumed Hispanic numbers: completed interviews (N = 1,669) plus refusals (N = 83) plus lost to survey (N = 285). The comparable noncontact rate for Study II is 18.3 percent. Figures taken from Wiseman and McDonald's[16] review of the literature show a median noncontact rate of 39.1 percent.

Cost of Survey

Based upon the time spent by all interviewers, we estimated costs of obtaining a completed interview. On the average, interviewers needed to spend 1.58 hours in order to obtain a completed interview during Study I and 1.66 hours during Study II. Each of the completed interviews cost approximately $15 based on paid interviewer and supervisor salaries but excluding overhead, training time, telephone lines, investigators' time, and data management. Interviewers were paid $7.90 per hour in this study.

Discussion

These results suggest that it is feasible and efficient to use random digit dialing techniques to obtain a sample of Hispanics. The similarity in the outcomes of the two studies imply that the findings are fairly stable across time.

The high completion rates and the correspondingly low refusal rates obtained in these surveys compare favorably with those found in studies of other Whites, which range between 66 percent in a study in Chicago[4] to 73.5 percent in a regional survey.[17] The low refusal rates (4.7 percent in Study I and 3.1 percent in Study II) are lower than those of other studies. Wiseman and McDonald[16] in their review of 182 studies using telephone surveys found a median refusal rate of 28 percent while Frey[1] reports refusal rates ranging between 20–25 percent. Low refusal rates among Hispanics have also been reported in a study conducted in Texas[18] where a telephone survey of a sample drawn from Spanish-surnamed individuals produced a refusal rate of 8.8 percent.

The high rate of participation in these surveys by Hispanics was not due to advance publicity about the study. Respondents were informed of the purpose of the study only at the time of the interview. One possible explanation for these results is the presence of a cultural characteristic that promotes cooperation and that dictates positive interpersonal relations—the social script of "simpatia."[19] This cultural norm would move Hispanic respondents to agree to be interviewed in order to maintain a fluid and positive social relationship with the interviewer when the relationship is perceived as nonthreatening and as sanctioned by a respected local institution (the study was introduced as being conducted by a state university in San Francisco).

The generalizability of our results may be limited by the following specific concerns:

- The sampling frame used excluded approximately one-third of the San Francisco Hispanic population according to the 1980 census. Hispanics who are living in census tracts excluded in this study (less than 10 percent Hispanic) may behave differently when asked for an interview over the telephone.
- At least 8 percent of San Francisco Hispanic households may not have a telephone at home and these persons are also excluded from the sample.
- The approach utilized in choosing the prefixes may not produce all of the relevant ones although a third study survey recently concluded by the authors has again identified the same 49 prefixes as the first two studies.
- Finally, San Francisco Hispanics (Central American and Mexican in this survey) may differ from Hispanics residing elsewhere.

Future studies should compare samples gathered with this method with census data in order to address the representativeness of a sample of minority-group respondents obtained through a random digit dialing technique such as the Mitofsky-Waksberg approach. Unfortunately, this analysis cannot be done at the present time due to the high undercount of Hispanics in the 1980 census and the later significant migrations from Cuba and Central America.

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Sex-specific and Race-specific Hip Fracture Rates

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Abstract: Sex-, race- and age-specific hip fracture rates were determined using Health Care Financing Administration data for Medicare-reimbursed hip fracture hospitalizations from 1980 to 1982. Rates were highest in White women, lowest in Black men, and intermediate in White men and Black women. Proportions of hip fracture patients dying during hospitalization and those discharged to nursing homes, respectively, were: White men (10.5%; 49%); Black men (9.3%; 32%); White women (5.0%; 54%); and Black women (8.2%; 30%). (*Am J Public Health* 1990; 80:326–328.)

Introduction

In 1986 in the United States, there were in excess of 250,000 hospital admissions for hip fracture.[1] Elderly individuals who sustain hip fractures experience increased morbidity[2–7] and mortality.[8–12] In an earlier paper, Farmer, *et al*,[13] reported that age-specific hip fracture rates were higher in White women than in White men, Black women, and Black men, but the differences among age-specific rates for White men, Black men, and Black women could not be estimated precisely.

In this paper we report age-, sex-, and race-specific hip fracture rates extended through age 85 years. In addition, we present findings for proportions of patients dying during hospitalization, and discharged to nursing homes, not previously reported for Blacks.

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Methods

Numbers of Medicare-reimbursed hospitalizations for hip fracture in Illinois residents over 65 years of age were obtained from data collected by the Professional Standards Review Organization in Illinois during 1980, 1981, and 1982. The Uniform Hospital Discharge Format was used in the data collection, and the data tape was made available through the Health Care Financing Administration (HCFA). Hip fracture was defined according to the ninth revised International Classification of Diseases[14] categories 820.0–820.9: transcervical fractures, closed and open; pertrochanteric fractures, closed and open; and fractures of unspecified parts of the neck of the femur, closed and open. Populations at risk for hip fracture were obtained from the 1980 United States Census figures for the Illinois population by age, race and sex.

Mean numbers of hip fractures for 1980, 1981, and 1982 were used to calculate the sex-, race- and age-specific annual incidence rates for hip fracture. Miettinen test-based confidence intervals were calculated for the hip fracture relative risks.[15] Age-adjusted odds ratios for death and nursing home placement were determined using the Statistical Analysis System multiple logistic regression program.[16] Race- and sex-specific means for days of hospital stay were adjusted for age with the Statistical Analysis System linear regression program.[17]

Results

Age-group specific hip fracture rates were highest for White women, and lowest for Black men in all age groups after age 70 years (Figure 1). The rates in Black women are

similar to those in White men, and intermediate to those observed for White women and Black men. Hip fracture rates began to increase exponentially after age 70 years in White women, after age 75 in White men and Black women, and at approximately 85 years of age in Black men. Hip fracture rates in White women were approximately twice those in White men from age 65 to 85 years.*

In-hospital hip fracture death rates were highest for White men (10.5%), followed by Black men (9.3%), Black women (8.2%), and White women (5.0%). Age-adjusted odds ratios for death among hip fracture cases are shown in Table 1. After age-adjustment, in-hospital death rates were twice as high for White men as for White women. Black men had only slightly higher death rates than Black women.

The means for lengths of hospital stays were: Black men (28.0 days), Black women (28.2 days), White men (24.2 days), and White women (23.1 days). Discharge status of survivors is shown in Table 2. Both Black men and women were discharged to nursing homes less frequently than White men and women. After age-adjustment, the odds ratio for nursing home placement in White women compared with Black women was 2.70 (95% CI: 2.21, 3.30), and in White men compared with Black men 1.93 (95% CI: 1.16, 2.61).

Discussion

These data are consistent with earlier observations showing highest age-specific hip fracture rates for White women.[13] However, our findings also indicate higher age-specific rates in women than in men among both Blacks and Whites, although the sex difference occurred at older ages in Blacks than in Whites, as did the race-related differences. These findings are consistent with the age-, sex- and race-specific hip fracture rates recently reported for hip fracture hospitalizations in California.[18]

Black men and women may have lower hip fracture rates because, as adults, they exhibit higher levels of bone mass attained during growth and development.[19–25] In addition, race-related differences in the Vitamin D endocrine system suggest that Blacks are protected from the bone-resorptive effects of parathyroid hormone,[26] and reports of lower bone turnover rates in Blacks may result in slower rates of age-related bone mass than occurs in Whites.[27] The race difference in women is consistent with evidence that obesity is more common in Black women which may increase their extra-ovarian production of estrogen,[28] and that Black women may experience lower rates of menopause-associated bone loss than White women.[29]

While risk for falling, in addition to bone mass, influences hip fracture incidence,[30–32] the extent to which falling risk contributes to race- and sex-differences in hip fracture rates remains unclear. Elderly men are reported to fall less frequently than elderly women.[32] While Black women are reported to have greater muscle mass than White women,[33–35] it is not known if muscle mass acts independently of its documented influence on bone mass, to influence falling risk.

The finding of higher mortality rates in White men than in women is consistent with observations from earlier studies.[8–12] The most comparable race-specific mortality data were reported for small numbers of non-White hip fracture patients in which the risk for mortality at three months post hip fracture in non-Whites versus Whites was 1.5 (95% CI: 0.6, 3.6).[12] In addition to length of hospital stay, in-hospital mortality rates are also influenced by the numbers and severity of co-existing illnesses.[12] In the absence of comorbidity and severity of illness data in this analysis, interpretation of the in-hospital mortality data reported is difficult.

Hospital lengths of stay observed in this study are for the period prior to the implementation of the diagnosis-related group (DRG)-based prospective payment system (PPS), and are consistent with those reported by Fitzgerald, *et al*,[36] which also noted that the shorter lengths of stay after the PPS (12.6 days) were associated with fewer in-hospital physical

TABLE 1—Age-adjusted Odds Ratios for Death during Medicare-reimbursed Hospitalization for Hip Fracture, Illinois, 1980–82

	Age-adjusted Odds Ratios for Death	95% CI*
White men vs White women	2.41	2.17, 3.00
Black men vs Black women	1.18	0.53, 2.35
White men vs Black men	1.02	0.63, 2.23
Black women vs White women	1.64	1.09, 2.47

*Miettinen, test-based confidence intervals.[15]

TABLE 2—Discharge Status of Survivors and Duration of Stay for Medicare-reimbursed Hospitalization for Hip Fracture, Illinois, 1980–82.

	White		Black	
	Men (N = 3575)	Women (N = 14,768)	Men (N = 204)	Women (N = 523)
Nursing home (%)	49	54	32	30
Skilled (%)	73	73	69	66
Intermediate (%)	23	24	29	30
Other (%)	4	3	2	4
Home (%)	46	43	65	66
Self-care (%)	91	90	94	93
Home-health (%)	9	10	6	7
Transferred to another hospital (%)	4	3	2	3
Left hospital against medical advice	1	1	1	1

*Exact data available upon request from author.

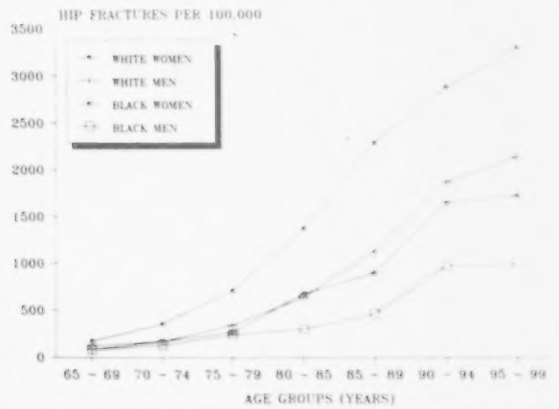

FIGURE 1—Comparison of Sex-specific and Race-specific Hip Fracture Incidence Rates by Age for Illinois Residents, 1980–82

therapy sessions, poorer ability to ambulate on hospital discharge, and higher rates of discharge to nursing homes.

The finding of lower proportions of Black men and women discharged to nursing homes is consistent with race-specific national data for nursing home use.[37]

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The Epidemiology of Vulvovaginal Candidiasis: Risk Factors

BETSY FOXMAN, PHD

Abstract: The effects of personal hygiene, sexual history, diet, and stress on the risk of vulvovaginal candidiasis were estimated from a case-control study of students attending a public university during 1986–87. Data from medical records and self-administered questionnaires were used to compare 85 cases to 1,245 other students using the Health Service, and to 113 subjects chosen from the total student population. Frequent sexual intercourse was the strongest risk factor (seven or more times a week versus none): OR = 4.3; 95% CI: 1.4, 12.9 (for cases versus Health Service controls). (*Am J Public Health* 1990; 80:329–331.)

Introduction

Vulvovaginal candidiasis, a common, irritating and frequently recurring infection, has been associated with broad spectrum antibiotic treatment, diabetes, and pregnancy.[1] Although factors such as diet high in sugar, using tampons or perfumed toilet paper, or wearing tight clothing are suspected to increase risk of vulvovaginal candidiasis,[1,2] few studies have examined these factors in a systematic fashion. In this study, I explored the relationships between personal habits, behaviors, and vulvovaginal candidiasis by analyzing a subset of data from a Health Service-based case-control study of urinary tract infection.[3]

Methods

Cases and controls were collected as part of a previously described study of urinary tract infection.[3] For that study, participants were recruited from women presenting with and without urinary symptoms to the University Health Service between September 1986 and December 1987. Prior to seeing a clinician, participants were given a self-administered questionnaire including questions about medical history, stress, clothing, diet, sexual activity, and birth control method during the past four weeks. The medical records of all participants recruited at the Health Service were reviewed to establish diagnosis and to confirm information on birth control method and history of other infections.

There were two control groups: women using the Health Service during the same time period as the cases (Health Service controls); and a sample stratified by year in school of 200 women enrolled at the University during January, 1988 (student population controls). Of these 200, 170 had complete and accurate addresses and 115 consented to participate. Student population controls completed essentially the same questionnaire distributed at the Health Service, but medical records were not reviewed.

Case and Control Definition

A case was defined as any woman who was recruited during a visit for which the diagnosis listed in her medical record was vulvovaginal candidiasis (n = 85). Health Service controls were women without a clinical diagnosis of vulvovaginal candidiasis recruited from the University Health Service. Population-based controls were limited to women who did not report current infection with either vulvovaginal candidiasis or a urinary tract infection. Both cases and controls were limited to non-pregnant women without urinary tract infection enrolled at the University.

Analysis

Exploratory analyses were conducted using multilevel contingency tables with both stratification and multivariate techniques to control for confounding.[4,5] Odds ratios (OR) and associated 95 percent confidence intervals (95% CI) were calculated from the logistic regression models as described by Schlesselman.[4] A separate model was fit for each factor of interest with age, frequency of sexual intercourse, number of previous infections with vulvovaginal candidiasis, and, for Health Service controls, month of enrollment, included as covariates. Related variables, such as birth control methods, were fit in a single model. All factors of interest were considered as potential confounders of other factors but only those variables that made a difference were included as covariates in the final models. For example, the OR for feminine hygiene use for cases compared to Health Service controls was calculated from a logistic regression model with presence of vulvovaginal candidiasis as the dependent variable, and age, frequency of sexual intercourse, number of prior infections with *Candida*, month of enrollment, and feminine hygiene use as independent variables.

Results

A total of 85 women met the study case definition; 1,245 Health Service and 113 population-sampled participants met the control definition. Study participants were similar to women students enrolled in the University with respect to year in school, age, ethnic group, and marital status. Cases and controls ranged in age from 17 to 72 (mean = 21.8 years). Age, year in school, and marital status were similar for cases and controls. Self-reported history of previous vulvovaginal candidiasis was common among both cases (77.7 percent) and controls (Health Service: 41.2 percent; student population: 19.6 percent). Most previous infections occurred within the past 12 months (cases: 74 percent, Health Service: 60 percent; student population: 62 percent).

When controlled for the confounding effects of age, frequency of sexual intercourse, and month of enrollment, women reporting one previous infection, compared to none, were about twice as likely to have a current infection (Health Service: OR = 1.55; 95% CI: 1.35, 1.78; population: OR = 2.06; 95% CI: 1.58, 2.69), and those with two infections about three times as likely (Health Service: OR = 2.39; 95% CI: 1.81, 3.16; population: OR = 4.25; 95% CI: 2.49, 7.25).

Cases were twice as likely as controls to have been sexually active during the past four weeks; the odds ratio increased with increasing frequency of sexual intercourse for both control groups (Table 1). However, having a new partner or engaging in sexual intercourse with multiple partners during the past four weeks showed no association

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TABLE 1—Associations between Vulvovaginal Candidiasis and Selected Behaviors during the Past Four Weeks

Variables	Cases (n = 85)		Health Service Controls (n = 1245)			Population Controls (n = 113)		
	#	%	#	%	OR (95% CI)	#	%	OR (95% CI)
Weekly frequency of sexual intercourse during the past 4 weeks								
never	19	22.4	557	44.7	reference	68	60.2	reference
< 1 time	14	16.5	192	15.4	1.31 (1.10, 1.57)	15	13.3	1.44 (1.10, 1.90)
1–2 times	20	23.5	231	18.6	1.73 (1.22, 2.45)	11	9.7	2.08 (1.21, 3.60)
3–6 times	25	29.4	218	17.5	2.27 (1.34, 3.84)	17	15.0	3.00 (1.32, 6.82)
7+ times	7	8.2	47	3.8	2.98 (1.48, 6.00)	2	1.8	4.34 (1.45, 12.93)
Birth control method† (sexually active women only)								
No method	15	22.7	16	1.3	reference	9	20.9	reference
Oral contraceptives	35	53.0	373	55.4	1.11 (0.60, 2.02)	23	53.5	1.52 (0.61, 3.74)
Diaphragm	10	15.2	74	37.9	1.23 (0.40, 3.80)	6	14.0	1.67 (0.30, 9.44)
Spermicides	18	27.3	79	11.7	1.01 (0.43, 2.35)	9	20.9	1.40 (0.36, 5.53)
Other methods	21	32.8	264	40.0	0.95 (0.51, 1.78)	22	51.2	1.01 (0.38, 2.70)

NOTE: Odds ratios and their 95 percent confidence intervals (OR, 95% CI) were adjusted for frequency of sexual intercourse, previous infections with vulvovaginal candidiasis, age, and for Health Service controls, month of enrollment, using a logistic regression model (see methods).

†Categories not mutually exclusive. Percents do not sum to 100, as some women used more than one method during the previous four weeks.

with *Candida*. Moreover, no one birth control method was associated with infection (Table 1).

Student population controls were more likely to use colored toilet paper and take tub baths than cases, but Health Service controls reported similar exposures to cases (Table 2). Other suspected risk factors, direction wiped following toileting, menstrual protection, use of feminine hygiene products and clothing showed little association with vulvovaginal candidiasis (Table 2). Diet and stress also showed no association.

Discussion

To my knowledge, no previous controlled study has reported a relationship between sexual activity and vulvovaginal candidiasis although case reports suggest a correlation.[1] Sexual intercourse might facilitate movement of *Candida* into the vagina and result in local minor trauma, creating conditions suitable for yeast to invade tissues. Some authors have suggested that *Candida* can be sexually transmitted;[6] however, in this study neither beginning a new relationship nor having multiple sexual partners in the previous four weeks was associated with candidiasis after controlling for frequency of sexual intercourse.

Although similar with respect to demographic variables to all University students, it is possible that women using the Health Service are a select group. They were more likely to be sexually active and use contraceptives than the student population controls. The resulting selection bias might decrease the observed associations between sexual activity, or variables related to sexual activity, and candida when cases are compared to Health Service controls. Further, Health

TABLE 2—Associations between Vulvovaginal Candidiasis and Selected Behaviors during the Past Four Weeks

Variables	Cases (n = 85)		Health Service Controls (n = 1245)			Population Controls (n = 113)		
	#	%	#	%	OR (95% CI)	#	%	OR (95% CI)
Wipe back to front vs reverse	21	25.0	403	33.0	0.86 (0.51, 1.45)	59	52.2	0.34 (0.16, 0.71)
Infrequent bowel movements	13	15.3	148	11.9	1.26 (0.68, 2.31)	11	9.7	1.63 (0.60, 4.44)
Use colored toilet paper								
never	25	29.8	505	41.0	reference	62	54.9	reference
rare	30	35.7	372	30.2	1.07 (0.88, 1.31)	29	25.7	1.41 (0.71, 2.00)
some	17	20.2	215	17.5	1.14 (0.77, 1.70)	15	13.3	1.99 (0.99, 4.00)
frequently	11	13.1	118	9.6	1.22 (0.67, 2.33)	7	6.2	2.81 (0.99, 8.01)
always	1	1.2	22	1.8	1.31 (0.59, 2.91)	—	—	—
Menstrual protection								
napkins	11	13.1	214	17.2	reference	26	23.4	reference
tampons	31	36.9	384	30.8	1.19 (0.58, 2.43)	27	24.3	1.34 (0.48, 3.76)
tampons & napkins	39	46.4	578	46.4	1.20 (0.61, 2.38)	49	44.1	1.43 (0.56, 3.65)
Tub baths (ever)	33	39.3	433	35.3	0.90 (0.56, 1.45)	18	16.1	3.49 (1.54, 7.95)
Feminine hygiene products	15	17.7	76	6.1	1.81 (0.94, 3.49)	1	0.9	12.89 (1.39, 119.79)
Underwear fabric								
always cotton	23	27.1	335	26.9	reference	38	33.6	reference
mix of fabrics	57	67.1	820	65.9	0.94 (0.56, 1.57)	71	62.8	1.05 (0.50, 2.21)
synthetic only	5	5.9	90	7.2	0.99 (0.36, 2.75)	4	3.5	0.96 (0.16, 5.88)
Wearing tight clothing	6	7.1	105	8.4	0.70 (0.29, 1.69)	8	7.1	1.72 (0.48, 6.19)
UTI in past year	20	23.5	204	16.6	1.28 (0.72, 1.38)	7	6.2	2.27 (0.74, 7.03)

NOTE: Odds ratios and their 95 percent confidence intervals (OR, 95% CI), were adjusted for frequency of sexual intercourse, previous infections with vulvovaginal candidiasis, age, and for Health Service controls, month of enrollment, using a logistic regression model (see methods).

Service users may be more likely to have diseases associated with sexual activity and thus be more likely to adopt behaviors thought to limit infection. This may account for the differences in associations when cases are compared to student population rather than Health Service controls. Cases and controls completed the same questionnaires minimizing any misclassification with respect to exposure. Because case status was based solely on clinical diagnosis, undoubtedly some misclassification of disease occurred. However, the bias should have been at random, biasing the results toward no difference between groups.

This study showed a strong association between sexual intercourse and vulvovaginal candidiasis among non-pregnant women. Other suggested factors—wearing tight clothing, hose or synthetic underwear; type of menstrual protection; use of feminine hygiene sprays; and diet—were not associated with candidiasis. Most women with recurring vulvovaginal candidiasis tend to avoid all suggested risk factors,[1] which do not include sexual intercourse. Although these findings need confirmation, suggesting to women suffering recurring vulvovaginal candidiasis that they temporarily decrease frequency of sexual intercourse has more scientific basis than some of the other standard advice (wear cotton underwear), would do no harm, and might potentially avoid recurrence.

ACKNOWLEDGEMENTS

The author thanks the University of Michigan Health Service for their support of this study. Without the efforts of the triage nurses who distributed the questionnaires, the study would not have taken place.

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Recurring Urinary Tract Infection: Incidence and Risk Factors

BETSY FOXMAN, PHD

Abstract: Urinary tract infection (UTI) is a common infection among young women, with a high recurrence rate. This study documents the six-month incidence of second UTI among a cohort of women with one initial UTI and the factors associated with recurrence. Among the cohort of 113 women, 30 (26.6 percent) experienced at least one culture-confirmed recurrence within the six months following initial infection. The presence of hematuria and urgency as symptoms of initial infection were the strongest predictors of second infection. Behavioral factors associated with initial infection (frequency of sexual intercourse, diaphragm use, and voiding after sexual intercourse) did not distinguish between women who would and would not experience a second UTI during the six-month follow-up period. (*Am J Public Health* 1990; 80:331–333.)

Introduction

Urinary tract infection occurs frequently among young women, with over one-fourth having a second infection.[1] Why otherwise healthy women with no known anatomical abnormalities have multiple recurrences is a subject of some controversy, although both host and agent characteristics have been suspected. However, most authors would agree with Kunin's statement that the risk of a second infection is greater than the first, and that the first "sets the stage" for recurring infection.[2]

Methods

All cases of first UTI among college women enrolled in a previously described case-control study* were eligible to participate in the follow-up study. A UTI was defined as the presence of significant bacteriuria (≥10,000 colonies of a single bacteria per milliliter urine) and one or more urinary symptoms: hematuria, dysuria, frequency, urgency, or suprapubic pressure. Urinalyses and urine cultures were performed on all women presenting with urinary symptoms using standard techniques.* History of UTI was assessed by review of medical records and self-report.

Prior to enrollment, participants completed a self-administered questionnaire which contained a consent form approved by the University of Michigan Institutional Review Board and questions on medical history, stress, clothing, diet, sexual activity, and birth control method. Responses to questions regarding birth control method, sexual activity, history of UTI, and demographics and the presence of UTI were confirmed by review of medical records. At the end of the six-months follow-up, participants' medical records were again reviewed to record the number and timing of UTIs treated.

Symptoms (frequency, dysuria, nocturia, urgency, hematuria, fever, chills, back or flank pain, and any other symptoms specified by the respondent), costs of treatment, measures of disease impact, and whether a woman had changed her health habits following the UTI were assessed via a self-administered questionnaire sent to all participants two weeks following enrollment.

Since bacteria normally found in the intestine cause the majority of UTIs in this age group, a woman may be

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*Foxman B, Chi JW: Health behavior and urinary tract infection 1990; J Clinic Epidemiol (in press).

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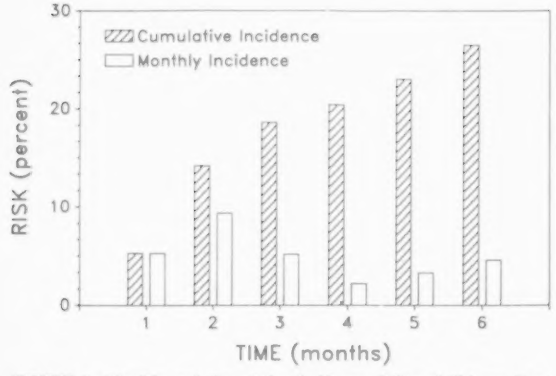

FIGURE 1—Monthly and Cumulative Incidence of Second Urinary Tract Infection (UTI) among a Cohort of 113 Women with One Prior UTI

re-infected with identical bacteria making it difficult to distinguish relapse from recurrence even in the presence of sophisticated bacteriologic techniques. As such techniques were not used in this study, I defined a recurrent urinary tract infection as the presence of ≥ 10,000 col/ml of a single bacteria and one or more urinary symptoms occurring at least two weeks after the initial infection. This may lead to some misclassification of outcome; however, as long as the classification is not associated with the exposure(s) of interest the resulting measures of association should be biased toward the null hypothesis of no difference.

The associations between hypothesized risk factors and repeat UTI were studied using multilevel contingency tables, with both stratification and multivariate techniques to control for confounding.[3] I calculated six-month cumulative incidences of UTI, risk ratios (RR), and Mantel-Haenszel summary RRs. Exact 95 percent confidence intervals (CI) were calculated around each RR using the method of Gart.[4]

A three-level symptoms scale, (0 = no hematuria or urgency, 1 = urgency alone, 2 = hematuria and urgency) was developed and used both as a dependent and independent variable in the analysis. When used as a dependent variable, I tested the hypothesis that the relationship was linear and the slope zero with a chi square test for trend.[5] Linear regression models with number of culture confirmed repeat UTIs and with the symptom scale as dependent variables were also fit to the data.

Results

The study cohort consisted of 113 women ages 17 to 39 (mean 21.3 years) with a first UTI. None of the participants had pyelonephritis, nor concurrent infection with gonorrhea, herpes, or chlamydia. The majority were White (83.8 percent), single (90.1 percent), and did not have a private physician (other than at the Health Service) within driving distance of their current residence (71.2 percent). Most had sexual intercourse during the four weeks prior to their first UTI (94.6 percent).

The six-month risk of a second UTI was 26.6 percent; the risk of a third UTI, 2.7 percent. Figure 1 shows the distribution of second infections; the median time to recurrence was approximately two months (56 days).

The presence of hematuria and/or urgency as symptoms of first infection was the strongest predictor of second infection (Table 1). Although the confidence intervals around the risk ratios include 1.0 (no difference), a significant relationship between symptoms and second infection was observed using a linear regression model including age as an independent variable (linear regression coefficient: 0.149, SE = 0.064). Other symptoms—fever, chills, flank or back pain, frequency, nocturia, or dysuria—were not associated with second infection.

Frequency of sexual intercourse, diaphragm use, and voiding around sexual intercourse were only weakly associated with second infection (Table 1). The trend in decreasing proportion of second infections with voiding after sexual intercourse is of interest (linear regression coefficient: −0.033; SE = 0.033).

Neither frequency of sexual intercourse, diaphragm use, nor voiding after sexual intercourse were associated with the presence of urgency and hematuria at first infection. However, increasing age was negatively associated with increas-

TABLE 1—Factors Associated with Second Urinary Tract Infection among College Women

Factors	Number	Percent Recurring	RR (95% CI)
*Symptoms of initial infection**			
Symptom scale			
no urgency or hematuria	22	13.6	1.0 (reference)
urgency alone	38	21.1	1.5 (0.5, 5.2)
urgency and hematuria	47	38.3	2.8 (0.9, 8.5)
Behaviors during four weeks prior to initial infection			
Frequency of sexual intercourse†			
<1 times per week	20	25.0	1.0 (reference)
1–2 times per week	38	21.0	0.8 (0.3, 2.2)
3 or more times per week	52	30.8	1.2 (0.5, 2.9)
Birth control method (sexually active women only)			
Oral contraceptives	47	25.5	1.0 (reference)
Diaphragm use	22	31.8	1.2 (0.6, 2.7)
Other	36	25.0	1.0 (0.5, 2.1)
Voiding after sexual intercourse (sexually active women only)			
Never/rare	36	33.3	1.0 (reference)
Sometimes	34	26.5	0.8 (0.4, 1.8)
Frequently/always	35	20.0	0.7 (0.3, 1.5)

*Two women had hematuria alone, four had missing data.
†Three women had missing data.

ing symptoms (linear regression coefficient: −0.012; SE = 0.013).

Discussion

In this study, factors observed to be associated with UTI in previous studies[6-9]—frequency of sexual intercourse, diaphragm use, and voiding after sexual intercourse—did not predict recurrence in the next six months. The strongest predictors were characteristics of the infection itself: the presence of hematuria and urgency. Indeed, the six-month risk of second infection was three times higher among women with both, compared to neither, symptom (13.6 percent vs 38.3 percent). This suggests that it is the characteristics of the bacteria causing the initial infection and the extent of infection which predict recurrence.

Since I depended upon medical records for report of recurrence in a fairly mobile population, the risk of second UTI was probably underestimated. Moreover, sophisticated bacteriologic techniques were not used, making it impossible to determine how a particular bacteria and associated virulence factors might set the stage for recurrence. However, the results imply that future studies should examine bacterial and host characteristics as well as health behaviors to better understand the etiology of recurring UTI.

In conclusion, among this cohort of college women with initial UTI, demographic and behavioral factors did not distinguish between women who would and would not experience a second UTI during the six-month follow-up period. The strongest predictor of second infection was the presence of hematuria and urgency as symptoms of initial UTI. Future studies should use consider both behavioral and bacterial characteristics as potential determinants of recurring UTI.

ACKNOWLEDGMENTS

The author thanks the University of Michigan Health Service for support of this study. Without the efforts of the triage nurses who distributed the questionnaires, the study would not have taken place. Thanks also to colleagues James Koopman and Carl Marrs for helpful comments on earlier drafts of this manuscript, and to research assistants Ana Carriazo, Patricia Crawford, Carl Li, D. Rebecca Prevots, Cynthia Rusk, and Ellen Ward for their help in coordinating and managing the study. This work was supported by grant #DK35368 from the National Institute of Diabetes, Digestive and Kidney Diseases.

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Potential for Lyme Disease in Maine: Deer Survey of Distribution of *Ixodes dammini*, the Tick Vector

ROBERT P. SMITH, JR, MD, MPH, PETER W. RAND, MD, AND ELEANOR H. LACOMBE, BS, MT (ASCP)

Abstract: A survey of deer brought to tagging stations at 24 sites in Maine revealed the presence of the deer tick, *Ixodes dammini*, on 5.1 percent of deer. Ticks were found almost exclusively on deer from southwest coastal sites in the state. The potential for endemic Lyme disease in coastal Maine merits further study. (*Am J Public Health* 1990; 80:333–335.)

Introduction

The current geographic range of *Ixodes dammini*, the principal vector of Lyme disease in the eastern United States, is incompletely established. Continued spread of *I. dammini* in coastal New England accounts for new foci of Lyme disease in previously non-endemic areas.[1,2] Recent studies document the rapid rise of Lyme disease prevalence in some areas following colonization by the deer tick.[1] As public education remains the primary means of prevention of Lyme disease, knowledge of the regional distribution of the vector ticks enables targeted surveillance and focused public health interventions. Prior to 1985, there were no records of *Ixodes dammini* from Maine.* Although Maine has been considered a non-endemic area for Lyme disease, infected *I. dammini* were recently reported from one coastal Maine site[3] and *I. dammini* have been found on a deer shot in a northern inland site.[4] Three locally acquired seropositive cases of Lyme disease meeting Centers for Disease Control (CDC) criteria were reported to the Maine Bureau of Health between 1986 and 1988. We undertook a survey of ticks on deer brought to Maine tagging stations in 1988 to further describe the distribution of *I. dammini* at its northeastern limit in the United States.

Methods

Twenty-four deer tagging stations were selected for surveillance throughout Maine in consultation with state

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*Dearborn R: Personal communication. Department of Conservation, State of Maine.

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Inland Fisheries & Wildlife officials. Sites were chosen on the basis of high deer registrations during the previous hunting season, their proximity to representative coastal and inland habitat, and their accessibility to survey teams.

Volunteers including college biology students and their instructors, physicians, and public health professionals, and personnel from Inland Fisheries & Wildlife participated in two weekend surveys October 29 and November 5, 1988. Each team was supplied with a study protocol, an information sheet for hunters, data forms for each deer examined, 6 ml screw-top polyethylene specimen bottles with 70 percent alcohol, fine-point plastic tweezers, and rubber gloves. After obtaining permission from the hunter, each deer was examined for five minutes in a search for adult ticks that included the face, neck and ears of each deer. Ticks were removed with tweezers placed as close to the skin as possible. For each deer, the quantity and location of the ticks found, the times of the kill and of the examination, and the topography and vegetation of the kill site were recorded.

Type specimens of ticks were sent for species confirmation to Dr. J. E. Keirans, Department of Entomology, Smithsonian Institution, Washington, DC.

Results

Seventy volunteers visited 24 tagging stations at least one day of the hunting season. Two hundred and thirty-seven deer (141 male, 68 females, 28 sex unrecorded) taken in 66 towns, townships and plantations, including one island community, were examined. Twelve deer (5.1 percent) harbored *Ixodes dammini* ticks. A total of 33 ticks (all adults) were collected with a range of 1 to 11 ticks per infested deer. Ticks obtained from the hides of three freshly killed deer at a southwest coastal site are also included in the total. The average time of the examination of deer after the kill was 3.5 hrs (range, 0.5–46.5 hrs). Not included are time data from one island community where deer were examined one to three days after being tagged. Ten deer were found to harbor the "winter tick," *Dermacentor albipictus*. No other ectoparasites were obtained.

With the exception of one tick, all *I. dammini* were obtained from deer in the southwest coastal areas of Maine (see Figure 1). No ticks were found on deer from the one island studied. No association could be made between type of habitat at kill sites and deer infestation by *I. dammini* ticks.

Discussion

This study documents the presence of *Ixodes dammini* on deer from the southwest coast of Maine and a relative paucity of ticks from deer in inland areas. The habitat of southwest coastal Maine is similar to that in some other coastal New England areas with a high prevalence of Lyme disease, and it is characterized by deer herd densities as high as 20–50 per square mile. Although infection rates of ticks collected in this study were not determined, 12 percent of a small sample of adult and nymphal deer ticks from Acadia National Park harbored *Borellia burgdorferi*.[3] An infection rate of at least 10 percent has characterized other New England sites of *Ixodes dammini* colonization.[4,6] Although tick density on the deer examined was low relative to highly endemic sites,[7] the degree of risk for transmission of this zoonosis in southwest coastal Maine deserves further study. All three cases of endemic seropositive Lyme disease reported in Maine through 1988 were believed to have been acquired in this region. As there are no records of *I. dammini*

FIGURE 1—Number of Deer upon Which *I. dammini* Were Found versus Total Deer Examined in Four Geographical Areas in Maine

or Lyme disease in Maine prior to 1985, we believe that *I. dammini* has appeared here recently.

The sensitivity of deer surveys for the determination of presence of *Ixodes dammini* is unknown. The method of examination used, rigorousness of search, and training of personnel could alter its effectiveness. Reporting of other ticks on the deer (i.e., *D. albipictus*) is considered an indicator of the sensitivity of the search, but the expected prevalence of *D. albipictus* on northern New England deer is not known. A recent study of 370 deer in Vermont by trained wildlife personnel documented only two *Ixodes dammini* and no other ticks, although other ectoparasites were noted.** Comparative information of the effectiveness of deer surveys, dog seropositivity studies, and other methods of surveillance is needed. Our study is limited by marked differences in the number of deer available for examination at many tagging stations. The low number of deer examined in most inland sites and our inability to canvass all areas of the state systematically makes conclusions regarding the full range of *Ixodes dammini* in Maine premature. Nevertheless,

**Mamolen M: Personal communication. Department of Health, State of Vermont.

the results of this deer survey coupled with the known ecology of southwest coastal Maine point to an emerging zoonosis in this area with potential public health importance.

ACKNOWLEDGMENTS

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HRSA Health Care Program for the Homeless Reaches Its Legislative Goals

In a report to Congress on January 3, 1990, the Health Resources and Services Administration (HRSA) reported that its health care for the homeless program had been "successful in reaching its legislative goals." The report, which covered activities in calendar year 1988, was the first annual report on the program and noted the following:

- Within four months after signing of the authorizing legislation, the Steward D. McKinney Homeless Assistance Act, HRSA awarded $44.5 million in grants to 109 health care projects for the homeless in 43 states, the District of Columbia, and Puerto Rico.
- The 109 projects provided health care services to more than 231,000 homeless persons.
- Forty percent of the homeless persons served were children and their parents (living as a family unit) or homeless/runaway youths.
- Fifteen percent of those served were children ages 14 or younger.
- Thirteen percent of pregnant homeless females treated were teenagers.
- Sixty percent of persons served were minorities: 42 percent Black, 10 percent Hispanic, and 8 percent Native American, Asian, or other non-White.

The report concluded by saying, "The health care for the homeless program has demonstrated that the federal government, in concert with local communities, can bring health care services to homeless persons within a short period of time." For further information, contact Frank Sis, HRSA, US Public Health Service, DHHS, 5600 Fishers Lane, Room 14-43, Rockville, MD 20857. Tel: 301/443-3377.

* * * * * * * *

Nursing Conference on Homelessness Scheduled May 3–4, 1990

A two-day forum for nurses and other health professionals involved in health care for the homeless projects will be held under sponsorship of a HRSA-supported project at State University of New York, Buffalo, May 3–4, 1990. Topics of the conference will include issues related to homeless children and mental health. For information about the conference, entitled "Homelessness: An Issue for the Nineties," contact: Continuing Nurse Education, School of Nursing, 14 Kimball Tower, SUNY at Buffalo, 3435 Main Street, Buffalo, NY 14214. Tel: 714/831-2391.

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Human Immunodeficiency Virus Seropositivity among Members of the Active Duty US Army 1985–89

PATRICK W. KELLEY, MD, MPH, RICHARD N. MILLER, MD, MPH, ROBIN POMERANTZ, MS, FRANK WANN, MS, JOHN F. BRUNDAGE, MD, MPH, AND DONALD S. BURKE, MD

Abstract: Between October 1985 and June 1989, most active duty US Army soldiers were screened for human immunodeficiency virus (HIV) antibody. Of 648,032 screened soldiers in this analysis, 1,588 were HIV-antibody positive. In a multivariate analysis, correlates of positivity included: *age* [Adjusted Odds Ratios (ref <20 years) = 20–24 years, 3.7; 25–29, 9.3; 30–34, 15.7; ≥35, 15.9]; *being male*, [4.2]; *being Black or Hispanic (vs white)* [3.7 and 3.0, respectively]; *being single (vs married)* [3.8]; *assignment to an HIV endemic location* [1.7], and *having a medical occupation* [2.7, 2.7, and 2.6 for negligible, low, and high blood exposure professions, respectively]. Seropositivity rate ratios for medical vs non-medical personnel were 0.7 [95% CI = 0.4, 1.4] for females and 2.9 [95% CI = 2.5, 3.3] for males. For male medical personnel, being single (vs married) correlated strongly with antibody positivity [prevalence ratio = 3.4, 95% CI = 2.6, 4.6]. Excess HIV risk among medical personnel appeared largely attributable to factors other than occupational exposures. (*Am J Public Health* 1990; 80:405–410.)

Introduction

Effective programs to prevent the spread of human immunodeficiency virus (HIV) infection in the general population require the identification of target populations for intense health education and voluntary test-linked counseling.[1–4] Although much is known about the epidemiology of the acquired immunodeficiency syndrome (AIDS), the end stage of HIV infection, the value of this information for educational purposes is limited by the fact that AIDS cases generally reflect transmission risk factors that were operating years ago.[5,6] Prevention programs must reflect HIV infection-transmission patterns operating today and those likely to emerge in the near future. Prevalence and incidence surveys of generally healthy populations can help identify populations for targeted HIV prevention education.

Except for the national blood-donor screening programs, military HIV screening programs currently provide the only HIV seroprevalence data on large cross sections of the US population. Several papers have previously summarized the demographic risk factors for military recruit applicants, a group largely between the ages of 17 and 24.[7–9] In that population, the cumulative seroprevalence is 1.4/1,000 although over time the monthly rates have progressively dropped to below 1.0/1,000, a phenomenon that probably reflects self-deferral of potential applicants who either know from other sources or suspect that they are seropositive. Among recruit applicants, seropositivity is several-fold higher in Blacks, Hispanics, and males. Similar findings have been noted for members of the US Army Reserve Components.[10]

This paper, the largest detailed summary of military HIV demography published to date, expands on the recruit applicant data by focusing on active duty soldiers, a somewhat older group of persons who are more likely to be married and to have had substantial employment experience.

Address reprint requests to Patrick W. Kelley, MD, MPH, Division of Preventive Medicine, Walter Reed Army Institute of Research, Washington, DC 20307-5100. Dr. Miller and Dr. Brundage are also with the Division of Preventive Medicine at Walter Reed; co-authors Pomerantz and Wann are with SRA Technologies, Inc., Alexandria, Virginia; Dr. Burke is with the Division of Retrovirology at Walter Reed. This paper, submitted to the Journal June 5, 1989, was revised and accepted for publication November 8, 1989.

Editor's Note: See also related editorial p 401 this issue.

Methods

Population

On October 24, 1985 the Secretary of Defense directed that all 2.1 million active-duty US service members undergo mandatory HIV education and antibody screening.[11,12] This objective was addressed through a variety of programs that included mass screening of military units, screening prior to overseas deployment, blood donor testing, sexually transmitted disease (STD) clinic patient screening, case-contact tracing, and testing for clinical reasons. In 1988, the unit screening program was largely replaced by a birth month screening program designed to ensure that each soldier is tested at least once every 24 months.

HIV Testing

Sera collected as part of the mass screening program were tested with a licensed whole-virus lysate enzyme-linked immunosorbent assay (ELISA). This work was largely done at either a central civilian contract laboratory (Damon Laboratories, Irving, TX) for soldiers assigned to the continental US, Alaska, or Panama; at the 10th Medical Laboratory, Landstuhl, Germany for soldiers stationed in Europe; or at Tripler Army Medical Center for soldiers in Hawaii and the Far East. Positive samples were immediately retested in duplicate with the ELISA.[13] Repeatedly positive samples from all screening laboratories were tested for reactivity with HIV proteins by the Western (immune) blot method by the civilian contract laboratory.[14] From October 1985 until May 1987, specimens were classified as positive if antibodies to HIV-encoded protein GP41 and/or protein P24 plus P55 were detected.[15] In May 1987, the Association of State and Territorial Public Health Laboratory Directors criteria for a positive blot were adopted: at least two out of three of the major HIV protein bands P24, GP41, and GP120/160. Soldiers found positive on the initial Western blot had a second specimen drawn for verification purposes. A detailed Army-administered quality-assurance program characterized by monthly on-site laboratory inspections and specific performance criteria (including the successful analysis of both in-house controls and blind and unblinded panels) has been described elsewhere.[15]

Statistical Analysis

Because the Army population is undergoing constant turnover with only HIV-seronegative applicants allowed to enter and because during the period of data collection

HIV-antibody positive soldiers have separated at a significantly faster rate than seronegative soldiers, this analysis, for reasons of clarity and reference, is confined to those soldiers on active duty when the program was just beginning, specifically on June 30, 1986. Laboratory results, social security number, first four letters of the last name, and test date were keyed onto computerized data files and merged with an abstracted copy of the June 30, 1986 active duty Army personnel file as supplied by the Defense Manpower Data Center, Monterey, CA. For soldiers with more than one test result recorded, only their most recent test was retained in the analysis data set. Antibody positivity was considered verified if an individual was reported by a separate clinical reporting system as HIV-infected (96.2 percent of those potentially considered positive were clinically verified as such) although in 22.9 percent of the cases clinical verification was based only on the documentation of an individual having had multiple positive HIV Western blots on separate specimens. Only soldiers verified to be HIV-infected prior to August 3, 1989 are considered HIV-infected for this analysis.

Seropositivity rates were calculated as both centrally screened and overall rates. The centrally screened rate is defined as the number of antibody positive soldiers who were reported as having been screened through the three centralized laboratories listed above divided by the total number of soldiers reported as screened by those laboratories.

Many soldiers found to be antibody positive were initially tested through testing activities conducted outside of the centralized testing laboratories (e.g., blood bank and some STD clinic screening). These individuals often were exempted from subsequent mandatory testing done by the centralized laboratories. The overall rate is based on a numerator that includes all antibody-positive active-duty soldiers who were in the Army on June 30, 1986 divided by the number centrally screened plus the number of positive soldiers not reported from the central laboratories. The centrally screened rate is a significant underestimate of the true rate because only 79 percent of the eligible antibody positive soldiers ever had a centralized screening test result reported. The overall rate is felt to be a modest overestimate because the denominator excludes a small though unquantifiable number of seronegative soldiers who either were tested but did not have a negative test reported or who were not centrally screened but were antibody negative in adjunct screening programs, such as blood bank and clinical screening. Some negative ELISA results from blood bank and clinical screening were not captured for epidemiologic purposes since not all of the adjunct screening was done by one of the three centralized HIV testing laboratories.

Because of centralized Western blot testing, positive blots from all testing programs were captured in an extremely complete fashion. For the most part, only overall rates and measures of effect based on overall rates will be described in the text since the overall rates are felt to best reflect the true rates. In the analyses of prevalence by occupation, broad occupational clusters were defined according to Army regulation, with minor modifications.[16] For the analysis of prevalence among medical personnel, the blood exposure groupings with minor modifications were those of Gardner, *et al.*[17]

To demonstrate the effect of a possible risk factor, seropositivity rate ratios with 95% confidence intervals were calculated.[18] In bar graphs, 95% CIs are based on the Poisson distribution. The main effects logistic regression analysis was performed with the Stepwise Logistic Regression (PLR) procedure of the BMDP Statistical Software Package (University of California at Los Angeles). All listed variables were forced into the model. Adjusted odds ratios were calculated by using logistic regression parameter estimates.[19] Confidence intervals for odds ratios and estimates of their statistical significance were derived from standard errors of the parameter estimates.[19]

Results

As of July 27, 1989, centrally screened results have been received for 647,700 soldiers out of the 763,675 individuals in the June 30, 1986 active-duty Army personnel file abstract. Demographically, this 84.8 percent sample (Table 1) constituted a representative sample of the whole Army. Three hundred and thirty-two soldiers who were verified as antibody positive but not reported as having been centrally screened were added to the analysis file. Of the 115,643 June 30, 1986 cohort soldiers without a documented HIV test result, 72 percent had separated from the active Army by December 30, 1988; 4 percent were accessions who had screened negative as part of the recruit-applicant screening instituted on October 1, 1985. The 28,863 remaining active-duty soldiers for whom valid test results were unavailable, were demographically similar to those tested based on race but women, married persons, officers and those at the extremes of the age spectrum appeared to be more likely not to have had a test reported.

Overall Seropositivity

The centrally screened seropositivity was 1.9/1,000 based on the 1,256 verified positive soldiers who had been centrally screened. The overall rate based on the 1,588 June 1986 cohort positives was 2.5/1,000. Of the 1,588 cohort soldiers who were verified as HIV antibody positive as of

TABLE 1—Characteristics of the Sample HIV Screened

Characteristic*	Army Population	Proportion of Army	Sample Population	Proportion of Sample
Age (years)		%		%
<20	78,543	10.3	66,430	10.3
20–24	282,792	37.0	234,073	36.1
25–29	169,579	22.2	147,229	22.7
30–34	108,248	14.2	96,530	14.9
≥35	123,665	16.2	102,846	15.9
Race/Ethnicity				
White	502,696	65.8	421,414	65.1
Black	205,192	26.9	177,991	27.5
Hispanic	27,047	3.5	23,635	3.7
Other	28,740	3.8	24,660	3.8
Sex				
Male	684,530	89.6	583,960	90.2
Female	79,093	10.4	63,691	9.8
Marital Status				
Single	313,655	41.1	260,183	40.2
Married	422,044	55.3	364,193	56.2
No Longer Married	27,001	3.5	22,593	3.5
Unknown	975	0.1	731	0.1
Rank				
Enlisted	652,490	85.4	551,974	85.2
Warrant	15,441	2.0	13,535	2.1
Commissioned	95,744	12.5	82,189	12.7
Length of Service (years)				
<1	115,513	15.1	98,209	15.2
1–3	267,989	35.1	219,130	33.8
4–6	110,967	14.5	96,922	15.0
7–9	83,915	11.0	74,232	11.5
≥10	185,311	24.3	159,161	24.6

*As recorded in June 30, 1986 military personnel file.

August 3, 1989 the reported sources of identification were: 809 (50.9 percent) through unit or birth month screening, 136 (8.6 percent) through clinical evaluations, 112 (7.1 percent) through pre-deployment screening, 101 (6.4 percent) through blood donor screening, 59 (3.7 percent) through STD clinic screening, 48 (3.0 percent) through self-referral, 17 (1.1 percent) through contact tracing, and 306 (19.3 percent) through other or unspecified sources.

Age

Antibody positivity varies significantly with age. For soldiers less than 20 years old at their most recent available test (a group heavily preselected to be negative by recruit-applicant testing), the overall rate was 0.5/1,000. Thereafter, there was a steady progression from an overall rate of 1.8/1,000 in those aged 20–24, to 3.1/1,000 in those 25–29 and to 3.4/1,000 in those 30–34. The rate among soldiers older than 34 drops to 2.2/1,000. A similar pattern was observed in the centrally screened rates.

Race or Ethnic Group

Soldiers who were Black or Hispanic had an overall seropositivity of 5.1/1,000 (900 cases) and 4.0/1,000 (95 cases), respectively. The HIV infection prevalence rate ratios compared to White soldiers were 4.0 [95% CI = 3.6, 4.4] for Blacks and 3.2 [95% CI = 2.5, 3.9] for Hispanics. Across age strata, Whites had a relatively flat prevalence trend; Blacks and Hispanics both showed a rise through the early thirties and then a fall (Figure 1). HIV infection would appear to be unevenly distributed among subsegments of the Hispanic population. The seropositivity rate for Hispanic soldiers with a home of record listed as Puerto Rico, New York, or New Jersey, a group assumed to be largely of Puerto Rican descent, was 5.6/1,000. In contrast, the Hispanic soldiers with a home of record in states along the Mexican border (California, Arizona, New Mexico, Texas) had a rate of 2.2/1,000 (PR = 2.5, 95% CI = 1.5, 4.3).

Sex

There were 1,516 verified positive cohort men, yielding an overall rate of 2.6/1,000. The corresponding female rate, based on 72 positive women, was 1.1/1,000. The crude male:female seropositivity rate ratio was 2.3 [95% CI = 1.8, 2.9]. Male:female overall rate ratios by race/ethnic group were 2.5 for Whites [95% CI = 1.5, 4.0], 3.0 for Blacks [95% CI = 2.3, 4.0], 2.1 for Hispanics [95% CI = 0.7, 6.7], and 3.1 for others (2 female cases). The overall rates in Black and Hispanic females were 1.9/1,000 (49 cases) and 1.9/1,000 (3 cases), respectively. The Black female rate exceeded the 1.3/1,000 overall rate in White males [RR = 1.4, 95% CI = 1.0, 1.9]. Age-specific male:female ratios did not show any definite trend across age strata (Table 2).

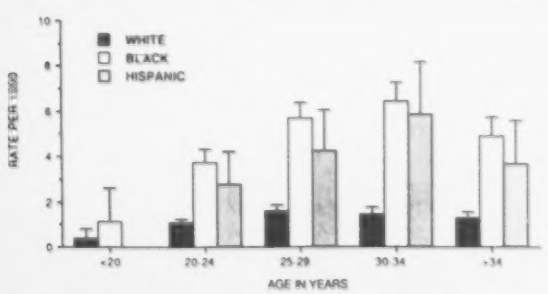

FIGURE 1—Overall HIV Seropositivity Rates and Upper 95% Confidence Interval Bounds for Active Duty US Army Personnel Stratified by Age and Race/Ethnic Group

Marital Status

Among the 1,588 cases, 639 were married, 878 were single, and 71 were no longer married, indicating that 44.7 percent of the cohort cases are or had been married based on the June 30, 1986 file. Compared to married soldiers, the HIV infection prevalence ratios for single soldiers and for those no longer married were 1.9 [95% CI = 1.7, 2.1] and 1. 8 [95% CI = 1.4, 2.3], respectively. Whereas overall rates were quite similar for married and single soldiers in the youngest age strata, single soldiers over 34 years of age had an overall prevalence rate that was 10.8 times that found in married soldiers in that stratum (Figure 2). Among White and Black soldiers, the rate ratios for single soldiers relative to married soldiers were 2.1 [95% CI = 1.8, 2.5] and 2.1 [95% CI = 1.8, 2.4], respectively. In contrast, the rate ratio for single Hispanic soldiers compared with their married counterparts was 1.1 [95% CI = 0.7, 1.7]. For males the overall single and married rates were 3.7/1,000 (844 cases) and 1.8/1,000 (607 cases), respectively. Among females the single (34 cases) and the married (32 cases) rates were both 1.1/1,000.

To explore the effects of age on estimates based on female marital status, female rates were directly age adjusted using the total male age distribution. The adjusted female rates were 1.2/1,000 for singles and 0.9/1,000 for marrieds.

Rank and Education

Based on 1,447 cases, the overall seropositivity rate for enlisted soldiers was 2.6/1,000. Warrant officers had an overall rate of 0.9/1,000 (12 cases) and commissioned officers an overall rate of 1.6/1,000 (129 cases). Within White, Black, and Hispanic groups, officer to enlisted prevalence ratios did not vary significantly (data available on request to author). Non-college graduates (less than 4 percent of soldiers in this cohort had not completed high school) had an overall rate of 2.6/1,000 (1,400 cases) in contrast to a rate of 1.9/1,000 (183 cases) for college graduates.

Occupational Category

HIV infection is distributed quite unevenly across job clusters (Table 3). Because health care workers had an elevated rate and because the potential for acquisition of infection in the course of performing job-related functions has been documented, several analyses were undertaken to better understand the source of risk for these individuals. Rate ratios comparing prevalence estimates for 42,697 health care providers [72.4% male] and 605,286 non-medical personnel [91.4% male] were 0.7 [95% CI = 0.4,1.4] for females and 2.9 [95% CI = 2.5, 3.3] for males. For male medical personnel being single [vs married] was a major correlate of

TABLE 2—US Army HIV Testing Overall Seropositivity Rate Estimates by Age and Sex

Age (years)	Male Rate* (No. Positive)	Female Rate* (No. Positive)	Male:Female Rate Ratio	95% CI
<20	0.4 (8)	0.9 (2)	0.5	0.1–2.2
20–24	1.9 (380)	0.7 (15)	2.8	1.7–4.7
25–29	3.4 (484)	1.2 (25)	2.8	1.9–4.1
30–34	3.5 (352)	2.0 (23)	1.8	1.2–2.7
≥35	2.3 (292)	0.9 (7)	2.6	1.2–5.5

*Rates are per 1000 persons

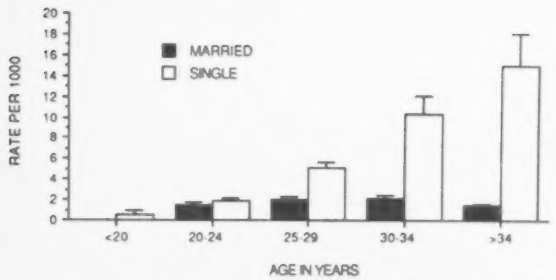

FIGURE 2—Overall HIV Seropositivity Rate and Upper 95% Confidence Interval Bounds for Active Duty US Army Personnel Stratified by Age and Marital Status

TABLE 3—US Army HIV Seropositivity Rates by Occupational Cluster

Job Cluster	Centrally Screened		Overall	
	Rate*	N Positives	Rate*	N Positives
Military Science	1.4	267	1.6	317
Electronics and Communications	1.9	140	2.6	189
Administration and Arts	3.8	212	4.9	270
Health Science	4.0	215	5.0	269
General Engineering and Science	1.7	36	2.1	44
Aviation	0.5	15	0.8	22
Intelligence/Military Police	1.0	42	1.3	58
Mechanical Maintenance	0.9	53	1.1	68
Missile Maintenance	1.3	11	1.4	12
Transportation, Supply, and Services	2.7	254	3.5	325
Other and Unknown	0.7	11	0.9	14

*Rate are per 1000 persons

positivity [prevalence rates = 3.4, 95% CI = 2.6, 4.6]. Among single medical males over the age of 34, the seropositivity ratio relative to their married counterparts was 15.9 [95% CI = 9.3, 27.3]. Rate ratios for male medical staff with high- and intermediate-level blood exposure relative to male care providers with minimal blood exposure were 0.9 [95% CI = 0.5, 1.5] and 1.1 [95% CI = 0.7, 1.6], respectively.

Multivariate Analysis (Adjusted Odds Ratios)

To control for the simultaneous effect of many mutually confounding factors, a main effects logistic regression model was constructed using the variables in Table 4. To reduce the preselection effect of recruit-applicant screening which started in October 1985, only soldiers with at least 10 months of service as of June 30, 1986 were included in the model. Noteworthy changes in odds ratios brought about by multivariate adjustment included the less obvious decline in risk for the oldest soldiers, the increased risk for men relative to women (adjusted OR = 4.2) and for singles relative to married soldiers (adjusted OR = 3.8). Adjustment significantly brought down the odds ratios associated with increasing length of service. The odds ratios by race/ethnic group, endemicity group, and medical blood exposure category were not significantly changed by the adjustment.

TABLE 4—Main Effects Multiple Logistic Regression Model for HIV Infection in the US Army

Variables	Crude Odds Ratio	Adjusted Odds Ratio	95% Confidence Interval
Age (vs <20)			
20–24	3.5	3.7	1.7–8.4
25–29	5.4	9.3	4.1–21.1
30–34	5.6	15.7	6.9–35.8
≥35	3.7	15.9	6.9–36.6
Sex (vs Female)	2.4	4.2	3.3–5.4
Race/Ethnic Group (vs White)			
Black	3.9	3.7	3.3–4.2
Hispanic	3.1	3.0	2.4–3.7
Others	1.8	1.6	1.2–2.2
Marital Status (vs Married)			
Single	2.3	3.8	3.4–4.4
Was Married	1.8	1.9	1.5–2.4
Endemicity*	1.7	1.7	1.4–1.9
Years of Service (vs <3 yrs.)			
4–6	1.1	0.8	0.7–1.0
7–9	1.4	0.8	0.7–1.0
≥10	0.9	0.6	0.5–0.7
Rank (vs Officer)	2.0	1.7	1.4–2.0
Blood Exposure Level (vs non-health care job)			
Negligible	2.2	2.7	1.8–4.0
Low	2.5	2.7	2.2–3.2
High	1.9	2.6	1.9–3.6

*Based on assignment in California, southern New York State, New Jersey, or the vicinity of the District of Columbia as of June 30, 1986.

Discussion

This analysis summarizes one of the largest population-based HIV antibody surveys undertaken to date. Although the observations are not directly generalizable to the 15 to 50 year old segment of the US general population due to various biases including the presumed underrepresentation of intravenous drug abusers and homosexuals in the military, the above results support the findings of other studies that HIV infection is an important issue for Black and Hispanic communities and for females.[7,20–23] The data appear to reasonably represent the people in the US Army on June 30, 1986. The entry and separation of over 100,000 people from the active component of the US Army each year plus the worldwide mobility of that population did not allow an HIV antibody screen to be completed on all members of the June 30, 1986 cohort.

The prevalence trend by age was similar to that noted for military recruit applicants and notable for a peaking in the late 20s and early 30s before tailing off in the mid-30s.[7] This trend in antibody positivity peaks several years before the peak described for AIDS cases. There are several possible explanations for the HIV antibody age pattern. First, those inclined to engage in risk behaviors may tend to moderate the intensity of their risk behaviors with age. Since the HIV virus has only been endemic in the US for an estimated 10–15 years, older soldiers may have passed through a period of more frequent risk taking before HIV was a significant presence. Second, older soldiers are more likely to be career soldiers than are those in the younger age strata. Soldiers who aspire to a military career and who have successfully adapted to the military environment (as suggested by many years of service) may be less inclined to practice some risk behaviors such as intravenous drug abuse and homsexuality because the practice of these behaviors is contrary to military policy. Since the early 1970s, random urine drug screens have been a regular occurrence in the Army, making even the covert use

of illicit drugs a risk to a soldier's career. After adjusting for marital status, length of military service, and other variables in the multiple logistic regression model, there is a steady increase in risk with age. Most importantly, however, these data suggest that significant risk of HIV infection begins in the late teens/early twenties and continues through the young adult period.

The observation that Black and Hispanic soldiers, even in the multivariate analysis, are at a three-to-four fold increased risk of being infected is consistent with civilian data.[7,20] The significant difference in prevalence trends by age for Blacks and Hispanics in contrast to Whites is again striking. A relatively flat prevalence trend for Whites and a dramatic rise and fall for Blacks and Hispanics may reflect a more rapid recognition of the HIV threat and its consequences on the part of Whites. The 2.5 fold increased risk of positivity for Hispanic soldiers with a home of record documented as Puerto Rico, New York, or New Jersey versus that for Hispanic soldiers with Mexican border state homes is reflective of Selik's finding that AIDS incidence appears to be higher in Hispanics of Puerto Rican descent than among other Hispanics.[24] Unfortunately, data on the risk behaviors directly responsible for the infections are not available to the authors. The observation that 50.4 percent of the 995 largely asymptomatic infected Black and Hispanic soldiers and 44.9 percent of the 536 largely asymptomatic White soldiers had at least seven years of service supports the impression that in the majority of cases the infection was acquired while the soldiers benefited from the economic, occupational, and social stability of a military environment. The fact that risk for both White and Black officers was similar to that of their enlisted counterparts further argues against a major role for current socioeconomic status in this military population.

As with all epidemics, the propagation of this epidemic depends on complex interactions among the agent, environment, and host. In the same way that high HIV prevalences (the agent-endemicity factor) among homosexual men tend to make sex acts (the behavioral-environment factor) more risky than similar acts among heterosexuals in a low HIV prevalence population, so too may risk activities in high prevalence minority populations lead to more infections than they might in lower prevalence communities. Although the data are inconclusive, race/ethnic differences in susceptibility (the host factor) to infection may be contributory factors also.[25–27]

In contrast to the 8:1 male:female AIDS case ratio in the US during the first half of 1989, the HIV infection prevalence ratio in these data is 2.4:1 (adjusted OR = 4.2:1 in multivariate analysis).[28] The prevalence ratio is similar to that reported for military recruit applicants.[7] In teenaged military recruit applicants (J. Brundage, unpublished data) and among people in the African HIV epidemic, the male-to-female ratio approximates 1:1.[21] Male:female prevalence ratios do not differ by race, but it is important to note that in the Army, the rate for Black and Hispanic women is 1.5 times higher than that found in White men, supporting the conclusion that women, particularly of racial/ethnic minorities, need special emphasis in prevention efforts. The marital status information further supports efforts to provide prevention education beyond single homosexual men. Although the marital status data do not allow any statement about the responsible risk activity, it shows that 45 percent of infected Army personnel are or were married. It would be reasonable to assume that most of these people participated in heterosexual sex at least some of the time and that many otherwise risk-avoiding heterosexuals were contacting the virus. One report of risk factors in a population of 57 US Army HIV-positive soldiers noted that 77 percent were heterosexually active, including 31 soldiers who said they were bisexual.[29] Centers for Disease Control AIDS data indicate that about 26 percent of reported AIDS cases are heterosexuals, although it is clear that most of these heterosexuals did not acquire their infections through sexual activity.[20]

Regarding risk by occupation, data presented here are consistent with other published estimates of the low risk for occupational acquisition of HIV infection in the medical setting.[30,31] The observations that excess risk for those in medical occupations appeared to be confined to males and that prevalence was not correlated with levels of occupational blood exposure are consistent with the conclusion that risk in Army health care personnel is largely due to factors other than occupational exposures. The logistic regression analysis also supports this and a comparable analysis of Army Reserve component data comes to a similar conclusion.[32]

The repeated testing of all active duty soldiers is allowing not only the ascertainment of antibody prevalence but also the current annual incidence of seroconversion. The first of these analyses for the period October 1985 to October 1987 identified 90 seroconvertors for a rate of 0.77 per 1,000 persons per year.[33] Current seroconvertors are carefully studied to ascertain potential shifts in transmission patterns since the design of prevention programs should reflect not only the transmission patterns in effect 10 or more years ago, but also recognize the role of emerging risk groups as the infection continues to spread, although probably more slowly, through initially unemphasized segments of the population.

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NCHS Survey: Characteristics of Persons Dying from Stroke

Cerebrovascular disease—or stroke—is the third leading cause of death in the United States, accounting for almost 150,000 deaths in 1986. The National Center for Health Statistics conducted a special survey based on a sample of such deaths among those 25 years of age and over which occurred in 1986. The survey analyzed the health and other care received in the last year of life and source of payment, health practices and behaviors, health status, and social and economic characteristics of those who died from stroke. These data will be used in disease prevention programs and to plan adequate health and support services for those with cerebrovascular disease. The major findings include:

- Nearly 70 percent of those who died from stroke were over the age of 75. Less than half of the deaths from all other causes occurred to those 75 years of age and over.
- More women than men died from stroke, 60 to 40 percent, respectively. Women were about three times more likely to be widowed at the time of death and only one-third as likely to be currently married.
- Most adults who died in 1986 had seen a physician five times or more in the year prior to death. Persons dying from stroke were more likely to have no physician visits or fewer than five visits; and to have resided in an institution during the year prior to death.
- Medicare was the primary source of payment for health care in the last year of life for most causes of death, including stroke.
- A larger proportion of those dying from stroke than from all other causes were reported to have hypertension.

For more information about this report, contact Sandra Smith, NCHS Public Affairs Officer, (301) 436-7135.

The Quality of Ambulatory Care in Medicare Health Maintenance Organizations

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Abstract: The quality of ambulatory care received by Medicare recipients who enrolled in health maintenance organizations (HMOs) was compared to the care received by fee-for-service (FFS) Medicare recipients, in a quasi-experimental, non-randomized design. Both samples were drawn from the four major geographic areas in the country, and included two types of HMO practices: staff/group models, and independent practice associations (IPAs). A panel of expert physicians developed criteria for evaluating ambulatory care, and medical record abstractions using these criteria were performed on 1,590 outpatient records: 777 FFS and 813 HMO (441 staff/group, 372 IPA). While individual items of medical histories and physical examinations were performed most often for staff/group HMO patients and least often in FFS patients, odds ratios (OR) for performance in staff/group HMO patients were particularly large for health maintenance items: tonometry (OR = 8.4), mammography (OR = 2.7), pelvic examination (OR = 5.3), rectal examination (OR = 2.9), fecal occult blood test (OR = 3.3). The results suggest that recommended elements of routine and preventive care are more likely to be performed for Medicare enrollees in staff/group HMOs than in FFS settings. (*Am J Public Health* 1990; 80:411–415.)

Introduction

Due to the rising costs of delivering health care to Medicare recipients, federal cost containment measures have been initiated. Among these initiatives, the use of prepaid care strategies has received increased attention. In 1982, the Health Care Financing Administration began a demonstration program of prepaid health care for Medicare recipients. The 27 participating health maintenance organizations (HMOs) provided comprehensive care, received prospective monthly payments for the enrollees, and assumed the financial risk of their complete health care. The purpose of the program was to show whether contracts with HMOs for Medicare recipients' health care could both reduce costs effectively and also maintain current standards of care. Since the demonstration program began, optional Medicare enrollment in HMOs has been extended, and there are now about one million Medicare enrollees in more than 100 separate HMOs.

Since HMOs reduce the cost of care mainly by replacing hospitalizations with less expensive ambulatory care, utilization rates of their enrollees have been observed to fall to as low as half the rates of Medicare recipients in traditional care arrangements.[1,2] Although the number of ambulatory visits in prepaid systems of care does rise to compensate for the reduction in hospitalizations, it is possible that such increases may strain the resources on which elderly HMO enrollees depend to manage their disabilities. Multiple chronic illnesses are the rule and many have significant functional limitations; outpatient management of elderly persons thus requires considerable time and effort. Hence, misgivings exist as to whether financial constraints, under prepaid systems like HMOs, may lead physicians to compromises that reduce the quality of outpatient care elderly persons need.[3,4]

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To study how prepaid reimbursement affects the care Medicare patients receive in HMOs, the federal government funded the National Medicare Competition Evaluation. This comprehensive study examined not only the effectiveness of prepaid care in cutting Medicare costs, but also its effects on patient satisfaction with the care,[5] and whether HMO Medicare patient care matched the quality of that received by fee-for-service (FFS) Medicare patients. Several components of the evaluation are now either in press or in preparation. The evaluation of the process of routine and health maintenance care for ambulatory patients is presented here.

Methods

HMO Selection

There were several considerations in selecting the study's sample of HMOs. First, adequate representation of the four principal HMO models was a concern: staff HMOs, group HMOs, individual independent practice associations (IPAs), and network IPAs. Staff HMOs employ physicians and other providers directly; group HMOs contract one or more group practices. Individual IPAs contract with many individual physicians who care for HMO enrollees in their own offices; network IPAs contract not only with individuals but also with groups of physicians, with whom they usually share some of the risk for covered services. Because evidence suggests that IPAs function similarly to FFS practices, adequate representation of all four HMO models in the sample was important. Second, those plans with fewer than 2,000 Medicare enrollees were eliminated because they were unlikely to have enough cases for statistical power. Third, medical care often follows the conventional practices of the area, so adequate representation by geographic location was important. Finally, to secure the participation of HMOs and ensure confidence in the integrity of the study objectives, individual HMO plans were not identified in the analysis. From the pool of 27 HMOs in the demonstration, eight, distributed among the four major geographic areas, were selected for study. Four were staff or group model HMOs (staff/group) and four were IPA or network model HMOs (IPA).

Patient and Physician Selection

An important factor for selecting the sample of patients was that HMOs would have a high proportion of new evaluations. Routine care for new patients requires more

detailed medical record-keeping than is required for care of established patients. Therefore, the samples of both HMO and FFS Medicare patients were limited to those who had new patient evaluations during the study time frame, the period from each plan's enrollment date (range: January 1983-May 1984) to March 31, 1986. From the HMOs selected for study, an appropriate sample of patients was identified by age, Medicare status, and the name of the primary care physician. Those enrolled in the HMO before becoming eligible for Medicare were excluded, as were rollovers, patients who had seen the same physician under FFS.

For the FFS sample, primary care physicians with eligible patients were identified and recruited using master files from the American Medical Association and the American Osteopathic Association. Within the geographic areas used in selecting HMOS, 590 primary care physicians were identified. They were recruited in random number order by telephone, and their primary provider status confirmed. Screening eliminated 342 physicians, of whom 42 percent were ineligible because they could not be reached by phone, and the others for a variety of reasons: currently practicing in a staff/group model HMO, not in an office-based practice, no new patients during the study time frame, not in primary care, dead or retired. Of the 248 eligible physicians, 135 refused to participate and 113 participated in the study (recruitment rate, 46 percent). About 58 percent of the participating primary care physicians in the FFS sample reported also seeing HMO patients not enrolled in a staff/group model HMO.

Each participating physician was promised confidentiality for the information collected in the abstracting process. Stipends were offered; they were accepted by 13 percent of participating physicians. Almost 60 percent of the participating physicians were board certified in family practice or internal medicine. About 50 percent had graduated from medical school by 1970. These data were not available for individual physicians in the HMO sample because agreement to participate in the study was reached with the HMOs. However, physician lists from HMO plans overall indicated professional profiles similar to those in the FFS sample. To avoid over- or under-representing a particular style of practice in the sample, records for about 10 patients were abstracted for each physician in the two groups.

Abstraction Instrument Design

An advisory panel of physicians was assembled to develop detailed criteria lists for evaluating ambulatory care. The physicians were chosen on the basis of experience in quality of care research, background with prepaid medical plans, and recommendations from colleagues. Among them were both general internists and clinicians with expertise in geriatrics.

The panel developed three categories of ambulatory patient evaluation: medical history-taking, physical examination, and preventive screening. The first two represent time-honored bedside techniques, and as such were employed to measure the quality of care in new patient evaluations. Elements of preventive care, such as immunizations and screening procedures, were emphasized because substantial evidence supports their efficacy in health maintenance for the elderly.[6] The abstraction instrument used is available by request.

Medical Record Abstraction

Data were collected by 10 registered nurse abstractors who had been specially trained. They first completed a set of exercises mailed to them along with the training manual, and then attended five days of closely supervised sessions. For periodic health examinations abstractors were explicitly instructed to follow current guidelines.[6] When a recommended examination was not performed, statements were accepted for deferral or noncompliance (e.g., history of a hysterectomy).

Interrater reliability was assessed by having all 10 abstractors and the principal investigator (SMR) review a medical record. Agreement among raters was assessed by a variation of the kappa statistic.[7] Kappa values between raters ranged from .45 to .85, indicating good to excellent agreement among nurse abstractors for most items. The lower interrater values occurred with medical-history items, and the higher values with physical examinations, screening tests, and procedures.

Analysis

While the study focused mainly on new patient evaluations, the follow-up time for the analysis of screening tests and procedures was extended from the initial patient evaluation to the time of the chart abstraction. Because there were differences between groups, the duration of time after the patient's initial evaluation was considered a potential confounder. Others were age and gender, since many screening items are both age- and gender-specific. To control for these confounders, a logistic regression model was used to test the relationship between the patient's membership in an IPA model HMO, a staff/group HMO or a FFS system of care, and the performance of individual health maintenance procedures. Performance of each health maintenance item was used as a single binary (0–1) dependent variable. Age was entered into the model as a continuous variable. Group status, gender, and duration of follow-up time were entered as categorical variables (duration of follow-up: 0 = <20 months, 1 = ≥20 months). Maximum-likelihood estimates were computed, and 95% confidence intervals were constructed around each odds ratio.[8,9] Since the purpose of the demonstration was to determine if the care received by HMO patients was at least equal to the care received by Medicare FFS patients, odds ratios were determined by comparing performance in each HMO group of patients to the performance in FFS patients.

Results

Records of 1,590 ambulatory patients were abstracted; of these, 777 were FFS and 813 were HMO patients. Of the 813 HMO enrollees, 441 (54 percent) were from staff/group HMOs and 372 (46 percent) were from IPAs. As shown in Table 1, the FFS and the two groups of HMO patients differed on several characteristics. Patients who had new patient evaluations from both types of HMOs had been in the practices longer than FFS patients. The staff/group HMO patients had more active health problems than either IPA or FFS patients; the FFS patients were most likely to have diastolic hypertension and obesity. All three groups had similar proportions of patients with diabetes mellitus; patients from IPA HMOs were somewhat more likely than the other two groups to have a history of a previous cardiovascular event. There were slight differences in race and living situations, with patients from both HMO groups more likely to be White and less likely to live alone; however, there were considerable missing data for these two variables.

When initial medical histories in the three groups were compared, as shown in Table 2, consistent differences were

TABLE 1—Patient Characteristics in the Three Plan Types

Characteristics	FFS n = 777 (%)	IPA n = 372 (%)	Staff/Group n = 441 (%)
Mean age at time of first visit to practice (SD)	73.6 (6.4)	73.0 (5.9)	72.6 (6.0)
Mean number of months between first and most recent visits to practice (SD)	17.5 (13.6)	22.2 (12.2)	22.6 (11.1)
Mean number of ongoing health problems (SD)	3.1 (2.0)	3.1 (2.0)	3.8 (2.8)
Number of female patients	496 (63.8)	195 (52.4)	243 (55.1)
Number of married patients	290 (51.2)	181 (57.3)	253 (61.7)
Number of patients with diastolic hypertension	434 (55.9)	208 (55.9)	300 (68.0)
Number of patients with diabetes mellitus	134 (17.3)	73 (19.6)	85 (19.3)
Number of patients with history of previous cardiovascular event	252 (32.4)	90 (24.2)	172 (39.0)

TABLE 2—Initial Patient Evaluation: Percentage of Records with Selected Medical History Elements, According to Plan Type

Historical Elements	FFS n = 777	IPA n = 372	Staff/Group n = 441
Record of past health problems or hospitalizations	84.0	87.4	89.1
Record of drug allergies	72.8	82.3	83.5
Listing of current medications	82.8	90.6	90.9
Explicit Review of systems:			
Cardiovascular	58.5	61.5	64.2
Gastrointestinal	52.1	56.9	59.5
Genitourinary	47.4	54.2	61.2
Mental status	38.5	40.2	51.7
Musculoskeletal	47.0	50.4	59.7
Neurologic	43.2	48.3	57.4
Respiratory	55.0	58.0	60.4
Record of history of tobacco	63.3	70.4	78.0
Record of history of alcohol	56.4	65.9	69.2
Record of social history	39.6	39.0	38.1
Record of functional status	55.3	49.0	68.3

found. IPA and staff/group HMO patients were more likely to have their drug allergies and current medications listed. Staff/group HMO patients were more likely than either IPA or FFS patients to have a review of explicit system histories recorded, to have histories taken for tobacco and alcohol use, and to have a history of functional status recorded. Although IPA HMO patients had a greater likelihood of having an alcohol history taken than did FFS patients, they were less likely to have functional status information on their medical records.

Differences were also found among the groups with respect to the initial examinations, as shown in Table 3. As with medical histories, the consistent trend was for the records to show that examinations were more complete for patients in the two HMO models, especially the staff/group model.

TABLE 3—Initial Patient Evaluation: Percentage of Examination Elements and Laboratory Tests Recorded According to Plan Organization Type

Elements and Tests	FFS n = 777	IPA n = 372	Staff/Group n = 441
Examination of lungs	75.7	82.8	88.9
Examination of abdomen	74.1	84.7	88.4
Cardiac examination	73.6	80.1	90.9
Male genitourinary examination*	50.4	55.4	65.7
Peripheral vascular examination	50.2	49.5	59.4
Mental status examination	53.0	50.0	61.7
Neurologic examination	48.3	49.5	70.3
Urinalysis	66.4	70.2	88.9
Hemoglobin	79.4	79.8	93.9
Blood urea nitrogen or serum creatinine	71.3	75.0	93.2
Electrocardiogram	74.3	70.2	85.0

*Only males used in denominator

In Table 4, performance of periodic health examinations is examined using logistic regression. The odds ratios with 95% CI comparing the two HMO groups to FFS patients, controlling for age, sex and duration of follow-up, are shown. Overall, HMO enrollees from both models were significantly more likely than FFS patients to have periodic health maintenance as part of new patient evaluations and follow-up; however, the enrollees in staff/group model HMOs were particularly likely to receive preventive care.

Discussion

The prospect of a continuing rise in the cost of health care for the elderly increases the likelihood of further initiatives to control it. Prepaid care has already been advocated as a promising means of reducing utilization. However, rationing Medicare recipients' health care by increasing the financial risk for the provider raises questions about the quality of care.[10,11] Our findings revealed that ambulatory HMO patients were likely to receive more of the routine evaluation and health maintenance procedures than were ambulatory FFS patients. Differences in performance of screening tests currently recommended for the elderly were particularly striking for enrollees in staff/group model HMOs.

The finding that patients in HMOs had higher performance rates of recommended health maintenance items than did FFS patients suggests that attention should be given to the effects of the structure of medical practice on quality of care. Several investigators have pointed out that physician

TABLE 4—Odds Ratios of Performance of Periodic Health Examinations Comparing Medicare Patients in HMOs and FFS with Logistic Regression Analysis*

Examination/Procedure	Staff HMO::FFS	IPA HMO::FFS
Visual Acuity	3.69 (2.83, 4.80)	1.47 (1.09, 1.97)
Tonometry	8.39 (5.41, 12.99)	1.92 (1.28, 2.87)
Breast Examination	2.00 (1.41, 2.82)	1.37 (.95, 1.99)
Mammography	2.67 (1.84, 3.88)	1.31 (.84, 2.04)
Pelvic Examination	5.26 (3.51, 7.89)	1.15 (.80, 1.66)
Rectal Examination	2.90 (1.89, 4.45)	1.17 (.77, 1.77)
Fecal Occult Blood Test	3.30 (2.54, 4.28)	.93 (.69, 1.24)
Influenza Immunization	1.03 (.76, 1.39)	.44 (.30, .66)

*95% confidence intervals in parentheses

characteristics may play an important role, particularly in how they interact. Studies have suggested that medical practices incorporating significant peer interaction, such as HMOs, are more effective in reducing utilization than are those in which physicians practice independently, as in FFS practices.[12] It may also be the case that models with greater peer interaction more effectively support professional standards of care in the face of incentives to reduce utilization.[13,14] That feature of HMOs has the greatest potential to influence quality of care. Improved ambulatory care for Medicare recipients under prepaid systems may also be a result of HMOs' closer scrutiny of quality of care. Specific federal guidelines require quality assurance programs in HMOs.

While the results from this study may have important implications for health policy for the elderly, several limitations are worth noting. First, the study design did not allow for randomization of Medicare recipients into HMOs. Therefore, plan and physician selections may not have produced a representative sample, making application of the study results to the population of Medicare recipients in all HMO or FFS settings problematic; for example, it is possible that the FFS patients selected came from practices with unusually poor performance rates for standard elements of routine care. Nevertheless, among the elements of care received more often by the HMO enrollees, those constituting preventive care were most notable. Moreover, the performance rates for many of the preventive procedures received by patients in the study's FFS sample are comparable to rates reported in other FFS settings. For example, performance rates for FFS patients in nationwide surveys of clinical breast examination[15] and mammography[16] are remarkably similar to the performance rates among FFS patients in this study. Second, the study used a medical record review to determine performance rates for recommended items of routine care. It thus should be categorized as a study of the process of care and record-keeping. However, studies of the reliability and validity of medical records have established that technical aspects of medical care, such as preventive interventions, are likely to reflect actual performance.[17,18] Furthermore, for elderly patients the medical record itself is an especially important component of care, since their complex medical needs often call for multiple providers.

Although our study revealed that patients in HMOs receive medical care at least as competent as those in FFS practices receive, our analysis did not include an evaluation of individual HMOs. Our design plan was limited to representative models of group, staff, and two types of IPA HMOs, and only eight plans were studied. In that respect, the results from the evaluation of the demonstrations in this study should be interpreted with caution when establishing policy about all plans. Projects are currently under way to continue monitoring prepaid systems of care under Medicare.

Subject selection for this study was restricted to those who had been evaluated as new patients during the study time frame. However, the study did include data on patient follow-up visits for those periodic health examinations recommended after initial evaluation (e.g., annual mammography for women 50 to 59 years old). Therefore, the criteria for judging quality of care covered routine initial patient evaluations and follow-up periodic health examinations. The conclusions from the study are limited to those particular areas. Evaluations of the management of ambulatory patients with chronic illnesses, the follow-up of unexpectedly abnormal results, and the quality of care of inpatients are available in other reports.

The result that HMO enrollees received better preventive care may be due in part to the relative lack of reimbursement to FFS patients for the cost of screening tests. Others have noted that whether a patient undertakes recommended mammography, for example, is directly related to whether or not she can afford it.[19] Previous research has shown that preventive health care utilization is higher among HMO enrollees than among non-enrollees.[20] However, aside from procedures affected by reimbursement, HMO patients in this study were also more likely than FFS patients to have other examinations specifically recommended for the routine care of the elderly. For example, HMO enrollees were more likely to have current medications listed, an item especially important for elderly patients,[21] and enrollees in staff/group models were more likely to have functional status assessments. Furthermore, intraocular pressure and visual acuity were screened much more often in HMO enrollees than in FFS patients.

Efforts to regulate Medicare health costs must undergo careful scrutiny mindful of elderly patients' extensive medical needs. In this study, the scope of the conclusions is limited in that the quasi-experimental design restricted the findings to new patient evaluations in a demonstration program. The data showed that the elderly enrolled in HMOs received ambulatory care at least equal to what patients in FFS settings received; however, further evaluations are needed to verify that conclusion.

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$5 Million Available for Pediatric AIDS Grants

Approximately $5 million is available for competitive grants for three types of AIDS (acquired immunodeficiency syndrome) Projects, according to an announcement from the federal Health Resources and Services Administration (HRSA). Six to eight awards will be made for **Pediatric AIDS Health Care Demonstration Projects** intended to demonstrate or develop:

- effective ways to prevent infection; particularly through reduction of perinatal transmission;
- community-based family-centered coordinated services for infected infants, children, and youth;
- programs to reduce the spread of HIV (human immunodeficiency virus) infection to vulnerable populations of young people.

Up to three grants will be made for **Pediatric AIDS Comprehensive Centers** intended to:

- establish public-private consortia among institutions and service providers; and
- develop a service, training, and research capability to support the community service base.

Four to six grants will be awarded for **National Issues of High Priority in Pediatric AIDS**. These grants will address critical issues for people with AIDS and their families, especially in prevention, early detection, and service provision. One grant in this category will establish a National Resource Center in Pediatric AIDS to identify and deal with issues that prevent patients and families from receiving services.

Public and private entities, nonprofit and for-profit, are eligible to apply for the grants. The *application deadline is April 9*. These AIDS project grants were announced in the February 13 issue of *Federal Register*.

Application materials are available from: Grants Management Officer, Bureau of Maternal and Child Health and Resources Development, Suite 100A, 12300 Twinbrook Parkway, Rockville, MD 20852; (301) 443-1440.

AIDS Risk Behavior Patterns among Gay Men in Small Southern Cities

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Abstract: Men entering gay bars in three small southern cities were administered questionnaires to assess AIDS (acquired immunodeficiency syndrome) risk knowledge, perceived norms concerning the social acceptability of AIDS risk precautions, and personal sexual behavior. Seventy-seven percent (N = 355) of male patrons completed the measures. While risk knowledge levels were high, respondents did not perceive strong norms favoring risk reduction precautions. Rates of risk behavior were considerably higher than those reported for gay men in large urban epicenters. (*Am J Public Health* 1990; 80:416–418.)

Introduction

A number of cohort studies provide evidence that gay men in large-city AIDS (acquired immunodeficiency syndrome) epicenters have substantially modified behavior patterns to reduce risk for HIV (human immunodeficiency virus) infection.[1–5] Recent studies suggest that fewer than 20 percent of gay men in New York City and under 10 percent of gay men in San Francisco have engaged in unprotected anal intercourse during the past 12 months.[6,7] As a result, the prevalence of HIV infection among these populations has essentially reached a plateau, although at very high saturation levels.[8,9]

Less is known about the risk behavior patterns of homosexual men in smaller cities where HIV infection prevalence is still relatively low. For AIDS primary prevention efforts to be successful in these areas, there must be a rapid shift in sexual behavior norms and practices comparable to those already achieved by most gay men in AIDS epicenters. Limited research to date suggests that has not occurred.[10–12] The current study examined AIDS risk knowledge, perceived sexual behavior norms, and risk behavior patterns in a sample of homosexual men in small cities with currently low prevalence of AIDS.

Methods

Over consecutive three-night periods in the spring of 1989, all males entering gay clubs in three southern cities (Monroe, LA; Hattiesburg, MS; and Biloxi, MS) completed written questionnaires. A mean of 77 percent (range 56 percent to 85 percent) of all men entering each bar completed the study measures individually and without personal identifiers (N = 355). The three cities have metropolitan populations of 50,000 to 56,600 persons and are located at least 60 miles from any larger population center. While data on the number of AIDS cases in these cities are not available, the annual AIDS incidence in the study states (8.8/100,000 population in Louisiana and 5.8/100,000 in Mississippi) is much lower than that of states which have original AIDS epicenters (36.9/100,000 in New York and 20.9/100,000 in California).[13] Two of the cities have one gay bar while the other has two clubs; all were surveyed.

Each questionnaire requested demographic information and included probe measures of AIDS risk behavior knowledge, perception of norms concerning the acceptability of taking risk reduction precautions, and self-reported sexual activities over the preceding two months. The AIDS risk behavior knowledge measure consisted of 23 true/false items which tap practical understanding of high-risk sexual practices and risk reduction steps.[10] Five Likert-scale items (from 1 = strongly agree to 5 = strongly disagree) assessed the respondent's perception of peer norms and social consequences of taking risk reduction steps (Table 1). Each respondent also reported the number of times and the number of different sexual partners over the past two months for the sexual activities listed in Table 2.

Results

Preliminary analyses revealed no significant differences in risk behavior rates, knowledge test, and social norm scores across the three cities so data from all cities were pooled. The mean age of respondents was 28.3 years (SD = 7.9), mean educational level was 14.6 years (SD = 2.7), and 91 percent of the sample was White.

On the knowledge test, subjects correctly answered an average of 20.8 (SD = 2.3) of 23 items indicating accurate understanding of risk behavior. However, the social norms measure reflected only mixed perception that friends behaved safely or that insistence on condom use and safer sex practices would be well-accepted by others. Table 1 presents the distribution of responses for each item. While 81 percent of respondents were neutral or disagreed with the statement that condom use implies a lack of trust in a sexual partner, only 31 percent agreed or strongly agreed that their friends always use condoms during intercourse, only 43 percent agreed that peers used condoms or engaged in non-insertive practices, and 54 percent believed friends talk about safer sex more than they practice it. Respondent's mean score on these questions was 16.0 (SD = 4.1) of a maximum possible score of 25.

Eighty-three percent of respondents reported sexual activity over the preceding two months; 52 percent reported one partner and 48 percent reported multiple sexual partners. As Table 2 shows, 25 percent of all respondents reported engaging in unprotected insertive, and 23 percent unprotected receptive, anal intercourse during the past two months. For the total sample, the mean frequencies of these behaviors were 1.41 and 1.04 occurrences respectively. However, for subjects who engaged in any unprotected anal intercourse, the mean frequencies were 5.55 and 4.59 respec-

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TABLE 1—Responses to Items Assessing Perception of Social Norms Concerning AIDS Risk Precautions

Statements	Respondents Who: Strongly Disagree N (%)	Disagree N (%)	Neutral N (%)	Agree N (%)	Strongly Agree N (%)
My friends always use condoms during intercourse	62 (18)	52 (15)	129 (36)	61 (17)	50 (14)
My friends talk about safer sex more than they actually practice it	35 (10)	36 (10)	92 (26)	85 (24)	107 (30)
My friends believe that insisting on safer sex implies that you don't trust your partner	146 (41)	40 (11)	102 (29)	36 (10)	31 (9)
Safer sex is completely accepted by my friends	27 (8)	37 (10)	85 (24)	74 (21)	132 (37)
My friends either use condoms or have sex that doesn't involve fluid exchange	47 (13)	44 (12)	114 (32)	63 (18)	87 (25)

N = 355

TABLE 2—Risk Behavior Patterns Reported by Study Respondents

Behavior	Number and Percentage of Total Sample Reporting Behavior in Past Two Months	Mean Frequency for Entire Sample in Past Two Months	Mean Frequency for Respondents Who Report Any Occurrence of the Behavior in Past Two Months	Mean Number of Partners for Behavior in Past Two Months for Subjects Who Report Any Occurrence of the Behavior
Unprotected anal intercourse as insertive partner	88 (25)	1.35 (4.85)	5.55 (9.45)	1.11 (.56)
Unprotected anal intercourse as receptive partner	81 (23)	1.11 (4.06)	4.59 (7.08)	1.03 (.62)
Unprotected oral intercourse as insertive partner, fluid exchanged	123 (35)	1.59 (3.76)	5.46 (11.12)	1.58 (2.91)
Unprotected oral intercourse as receptive partner, fluid exchanged	102 (29)	1.85 (6.11)	6.67 (11.08)	2.19 (5.75)

N = 352

tively. A proportionate calculation revealed that condoms were used by the insertive partner in only 51 percent of all anal intercourse occasions. In addition, 35 percent of the sample reported unprotected insertive, and 29 percent in receptive, oral intercourse with fluid exchange over the same two-month period. Finally, a median split was performed to subdivide the sample into those with high and those with low total scores on the social norm measure. Respondents with high social norm scores engaged less often in insertive and receptive unprotected anal intercourse and in receptive oral sex (Table 3).

TABLE 3—Means and 95% Confidence Intervals Comparing Sexual Behavior Practices of Respondents Scoring Above and Below Median in Social Norm Total Score

Sexual Practice Variable	Perceived Social Norm Score Median Split: Respondents in Upper Median M (95% CI)	Respondents in Lower Median M (95% CI)
Unprotected Insertive Anal Intercourse	0.6 (0.2,0.9)	2.3 (1.2,3.4)
Unprotected Receptive Anal Intercourse	0.4 (0.1,0.7)	1.7 (0.9,2.5)
Unprotected Insertive Oral Intercourse, Fluid Exchanged	1.3 (0.6,2.0)	2.6 (1.2,4.0)
Unprotected Receptive Oral Intercourse, Fluid Exchanged	0.8 (0.3,1.4)	3.0 (1.7,2.7)

N = 352

Discussion

Rates of high-risk sexual behavior in these cities are much greater than those recently reported for gay men in most large AIDS epicenters. The percentage of respondents in this study reporting unprotected anal intercourse during just the past two months is two to three times higher than the percentage of San Francisco gay men who report engaging in this practice during the last 12 months.[7] Conclusions that homosexual men have already effectively made changes needed to reduce risk for new HIV infection based upon large-city cohorts may not generalize to smaller cities.

Factual knowledge about AIDS was high in this sample. However, we suspect that gay men in small cities encounter fewer social supports for making AIDS precautionary behavior changes, do not benefit from well-organized community social structures which permit efficient behavioral norm redefinition, and still perceive AIDS as a distant threat. These social norms have considerable impact on gay mens' sexual behavior.[14]

The cross-sectional method used in this study does not permit conclusions to be drawn concerning behavior changes which may have occurred from earlier time periods, the sample is limited to patrons of gay bars in several southern cities, and there is a wide range in behavior reported by these respondents. Nonetheless, intensified primary prevention interventions are necessary for the many gay men in communities outside the urban areas who remain at great risk for HIV infection.

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NIH Consensus Development Conference on Colon/Rectum Cancer, April 16–18

Adjuvant Therapy for Patients with Colon and Rectum Cancer is the subject of an upcoming consensus development conference sponsored by the National Institutes of Health (NIH). The conference, scheduled for April 16–18, 1990, in Masur Auditorium of the NIH Clinical Center, is open to the public.

The purpose of the conference is to reach agreement on adjuvant therapy for patients with colon and rectum cancer, as well as directions for future research.

Large bowel adenocarcinoma is a major public health problem in the United States. More than 150,000 new cases of colon and rectum cancer will be diagnosed in 1990 and approximately 75 percent will have a primary surgical resection with the hope of complete tumor eradication. Despite this high resectability rate, nearly half of all colorectal cancer patients die of metastatic tumor.

The conference will bring together specialists in surgery, oncology, gastroenterology, pathology, statistics, and other relevant fields. On the first two days, experts will present current scientific thinking about the diagnosis, management, and prevention of adjuvant therapy for patients with colon and rectum cancer, and concerned voluntary organizations will be invited to make statements. On the third day, after considering the scientific evidence, the consensus panel will present its draft report and invite comments from the audience. Glenn D. Steele, Jr., MD, PhD, New England Deaconess Hospital, will chair the panel.

To register for the Consensus Development Conference on Adjuvant Therapy for Patients with Colon and Rectum Cancer, or to obtain further information, contact: Conference Registrar, Prospect Associates, Suite 500, 1801 Rockville Pike, Rockville, MD 20852; (301) 468-MEET. FAX: (301) 770-5164.

HIV-1 Counseling and Testing Sites, Minnesota: Analysis of Trends in Client Charactertistics

Richard N. Danila, PhD, MPH, James M. Shultz, MS, PhD, Michael T. Osterholm, PhD, MPH, Keith Henry, MD, Margaret L. Simpson, MD, and Kristine L. MacDonald, MD

Abstract: We report here a summary of the data obtained from two HIV-1 antibody counseling and testing sites in Minneapolis-St. Paul for the first 48 months of operation (24,911 persons tested). The HIV-1 antibody seroprevalence rate for all persons tested was 5 percent. The highest seroprevalence rates were in male homosexual/bisexual intravenous drug users (23 percent) and homosexual/bisexual men (13 percent). There was a significant decrease in the HIV-1 antibody seroprevalence rate among clients during the 48-month period from 14 percent in the first six months to 3 percent in the last six months. This decrease coincided with an increase in the number of low-risk female clients and low-risk heterosexual male clients, and a decrease in the number of homosexual/bisexual males participating in the programs. These findings suggest the need for development and implementation of other strategies to identify and reach persons at highest risk for HIV-1 infection. (*Am J Public Health* 1990; 80:419–422.)

Introduction

In July 1985, the Minnesota Department of Health (MDH) established human immunodeficiency virus (HIV-1) counseling and testing sites at the two major public health sexually transmitted disease clinics in the Twin Cities (Minneapolis, St. Paul) to allow persons at risk for HIV-1 infection to ascertain their HIV-1 antibody status.

To examine the potential role of these Minnesota programs in HIV-1 infection prevention efforts, data on client demographic and risk factor characteristics and HIV-1 antibody seroprevalence were analyzed for the first 48 months of operation (July 1985 to June 1989).

Methods

The two programs provide free services to all persons requesting them. Prospective program clients are scheduled for appointments by telephone. All clients are interviewed by trained nurses or clinicians according to protocols established by the US Public Health Service Centers for Disease Control[1] and the MDH (available upon request to authors). During pre-test and post-test sessions, staff assess client risks for HIV-1 infection, interpret HIV-1 antibody test results, and provide individualized risk reduction and disease prevention counseling. Self-reported demographic and risk factor data are recorded on standardized client information sheets.

Serum specimens are initially screened for HIV-1 antibody using enzyme immunoassay (EIA) test kits from Abbott Laboratories, Electronucleonics, or Genetic Systems. Specimens are considered reactive if the absorbance values are above the cutoff levels specified by the manufacturers' instructions. Initially reactive specimens are tested a second time; specimens that are repeatedly reactive are then tested using the Western blot technique.[2] Western blot testing utilizes HIV-1 antigen from Bio-tech Dupont, Rockville, Maryland, or Litton Bionetics, Charleston, South Carolina. Specimens are considered positive by Western blot assay if the following bands are detectable: p24 or p31 and either gp41 or gp120/gp160, with or without additional HIV-1 characteristic bands.[3] Specimens with other HIV-1 band patterns not meeting the criteria for a positive assay are considered to be indeterminate.

Data were analyzed for 24,911 clients who had HIV-1 antibody serology completed from July 1985 through June 1989. Male clients were categorized into one of five HIV-1 transmission categories: homosexual/bisexual, homosexual/bisexual intravenous drug user, heterosexual intravenous drug user, heterosexual sex partner of a person at high risk for HIV-1 infection, or other (immigrant from a high-risk country or no identified risk). Female clients were categorized into one of three HIV-1 transmission categories: intravenous drug user, sex partner of a person at high risk for HIV-1 infection, or other (immigrant from a high-risk country, female prostitute, or no identified risk). Data were analyzed using standard univariate methods and chi-square test for trend measures.[4]

Results

Between July 1, 1985 and June 30, 1989, 25,306 client visits were recorded; 24,911 clients (98 percent) were tested for HIV-1 antibody and 395 clients (2 percent) declined testing after the pre-test visit. An increase occurred in the number of clients tested per month over the first 36 months with a leveling over the last 12 months (Figure 1). The HIV-1 antibody seroprevalence rate for all clients tested was five percent (1,299 of 24,911).

First-time and Repeat-Test Clients

For tested clients, 18,288 (73 percent) were persons being tested for HIV-1 antibody for the first time (first-time clients) and 6,623 (27 percent) were clients who had been tested at least once before (repeat-test clients). The HIV-1 antibody seroprevalence rate for first-time clients (898 of 18,288; 5 percent) was lower than the rate for repeat-test clients (401 of 6,623; 6 percent). For the repeat-test clients, 2,672 (40 percent) had been tested previously at a health department site, 353 (5 percent) at a family planning clinic, 306 (5 percent) at a physician's office, 81 (1 percent) at a plasma center, 58 (1 percent) at a blood collection facility, and 3,153 (48 percent) at other unspecified sites. Of the 401 clients who were HIV-1 antibody positive on the repeat test: 175 (44 percent) had tested positive for HIV-1 antibody previously; nine (2 percent) had prior tests with indetermi-

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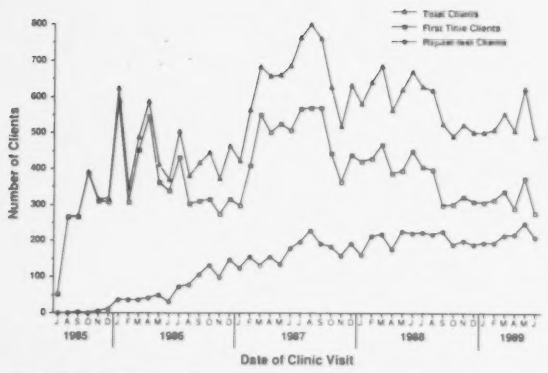

FIGURE 1—Number of Clients Attending Counseling and Test Site Programs Tested for HIV-1 Antibody by Visit. Status by Month, Minnesota, 1985-89

nate Western blot test results; 107 (27 percent) had previously tested either EIA negative or EIA reactive and Western blot negative; 33 (8 percent) had tested EIA reactive previously but had not had confirmatory Western blot testing performed; and information on previous testing was unavailable for 77 (19 percent).

Most clients (91 percent for first-time clients) were between 20 and 49 years of age. The HIV-1 antibody seroprevalence rate was higher in males than females (Table 1). The HIV-1 seroprevalence rate for Blacks was 1.6 times higher than the rate for Whites and the rate for Hispanic clients was 1.8 times as high as the rate for Whites.

The highest HIV-1 seroprevalence rate was found in male homosexual/bisexual intravenous drug users (23 percent; Table 2). Rates were also analyzed by transmission category and race for first-time clients; higher rates were consistently found in Black and Hispanic males (data available upon request to authors). HIV-1 seroprevalence rates for first-time clients were 6 percent and 4 percent at each of the two clinics, respectively; the higher seroprevalence rate was due to a larger proportion of homosexual/bisexual male clients seen at that site.

TABLE 1—HIV-1 Antibody Seroprevalence Rates by Gender, Age, and Race for First-Time Clients; Minnesota Counseling and Testing Sites; July 1985 through June 1989

Client Characteristics	No. Tested (%)	No. HIV-1 Anitbody Postive	Seroprevalence Rate
Total	18,288 (100.0)	898	4.9
Gender			
Male	12,085 (66.1)	876	7.2
Female	6,128 (33.5)	22	0.4
Unrecorded	75 (0.4)	0	0.0
Age Group (years)			
0–12	22 (0.1)	2	9.1
13–19	612 (3.3)	9	1.5
20–29	7,495 (41.0)	384	5.1
30–39	6,830 (37.3)	377	5.5
40–49	2,307 (12.6)	89	3.9
50–59	598 (3.3)	22	3.7
60 and over	229 (1.3)	3	1.3
Unrecorded	195 (1.1)	12	6.2
Race			
White	16,609 (90.8)	788	4.7
Black	1,020 (5.6)	79	7.7
Hispanic	188 (1.0)	16	8.5
Other*	303 (1.7)	11	3.6
Unrecorded	168 (0.9)	4	2.4

*Includes Native American and Asian/Pacific Islander.

TABLE 2—HIV-1 Antibody Seroprevalence Rates by Client Transmission Categories for First-Time Clients, Minnesota Counseling and Testing Sites, July 1985 through June 1989

HIV-1 Transmission Categories	No. Tested (%)	No. HIV-1 Antibody Positive	Seroprevalance Rate (%)
Males			
Homosexual/Bisexual	5,866 (48.5)	761	13.0
Homosexual/Bisexual Intravenous Drug User	172 (1.4)	40	23.3
Heterosexual/Intravenous Drug User	803 (6.7)	50	6.2
Heterosexual Sex Partner at High-Risk	1,403 (11.6)	5	0.4
Other Heterosexual/ No Identified Risk	3,841 (31.8)	20	0.5
Total	12,085 (100.0)	876	8.1
Females			
Intravenous Drug User	405 (6.6)	6	1.5
Sex Partner at High-Risk	1,881 (30.7)	7	0.4
Other/No Identified Risk	3,842 (62.7)	9	0.2
Total	6,128 (100.0)	22	0.4

Analyses of Trends Over Time

A significant downward trend in the monthly HIV-1 antibody seroprevalence rate was noted over time for all clients tested (both first-time and repeat-test clients) ($p < 0.001$) (Figure 2). The seroprevalence rate for all clients tested during the six months of testing in 1985 was 14.3 percent. The seroprevalence rate dropped to 8.3 percent during 1986 and declined further to 3.9 percent during 1987 and to 3.5 percent during 1988. During the first six months of 1989, the overall seroprevalence rate was only 2.5 percent.

The number and proportion of repeat-test clients increased significantly over time ($p < 0.001$) (Figure 1). Repeat-test clients accounted for 1.2 percent of all clients during the first six months of operation in 1985, but comprised 15.9 percent of all clients in 1986, 26.1 percent of all clients in 1987, 35.0 percent of all clients in 1988, and 40.2 percent of all clients during the first six months of 1989. This increase in the proportion of repeat-test clients did not account for the observed decline in the overall HIV-1 antibody seroprevalence, since the seroprevalence rate for repeat-test clients was actually higher than for first-time clients, as noted previously.

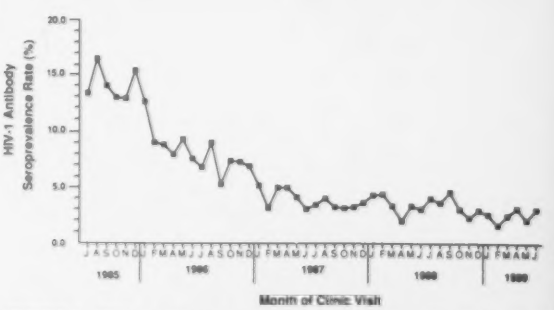

FIGURE 2—HIV-1 Antibody Seroprevalence Rates among Clients Attending Counseling and Test Site Programs by Month, Minnesota, 1985–89

Analysis of data from first-time clients only demonstrated that the HIV-1 seroprevalence declined from 14.2 percent for the first six months of operation in 1985 to 1.8 percent for the last six months of operation in 1989. The HIV-1 seroprevalence rate for repeat-test clients similarly declined from 26.3 percent in the first six months in 1985 to 3.5 percent in the last six months of operation in 1989 (data available on request to authors).

Major changes were noted in client characteristics over the 48-month period. Initially, most clients were at increased risk for HIV-1 infection; however, the number and proportion of clients at low risk for HIV-1 infection increased over time. During the first six months of the program, females comprised 8 percent of all first-time clients, whereas they made up 43 percent of all first-time clients during its last six months. During the first six months, 54 percent (69 of 128) of the females were at increased risk (intravenous drug user or sex partner of a person at high risk) and 46 percent (59 of 128) were at low risk; whereas, during the last six months, 33 percent (261 of 795) were at increased risk and 67 percent (534 of 795) were at low risk. There were no seropositive females in the first six months and only one for the last six months. During the first six months, 81 percent of all first-time clients were homosexual/bisexual males and 10 percent were heterosexual males; whereas, only 14 percent were homosexual/bisexual males and 43 percent were heterosexual males during the last six months of observation (Figure 3). For homosexual/bisexual male clients, a significant decline in HIV-1 antibody prevalence over time was noted ($p < 0.05$) (Figure 4).

Discussion

Counseling and testing site programs were originally conceived, funded, and opened in 1985 to assure safety of the blood supply by providing alternate sites for potential volunteer blood donors to ascertain their HIV-1 antibody status.[5] They have become a major cornerstone for AIDS (acquired immunodeficiency syndrome) prevention programs throughout the country and an integral part of the Minnesota statewide plan for AIDS prevention.[6] In evaluating the contribution of these programs to primary prevention of HIV-1 transmission, three issues need to be considered.

- First, are they currently serving clients most at risk for HIV-1 infection?
- Second, are they successful in HIV-1 infection prevention efforts through effecting and maintaining behavior change in clients?

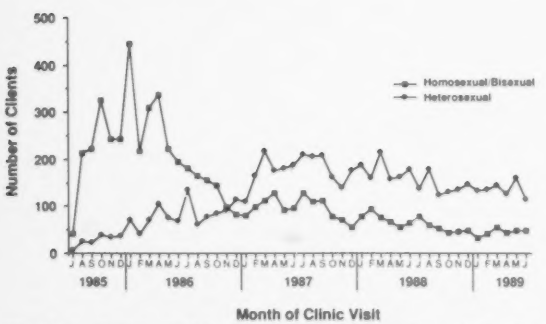
Number of Clients
500
400
300
200
100
0
Homosexual/Bisexual
Heterosexual
J A S O N D J F M A M J J A S O N D J F M A M J J A S O N D J F M A M J J A S O N D J F M A M J
1985
1986
1987
1988
1989
Month of Clinic Visit

FIGURE 3—Number of Male Clients Attending Counseling and Test Site Programs by Month, by Sexual Orientation, Minnesota, 1985–89

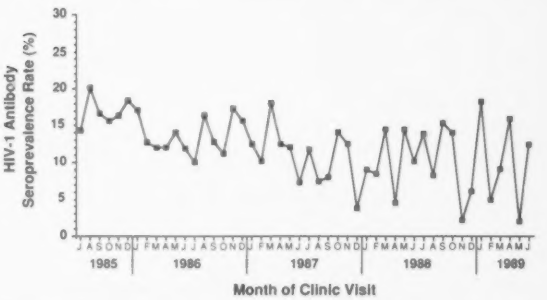
HIV-1 Antibody Seroprevalence Rate (%)
30
25
20
15
10
5
0
J A S O N D J F M A M J J A S O N D J F M A M J J A S O N D J F M A M J J A S O N D J F M A M J
1985
1986
1987
1988
1989
Month of Clinic Visit

FIGURE 4—HIV-1 Antibody Seroprevalence Rate for Homosexual/Bisexual Males Attending Counseling and Test Site Programs by Month, Minnesota, 1985–89

- Third, are current counseling-testing program activities the best use of public health resources or are additional outreach strategies needed to target persons at highest risk of infection?

As these programs evolved in Minnesota over this 48-month observation period, they progressively served fewer clients at increased risk for HIV-1 infection and increasingly served clients at low risk for HIV-1 infection. A significant decrease in the number of homosexual/bisexual males seeking counseling and testing occurred over time. The HIV-1 antibody seroprevalence in homosexual/bisexual males seeking program services later in the observation period was lower than those seen early in the observation period, suggesting that men seen later were at lower risk for HIV-1 infection. In addition, 27 percent of the clients seen during this time period had been tested previously. Of these, most (97 percent) who initially tested negative were still negative for HIV-1 antibody on repeat testing. Some were clients who were instructed to return for follow-up testing to confirm their seronegative status, since they were in a potential "window-period" for HIV-1 antibody development based on their exposure history. Some of these repeat-test clients possibly were persons who continued to engage in high-risk behaviors. However, based on seroprevalence, it is likely that the majority of these repeat-test clients represent a low risk "worried well" population. Also, of the repeat-test clients who tested HIV-1 antibody positive, 175 (44 percent) had tested positive on a previous occasion, indicating that they likely were returning only to verify their infection status.

Counseling and testing site program policies remained stable during this time period. HIV-1 infection is a reportable condition in Minnesota and has been since shortly after the opening of these programs (October 1985), but clients are not denied services if they refuse to give a name or show proof of identity. This decrease in the number of persons at increased risk and corresponding increase in the number of persons at low risk for HIV-1 infection cannot be attributed to any change in program policy over the observation period. Also, similar trends demonstrating a decrease in HIV-1 seroprevalence at programs have been noted elsewhere in the country in states where HIV-1 infection is not reportable.[7–9]

Because long-term behavioral data were not collected at the sites, we have no direct measures of the effectiveness of our programs in promoting behavior change which prevents HIV-1 infection. Data on repeat-test clients can be used as an indirect measure. Of the 401 clients who were HIV-1 antibody positive on repeat testing, 107 (27 percent) had tested negative

previously; data are not available on the time of the previous test for 28 (26 percent) of the clients. Eighteen (17 percent) had tested HIV-1 antibody negative within the previous six months and thus may represent persons who were infected but had not yet seroconverted at the time of the prior test. However, 61 (57 percent) of the 107 clients reported testing HIV-1 antibody negative in the previous seven months or longer and thus may represent new infections due to ongoing high-risk activity, despite having received risk reduction counseling at the time of previous testing.

Current data regarding knowledge of HIV-1 infection status and subsequent behavior change are unclear and suggest different effects. Knowledge of HIV-1 infection, particularly of a positive status, has been associated with a reduction of high-risk behavior in some studies[10–17]; however, only one of these studies examined persons tested at counseling and testing sites. Other studies have not demonstrated behavior changes associated with knowledge of HIV-1 infection.[18–21] These studies may not necessarily be extrapolated to persons tested at counseling and testing sites nor are there data regarding long-term behavior change after a single instance of counseling and testing.

In recent years, large amounts of federal and state dollars have gone into expanding and maintaining HIV-1 counseling and testing sites. Approximately $1.4 million was spent in Minnesota on this program at these two sites during this 48-month observation period. Despite expenditure of these resources, the trends identified here indicate that the program may not be reaching persons at highest risk and that providing free services to anyone requesting them may not be the best use of available public health dollars for AIDS prevention. In areas such as Minnesota where heterosexuals (other than intravenous drug users) are at low risk for HIV-1 infection, services need to be targeted to those at highest risk. In addition, counseling and testing sites might attract more persons at increased risk for HIV-1 infection by being linked to additional services such as HIV-1 disease diagnostic and treatment services.

Strategies to both identify and develop outreach programs for those at highest risk are also needed. Clinic-based sites likely to provide services to persons who are engaging in high-risk behaviors, such as chemical dependency treatment programs and clinics for treatment of sexually transmitted diseases, need to have well-developed risk assessment programs and on-site availability of HIV-1 antibody testing. Other outreach strategies, such as programs involving street workers, need to be expanded. Third-party partner notification programs, which actively and selectively reach those at highest risk for HIV-1 infection, need to be considered by state health departments as another outreach mechanism.[22–24] The data presented here may not be applicable to all areas of the country, particularly where HIV-1 infection has become endemic in certain racial and high-risk groups. Criteria can be developed locally to determine which persons are at highest risk for HIV-1 infection and testing sites can then be tailored accordingly.

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Potential for Laboratory Exposures to Biohazardous Agents Found in Blood

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Abstract: The magnitude of risk for occupational exposures to biohazardous agents found in blood was assessed by 800 environmental samples taken from a total of 10 clinical and research laboratories at the National Institutes of Health (NIH). Thirty-one samples from 11 work stations in three laboratories contained hepatitis B virus surface antigen (HBsAg). Observations of workers indicated that environmental contamination arose from several sources. Among the 11 work stations with HBsAg environmental samples, eight had high work loads, seven had inappropriate behaviors, and nine had flawed laboratory techniques. This information suggests that a multifactorial approach is needed to minimize the risk of laboratory-associated infections. (*Am J Public Health* 1990;80:423–427.)

Introduction

Hepatitis B virus (HBV) infection is among the most commonly reported laboratory-associated infections.[1–4] Laboratory workers in an urban medical center are at almost three times the risk of acquiring HBV infection than other hospital employees, based solely on exposure to patients' blood.[5] HBV infection is seven to 10 times more common among occupational groups in laboratory medicine than in the general public.[6,7] Workers' anxiety is heightened because of the concomitant risk of acquiring other infections with human immunodeficiency virus (HIV) and agent(s) of non-A, non-B hepatitis.

Strict adherence to biosafety guidelines minimizes the risk of acquiring laboratory-associated infections.[8,9] Immunization of workers is clearly the most effective means to control infection.[10] However, many employees are reluctant to be immunized and, as is the case with HIV, licensed vaccines are not always available. Thus, these types of laboratory-associated infections continue to occur,[11] despite the fact that most are preventable. Recently, universal blood and body-fluid precautions were recommended to further enhance workers' safety.[12]

This study dealt with an assessment of similarities and differences in clinical and research laboratory activities concurrent with a survey to detect hepatitis B surface antigen (HBsAg) in the environment. For this study, HBsAg served as a marker for contamination with pathogens found in human blood. Based on data from viral survivability on fomites, the selection of HBsAg as a marker for blood-borne contamination exaggerates the viability of a labile microorganism such as HBV. Nevertheless, contamination with HBsAg suggests a common source and depicts a potential for transmission of infection in the laboratory. Given this caveat, the purpose of this study was to elucidate factors associated with HBsAg in the environment and, therefore, potential exposures to blood-borne pathogens in biomedical laboratories at the authors' institution.

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Methods

Recruitment and Stratification of Laboratories

Ten laboratories, representing a cross section of operations involving the use of human blood at the National Institutes of Health (NIH), were entered into a survey conducted from August 1984 through February 1985. We invited 119 laboratory workers to participate. Each volunteer was assigned a code to assure anonymity.

The laboratories surveyed fulfilled criteria for assignment to Biosafety Level II[8] and were chosen to represent a spectrum from high to low risk of workers handling blood containing HBV. The 10 participating laboratories were divided into 44 distinct work stations for purposes of data collection. Each of these stations was assigned a level of risk prior to environmental sampling based on the volume of blood processed and the hypothesis that the probability of detection would be greater in laboratories which processed blood drawn from individuals known to be infected with HBV than from those which excluded such blood samples. High-risk work stations were arbitrarily defined as those locations where the estimated volume of blood processed per week equaled or exceeded 1.5 L or the specimens received were from individuals known to belong to groups with a high prevalence of HBV infection. Stations assigned to the low-risk category were arbitrarily defined as areas where workers processed less than 1.5 L blood per week, with all blood drawn from donors known to be negative for HBsAg.

There were 75 hours of direct observation with the amount of time per laboratory depending on the continuity of tasks involving the use of blood. Observations were obtrusive because this approach allowed for interviews with workers while laboratory activities were assessed.[13] Data obtained in this manner may have been biased because of the worker's apprehension and/or attempts to modify unsafe practices. To minimize worker's reaction to being observed, the investigator sought to establish a good rapport with participants, reiterate key issues of the study, and assure the worker's anonymity. Stratification of risk in the work stations of the laboratories surveyed and the observation periods completed are listed on Table 1.

Laboratories 2 and 3 (Clinical Pathology and Transfusion Medicine Departments, respectively) provided diagnostic services for the Warren G. Magnuson Clinical Center (the hospital at NIH); the majority of work stations in these two laboratories were classified in the high-risk category. The work completed in Laboratory 4 was classified in the high-risk category because numerous specimens were analyzed for large-scale serological surveillance studies. Individuals in

TABLE 1—Distribution of Laboratories, Type of Operations, Level of Work Station Risk, and Observations

Laboratory Code	Operation	Categories of Risk: High (No.)[a]	Categories of Risk: Low (No.)[a]	Observations (h)[b]
1	Immunological profiles	0	2	2
2	Diagnostic	14	1	28
3	Diagnostic	11	1	15
4	Immunoassays	2	0	4
5	Immunological profiles	3	0	5
6	Immunological profiles	0	1	2
7	Flow cytometry	1	0	1
8	Liver disease research	3	0	4
9	Tumor immunology	3	0	9
10	Virology research	2	0	5
Total		39	5	75

[a]Number of work stations.
[b]Hours.

Laboratories 5, 7, 8, and 10 processed blood specimens that were obtained from patients with chronic liver disease or HIV infection. In Laboratory 9, a tumor immunology unit, workers analyzed specimens from patients likely to have a history of multiple blood transfusions.

Assessment of Laboratory Operations

Methods for assessing skilled performance (both behaviors and techniques) were adapted from a task strategies approach.[14] To control for spurious conclusions due to inconsistencies with data collection,[13,14] all data were recorded by a single investigator who reviewed the study parameters frequently to control for observer drift. No intervention was attempted during the study.

Observation of activities involving the use of human blood were assessed by two indices. First, the frequencies of 13 different manipulations were each tallied to estimate the extent of workers handling blood that was required in order to complete a task. A task was defined as the cumulative manipulations generating a single result. The second index was the work load (the number of blood specimens processed per week). These indices allowed for comparison of laboratories performing quite different operations.

Behaviors were also assessed in an attempt to identify practices associated with an increased risk of contamination. Specific behaviors were assessed when a worker performed a movement either accidentally or intentionally that involved contact between human blood and the employee's skin, conjunctiva, or mouth. Each behavior was classified either as appropriate, if the practice was consistent with biosafety recommendations,[8,9] or inappropriate, if the worker was placed at risk of an exposure (e.g., mouth pipetting fluids or skin contact with blood) as a result of a breach in good laboratory practices. In the event of an overt accident the worker's behavior was judged to be appropriate only if the contamination occurred despite good laboratory practices (e.g., mechanical failure or poor instrument design). Observations were tallied prior to knowledge of sample testing.

Environmental Survey

Samples collected for the detection of HBsAg were processed in a manner consistent with Biosafety Level II guidelines.[8] HBsAg was eluted from various surfaces using a modification of a procedure described by Favero, *et al.*[15,16] The elution fluid (FBS/PBS) was prepared using 20 percent fetal bovine serum (Lot #14-501B: M. A. Bioproducts, Walkersville, MD) (FBS), phosphate-buffered saline (0.15 M NaCl and phosphate: M. A. Bioproducts, Walkersville, MD) (pH 7.4 ± 0.5) (PBS), and sodium azide (1.0 mg/ml) (Sigma Chemical, St. Louis, MO).

Samples were collected from work surfaces measuring approximately 35 cm^2 using moistened swabs and from swatches of gloves worn by workers or absorbent paper placed on the work surface prior to handling blood. Pledgets (each with a surface area measuring 5.0 cm × 7.5 cm) cut from Grade 304 stainless steel by the Biomedical Engineering and Instrumentation Branch, NIH, Bethesda, MD, were used for indirect sampling. These pledgets were placed in the laboratories either in the working area or, for negative controls, outside the working space. Negative controls were graded unsatisfactory if the pledgets were either moved or visibly soiled during the exposure period. Metal pledgets were used because preliminary modeling studies demonstrated that nanogram amounts of HBsAg could be eluted from four surfaces (absorbent paper, glass, polystyrene, and stainless steel) with recovery rates averaging 20 percent, 77 percent, 82 percent, and 96 percent, respectively. Other data have shown that HBsAg can be recovered from metal surfaces with greater than 99 percent efficiency.[15] All samples were eluted in 1.5 ml FBS/PBS and stored at 4°C.

Hepatitis B Surface Antigen Detection

Samples were screened for the presence of HBsAg using a radioimmunoassay (Ausria II: Abbott Laboratories, Chicago, IL) according to the manufacturer's instructions with these modifications: a) each control and sample was assayed in duplicate using 200 ul eluate per well; b) six distinct negative environmental controls were assayed with each run; and c) each assay tube was counted for 10 min. Tubes were counted in a gamma spectrophotometer (Tracor Analytic, Model 1185: Nuclear-Chicago, Des Plains, IL) with a calculated counting efficiency for I-125 of 83 percent. The average cpm for each paired assay was calculated. Samples with discrepant paired results were retested. Those samples with repeatedly discrepant results were excluded from the study.

A sample was classified as reactive if the cpm for the sample was greater than three standard deviations from the mean cpm for the negative controls. The coefficient of variation (CV) was calculated and the mean of the negative controls for each screening run (values ranged from 3 percent to 17 percent with a mean of 7 percent).

Reactive samples were confirmed using specific neutralizing antibody for HBsAg (anti-HBsAg) (Ausria II Confirmatory Neutralizing Kit: Abbott Laboratories, Chicago, IL). Appendix A outlines the method used for the confirmation assay. A sample was confirmed HBsAg positive if the reduction in the cpm of the sample reacted with anti-HBsAg was greater than 50 percent compared with the sample reacted with FBS/PBS and if these latter cpm were between the range of 50 percent to 150 percent of the mean cpm of the negative control. Reactive samples with cpm less than 1.5 times the mean cpm of the negative control were diluted beyond the sensitivity of this latter assay and were interpreted as borderline. Within run variation for the confirmation runs was expressed as the CV calculated for the negative control (values ranged from 39 percent to 59 percent with a mean of 4 percent).

Statistics

The association of the potential factors with HBsAg was assessed by determining odds ratios and 95 percent confidence intervals.[17]

Results

The results for the HBsAg sampling data are summarized on Table 2. These data show that our hypothesis was correct: high-risk operations do differ from low-risk operations in terms of the potential for blood-borne contamination in the laboratory. All environmental samples collected from the low-risk work stations were negative for HBsAg. A total of 69 samples collected from high-risk work stations in Laboratories 2–4 and 8–10 were reactive for HBsAg on the initial screening. Except for the tumor immunology research unit, these laboratories routinely processed blood drawn from individuals chronically infected with blood-borne pathogens. Approximately 55 percent (38/69) of the reactive samples were graded as borderline (low cpm for positive samples overlapped with high cpm for negative controls). Insufficient eluate volumes prohibited further analysis of these borderline samples. Thus, we analyzed a total of 800 samples, of which 31 (3.9 percent) were HBsAg positive, 38 (4.8 percent) were borderline, and 731 (91.4 percent) were negative.

Samples confirmed positive for HBsAg were collected from Laboratories 2, 3, and 8. The HBsAg positivity from these laboratories ranged from a high of 17.8 percent (8/45) (liver disease research unit) to a low of 1.4 percent (2/139) (Transfusion Medicine). The frequency of obtaining positive samples in Clinical Pathology was 8.6 percent (21/245).

Factors Associated with HBsAg in High-Risk Work Stations

Individuals who worked in nine different locations in the Clinical Chemistry and Hematology Services had the highest work loads (average 1,430 specimens/week: range from 210–2,700). Workers in the Transfusion Medicine Department averaged 106 specimens per week. The liver disease unit, the laboratory with the greatest percent of samples positive for HBsAg, averaged 27 specimens per week. Work loads for Laboratories 4 and 7 averaged 1500 and 350, respectively. The remaining laboratories analyzed fewer than 60 specimens per week (range from 13–60).

Inappropriate behaviors associated with an obvious risk for cutaneous contact with blood were identified at nearly 40 percent (15/39) of the areas surveyed. One example of these inappropriate work practices which occurred was that workers routinely handled contaminated fomites without wearing gloves. Despite the presence of warning labels (e.g., blood and body-fluid precautions) on some specimens, workers at 60 percent (9/15) of these same areas were not motivated to wear gloves. Workers who consistently wore gloves when working with blood (whether a warning label was present or absent) were observed at only 33 percent (13/39) of the stations. Surprisingly, two workers who routinely analyzed blood donned gloves only when they handled blood with a warning label.

Four workers committed flagrant violations of good work practices during the observations. These individuals either mouth-pipetted serum or live virus suspension or splashed milliliter amounts of body-fluid onto their skin. They were also observed to disregard basic hygienic practices (e.g., placing laboratory marking pens in the mouth) and misused equipment. Examples of other unsafe work practices observed in this study included: sonicating infectious materials without physical containment and hand braking a centrifuge rotor. Curiously, three employees acknowledged that they were aware of the risks associated with their actions.

Table 3 shows the frequencies and crude odds ratios of HBsAg contamination in 39 high-risk work stations. High work load and flawed technique were associated with the greatest risk of contamination, whereas there was a borderline association with inappropriate behavior (Table 3). Flawed techniques were of two kinds: those related to instrument design and those related to procedures which were intrinsically defective (i.e., workers may have been following the procedure as described, yet blood spills routinely occurred).

Approximately 85 instruments that workers used during the study were sampled for HBsAg. The design of these instruments was considered flawed if a mechanical feature generated a spatter of blood or necessitated frequent maintenance of components contaminated with body-fluids. HBsAg was detected on surfaces near several instruments where blood was spattered: Sequential Multiple Analyzer and Computer, Model SMAC II Systems: Technicon Instruments Corporation, Tarrytown, NY; Automated pH/Blood Gas System, Model 175: Corning Medical and Scientific, Medfield, MA: Pentawash II: Abbott Laboratories, Chicago, IL; and Coulter Counter, Model S-Plus System, Coulter Electronics, Inc., Hialeah, FL. Similarly, HBsAg was detected on instruments designed with samplers that required external washing. Compared with all other types of instruments surveyed in our laboratories, the continuous flow analyzers were the most vulnerable to malfunctions of

TABLE 2—Summary of HBsAg Environmental Sampling Data

Laboratory No.	No. Tested[a] %[b]	HBsAg Reactive: Initial Screen No.	Initial Screen %[b]	Confirmed No.	Confirmed %[b]
1	87	0	0.0	0	0.0
2	245	44	18.0	21	8.6
3	139	11	7.9	2	1.4
4	46	1	2.2	0	0.0
5	90	0	0.0	0	0.0
6	19	0	0.0	0	0.0
7	14	0	0.0	0	0.0
8	45	10	22.2	8	17.8
9	47	1	2.1	0	0.0
10	68	2	2.9	0	0.0
Total	800	69	8.6	31	3.9

[a]No. = Number.
[b]Percent of total tested.

TABLE 3—Comparison of Factors with HBV Contamination in Laboratory Work Stations Assigned to the High-Risk Category

Factor	HBsAg Survey: Number of Stations with HBsAg/Total	Odds Ratio and 95% Confidence Interval
Work load (sera/wk)		
≤150	3/24	
>150	8/15	8.00 (1.55, 31.19)
Behavior		
Appropriate	4/24	
Inappropriate	7/15	4.38 (0.97, 16.61)
Containment Measures		
Appropriate Safeguards	2/22	
Flawed Techniques	9/17	11.25 (1.84, 46.06)
Complexity (manipulations/task)		
<30	10/29	
≥30	1/10	0.21 (0.04, 1.92)

intricate serum-containing components. All of these design features were associated with an increased occurrence of contamination in our laboratories.

Instruments with discrete reaction containers and samplers with fluid sensors that do not require external wash steps minimize contamination. Visible soiling was not observed and HBsAg surveys were negative after operating the following instruments: Automatic Clinical Analyzer III: Dupont Instruments, Wilmington, DE; Automated STAT/Routine Analyzer-8: Beckman Instruments, Brea, CA: Immunochemistry Systems Analyzer II: Beckman Instruments, Brea, CA; and Cobas-BIO: Roche Analytic Instruments, Inc., Nutley, NJ.

Techniques which frequently resulted in blood spills, despite observations that workers adhered to written procedures, were considered defective because these methods did not reasonably ensure containment of biohazardous fluids. They were frequently associated with a need for repetitious separations and intermittent mixing of blood without adequate measures to contain spills. Both image and flow cytometric systems were identified as techniques with the most frequent occurrence of overt spills. These types of systems were associated with the presence of HBsAg in Laboratories 2, 3, and 8.

To approximate the risk of contamination when processing blood contained in vacuum-sealed tubes, samples were collected from a device designed for removing rubber stoppers from tubes. Operation of the device entails aligning the tube into an opening and, then, mechanically directing a metal arm to swing against the stopper forcing it to pop off simulating the manual technique. HBsAg was detected repeatedly on the rim of the device opening where spatter occurred. These data suggest that removing stoppers by either method is a subtle source of contamination. We endorse the technique of "wrap and snap" to minimize spatter of blood.

Tasks with few manipulations usually involved processing small volumes of blood using microscale techniques (both manual and automated). Automated instruments were used in 8 of 15 stations surveyed in Clinical Pathology compared with only 2 of 11 stations in Transfusion Medicine. Exclusive of the flow cytometry service, only manual techniques (e.g., immune profiles) were observed in the remaining research laboratories. Automated assays using less than 15 ml of blood averaged three manipulations per task: whereas, comparable manual assays averaged 10 manipulations per task. Tasks using greater than 1 L of blood required the most handling (average 30 manipulations/task: range from 6–92). These latter procedures were typically performed in research laboratories.

To examine the association between different tasks and contamination, we arbitrarily grouped each work station into those involving less than 30 manipulations and another group with 30 and more activities (complex tasks). In contrast to the factors mentioned earlier, complexity appeared to be inversely associated with contamination (OR = 0.21, 95% CI = 0.04, 1.92) (Table 3). A review of the data revealed that complexity was inversely correlated with work load (no complex tasks were performed in stations with high work load).

To further examine the predictors of blood-borne contamination, logistic regression analysis was performed to determine the effects of the factors shown on Table 3 while controlling for the effects of each other. Because of the negative correlation between complexity and high work load, we chose to remove complexity from further analysis. In the analysis with the three remaining factors, the point estimates for the adjusted odds ratio for flawed technique decreased to 9.78 (95 percent CI = 1.46, 65.49) and was the factor most strongly associated with HBsAg. Only a modest decrease in the adjusted odds ratios was observed for high work load (OR = 5.06, 95% CI = 0.80, 31.96) and inappropriate behavior (OR = 2.75, 95% CI = 0.44, 17.4) compared with the crude odds ratios. In a similar analysis with only two variables, both high work load (OR = 6.82, 95 percent CI = 1.73, 39.66) and flawed technique (OR = 9.78, 95 percent CI = 1.52, 63.01) were associated with an increased risk of blood-borne contamination. (Data available on request to authors.)

Discussion

Data from our study are consistent with the concept that workers who process blood from numerous donors (high work loads typically associated with diagnostic testing) increase their risk of occupational exposures to blood-borne pathogens. However, a high prevalence of HBV infection among blood donors can also contribute to risk. For example, the liver disease research unit had the highest HBsAg positivity rate (17.8 percent) but only averaged 27 specimens/week. One-third of the specimens submitted to this laboratory were from individuals known to be chronic carriers of HBV, compared with the other laboratories surveyed where fewer than one-tenth of the specimens were obtained from infected patients. It is important to note, however, that an analogous risk can be identified in any laboratory where serial specimens from infected patients are analyzed (e.g., therapeutic drug monitoring, diagnostic microbiology, and similar types of research involving HBV or HIV). In support of this observation is the recommendation that the function or nature of the laboratory should also be considered in the hazard assessment to protect these workers.[8]

Inappropriate behaviors (a second factor associated with HBsAg) were noted when workers disregarded standard practices or misused equipment. Somewhat alarming were observations that individuals at almost 50 percent of the high-risk areas experienced a seemingly innocuous, but preventable cutaneous contact with blood as a result of poor work practices. Complacency regarding the wearing of gloves while handling contaminated fomites was cited as the most frequent cause of these exposures.

During 75 hours of observation, only four overt accidents resulting in contact between the employees' protective garb and blood were observed. No accidental parenteral inoculations occurred and none of the four accidents was associated with inappropriate behaviors. These data can be contrasted with our observations cited above that poor work practices were associated with the employee experiencing cutaneous contact with blood. Based on these data, we concluded that the majority of exposures to blood that occur in the laboratory are occult in nature, as well as, preventable. Further, the risk of processing blood containing a pathogen is likely to be underestimated.[18] Despite the presence of warning labels on specimens submitted to NIH laboratories, during our observations, the majority of workers who engaged in poor work practices were not motivated by these labels to modify their inappropriate behaviors. Equally important was our observation that none of the individuals with good work practices experienced an adverse exposure to blood. In combination, these data reinforce the concept that workers who handle human blood strictly adhere to precau-

tions as an effective means to reduce their risk of laboratory-associated infection. These practices were observed prior to the implementation of universal precautions in our hospital.[12]

Our results indicate that automation contributed to contamination only when the design of certain instrument components was flawed with respect to containment.[19] Just as good engineering design can be negated by poor work practices, flawed design (the factor associated with the greatest risk of contamination) can also overcome good work practices.[9] Our study demonstrates the need to modify instrument components which represent an increased risk for contamination and, perhaps, infection among laboratory workers.

Manual techniques such as cytometric systems that involved sequential manipulations of specimens represented an increased risk for contamination in our laboratories. The frequent presence of HBsAg in these areas indicates that blood-borne contamination is predictable for many of these procedures. We concur that workers should modify these techniques in order to enhance their safety.[9]

In our study, the complexity of laboratory tasks (in terms of cumulative manipulations per task) was weakly associated with the risk for HBsAg. Also, we observed a negative correlation between complexity and high work load which probably reflected differences in the processing of specimens for research protocols compared to diagnostic testing. We submit, therefore, that the complexity of tasks performed represents minimal risk to the worker and recommend that predictors associated with greater risk of blood-borne contamination be given priority in the assessment of laboratory safety. Use of these data and previously published data[2,8,12] in designing interventions to eliminate adverse exposures to blood may prevent implementing strategies that unnecessarily disrupt the laboratory while not enhancing worker safety.

APPENDIX A

Method Used for HBsAg Confirmation Assay

Modifications to the manufacturer's instructions (Ausria II Confirmatory Neutralizing Kit: Abbott Laboratories, Chicago, IL) were as follows. The HBsAg positive control (Ausria II: Abbott Laboratories, Chicago, IL) was processed in the same manner as the samples. After the addition of a polystyrene bead coated with guinea pig anti-HBsAg, 150 µl of eluate or HBsAg positive control were dispensed to each of four wells. The negative control (FBS/PBS) was assayed in replicates of six using 150 µl per well. After the initial wash step, 150 µl of FBS/PBS (diluted 1:5 with 0.9% NaCl) were added to the negative control wells. Each tray was incubated (25°C for 16–24 h). Each well was then washed four times using 5 ml distilled water per rinse. A 150 µl volume of confirmatory anti-HBsAg (diluted 1:5 with 0.9% NaCl) were added to two of the four wells and 150 µl of FBS/PBS (diluted 1:5 with 0.9% NaCl) were added to the remaining two wells of each set of assays and to each of the negative control wells. Each tray was incubated (45°C for 1 h). Without aspirating the contents of the well, 200 µl of I-125 anti-HBsAg were then added to all of the wells and allowed to react (45°C for 1 h). After repeating the wash step, each bead was counted.

The average cpm was calculated for each replicate assay that reacted with either anti-HBsAg or FBS/PBS. The percent reduction in the cpm for the positive control and each sample was calculated using the following formula:

$$\% \text{ Reduction} = \frac{\{\text{cpm(sample + FBS/PBS)} - \text{cpm(sample + anti-HBsAg)}\} \times 100}{\{\text{cpm(sample + FBS/PBS)} - \text{cpm(negative control)}\}}$$

Validity of a confirmation run was based on two arbitrary criteria: 1) neutralization with anti-HBsAg had to result in a greater than 50 percent reduction in the cpm for the positive control when compared to the cpm of the control reacted with I-125 anti-HBsAg (the parameter was >97 percent for each run); and 2) no more than two replicates of the negative control could be considered aberrant. This latter event did not occur. However a single value was greater than 1.5 times the mean of the negative control for three of the five runs. In each case, the aberrant value was excluded and the mean recalculated.

ACKNOWLEDGMENTS

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Recovery of Hepatitis A Virus from a Water Supply Responsible for a Common Source Outbreak of Hepatitis A

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Abstract: An outbreak of hepatitis A occurred in a north Georgia trailer park served by a private well. Of 18 residents who were serosusceptible to hepatitis A virus (HAV), 16 (89%) developed hepatitis A. Well water samples were collected 3 months after illness onset in the index case and 28 days after illness onset in the last trailer park resident. Hepatitis A virus antigen (HAVAg) was detected in the samples by enzyme immunoassay from three of the five cell lines following two 30-day passages and from a fourth cell line following a third passage of 21 days. (*Am J Public Health* 1990; 80:428–430.)

Introduction

Infection with hepatitis A virus (HAV) accounted for 39 percent of the 59,633 cases of acute viral hepatitis reported in the United States in 1985.[1] Virus transmission occurs primarily through the fecal-oral route, resulting in both sporadic cases and common source outbreaks.[2] Common source outbreaks of hepatitis A have been associated with ingestion of contaminated food[3,4] or consumption of inadequately treated drinking water.[5–9] The incrimination of food and water in HAV transmission has largely been based on the epidemiologic characteristics of the outbreak and serologic evidence that the resulting viral hepatitis was type A. Only recently has HAV been recovered from water associated with hepatitis A outbreaks.[10]

HAV is classified as a picornavirus, and most wild-type HAV does not produce a cytopathic effect, although infectious units can be quantitated by the radioimmunofocus assay (RIFA).[11] We describe the cell culture isolation of HAV from drinking water implicated as the source of virus transmission in a common source outbreak.

Methods

Epidemiologic Investigation

A 21-year-old man had onset of serologically confirmed hepatitis A on July 10, 1982 while living from July 9 through July 23 in a trailer park in Bartow County, Georgia. Approximately three to nine weeks following onset of illness, other residents of the trailer park developed hepatitis A. All cases of hepatitis A during 1982 were identified using county health department case reports and laboratory records for hepatitis A serologic testing. Each trailer park resident was interviewed and a blood sample obtained. All serum samples were tested by radioimmunoassay for total and IgM-specific antibody to HAV (HAVAB and HAVAB-M, Abbott Laboratories,* North Chicago, IL).

Case Definition

A case was defined by a positive serologic test for IgM anti-HAV. One trailer park resident who declined a blood test but reported a history of jaundice and abdominal pain was also included.

Water Sampling

Tap water from the trailers was tested for fecal coliform bacteria by standard methods.[12] Fluorescein dye was discharged from the toilet of the index case, and tap water samples were collected after 19 and 81 days.

Three months after onset of illness in the index case, 400 and 1600 liters of ground water from the well pump supplying the park were passed through an electropositive charge-modified, 25-cm long, cellulose-diatomaceous earth composite, microporous filter (Virosorb 1MDS; Cuno, Meriden, CT).

Cell Cultures

Adsorbed viruses were eluted from the filter cartridges with 1 liter of 3 percent beef extract adjusted to pH 10, and after elution, sufficient 1M glycine was added to lower the pH to 3.5. The flocculated organic material was processed according to the method of Katzenelson, *et al.*[13] Virus culture was carried out in four monkey kidney cell lines: BGM, BS-C-1, FRhK-4 (American Type Culture Collection, Rockville, MD) and MA104 (Microbiological Associates, Bethesda, MD); and in primary Rhesus monkey kidney (PMK) cells.

The concentrated sample was filter sterilized directly onto the cell monolayer, and virus was adsorbed at 37°C for two hours with periodic rocking. PBS mock-infected cell cultures were included for controls. All cells except FRhK-4 were grown in Eagle's minimum essential medium containing Earle's balanced salts solution and supplemented with 10 percent fetal bovine serum, 25 mM HEPES (N-2-hydroxyethylpiperazine-N'-2-ethanesulfonic acid), 2 mM L-glutamine, 0.1 mM nonessential amino acids, 0.2 percent sodium bicarbonate, and antibiotics (20 mcg/ml gentamycin, 100 mcg/ml streptomycin, and 100 U/ml penicillin). FRhK-4 cells were grown in William's medium E as previously described.[14] Maintenance media consisted of the same basal medium

Address reprint requests to J. David Smith, BS, Office of Epidemiology, Georgia Department of Human Resources, 878 Peachtree Street, Atlanta, GA 30309. Three co-authors are also with that office (Bloch, McKinley, and Sikes); others are with the Hepatitis Branch, Division of Viral Diseases, Center for Infectious Diseases, CDC, Atlanta (Stramer, Margolis, Fields, Maynard); and two are affiliated with the Department of Microbiology and Immunology, and Department of Nutrition and Food Science, University of Arizona, Tucson (Stramer and Gerba). This paper, submitted to the Journal December 2, 1988, was revised and accepted for publication September 28, 1989.

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*Use of trade names is for identification purposes only and does not imply endorsement by the Public Health Service, US Department of Health and Human Services, or the Georgia Department of Human Resources.

containing 2% fetal bovine serum. All media and media components were obtained from Gibco Laboratories (Grand Island, NY).

Continuous cell lines were passaged twice at 30-day intervals and four times at 21-day intervals. Virus was recovered from PMK cells by trypsinizing, freeze-thawing, sonication, extraction with 2,3,5 trichloro-trifluoroethane (Dupont Chemicals, Wilmington, DE), and passaged acutely.

Assays for HAV

Trypsinized cell samples were extracted as described above and assayed for HAVAg with a double-antibody sandwich enzyme immunoassay (EIA), as previously described.[15] A ratio of positive to negative values (P/N) >2.1 was considered positive for HAVAg.

Infectious units of HAV were quantitated by the RIFA, using both human and rabbit anti-HAV.[11] HAV titers were expressed as radioimmunofocus-units (RFU) per ml.

Results

Epidemiologic Investigation

Thirty-five cases of hepatitis A with onset of illness from July 10 to November 24, 1982 were identified in the county. Before July 10, there had been no reported hepatitis A cases in the county during 1982. Of the 35 cases, 17 were residents of the trailer park. In 31 cases the date of onset of symptoms could be recalled (Figure 1). Among the 13 residents of the trailer park who could recall date of onset of illness, 12 became ill within 22 to 41 days following disease onset in the index case.

The index case was a 21-year-old male who lived with a family in the trailer park from July 9 to July 23. He denied contact with the seven other families in the park. Of the 30 trailer park residents, seven had serologic evidence of prior exposure to hepatitis A. The five persons living with the index case received immunoglobulin prophylaxis, and one became IgM anti-HAV positive. Among the 18 remaining trailer park residents considered serosusceptible to hepatitis A, 16 developed hepatitis A (89 percent attack rate). The epidemiologic investigation revealed no common social activity, meal, or food which could account for the outbreak. The only two serosusceptible individuals who did not become ill were 11 and 18 months of age, and their mothers were positive for total anti-HAV but negative for IgM anti-HAV.

Of the 18 cases who were not residents of the trailer park (including the index case), 11 had visited the trailer park and drunk water during their visit (Figure 1). Another four patients had contact with cases only outside the trailer park, while three had no known contact with other cases.

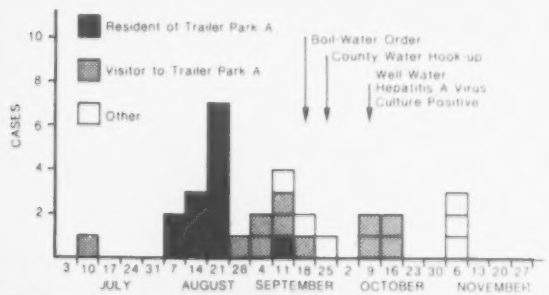

FIGURE 1—Onset of Reported Hepatitis A Cases with Known Date of Onset in Bartow County, Georgia, July through November 1982. (A county order to boil water was issued September 17. The trailer park was connected to the county drinking water supply on September 24. The hepatitis A virus-containing water samples were collected from the implicated well on October 6.)

Environmental Investigation

The trailer park's sole water supply was a 15-cm diameter drilled well having a depth of 24 to 30 m, encased within steel pipe. The trailers encircled the well at a distance of 18 to 60 m, and water was distributed through a 2-cm galvanized pipe buried approximately 50 cm below the ground surface. Wastewater from each trailer was treated by individual septic tank systems having gravity feed, subsurface drainage tile distribution networks; each septic tank system was separated from the well by a distance of 30 to 60 m.

The concentration of fecal coliforms in tap water ranged from zero to greater than 16 per 100 ml and in water collected from the wellhead was 2 per 100 ml. Fluorescein dye was flushed from the toilet of the trailer in which the index case had lived, but could not be detected on days 19 and 81 after charging the system.

A boil-water order was issued on September 17, and on September 24 the well was disconnected, and the water distribution system was connected to the county water supply.

Virus Assays

HAVAg could not be detected in the ground water concentrates by EIA. Following amplification in cell culture, HAVAg was detected in the monkey kidney cell lines (Table 1). After primary cell adaptation to FRhK-4 and PMK cells, foci of HAV were detected by RIFA (Figure 2). Passage in FRhK-4 and PMK cells yielded titers of 3.0×10^7 RFU/ml and 2.0×10^7 RFU/ml, respectively.

Discussion

The importance of HAV transmission by water is probably underestimated since outbreaks appear to be infrequently reported, poorly documented, and the virus has been only rarely isolated from this source.[9,10,16] Using currently available methods for enterovirus detection, HAV can be concentrated 100- to 10,000-fold onto microporous filters and concentrated another 10- to 100-fold by organic flocculation.[10,16,17] In one study in which these concentration techniques were used, samples of ground water implicated in a large outbreak of hepatitis A revealed low levels of HAVAg by RIA.[16] In contrast to a previously reported outbreak where HAV was isolated from contaminated water,[10] the fecal coliform counts obtained at the peak of the presumed period of contamination in the present outbreak were 10- to 100-fold lower, suggesting

TABLE 1—Detection by Enzyme Immunoassay of Hepatitis A Virus Recovered from Ground Water following Growth in Monkey Kidney Cells. (Ground water samples were collected October 6, 1982 from a well in a trailer park in Bartow County, Georgia.)

Monkey Kidney Cell Line	Hepatitis A Virus Antigen by Enzyme Immunoassay[a] by Passage Level					
Passage:	1[b]	2	3[c]	4	5	6
MA104	<1	<1	<1	<1	<1	<1
BGM	<1	1.1	2.4	3.6	6.9	21.0
BS-C-1	<1	2.9	8.5	11.0	21.0	>115.0
PMK	1.5	3.2	8.5	11.0	41.3	>115.0
FRhK-4	<1	2.4	5.6	10.6	57.0	160.0

[a]HAVAg expressed as positive/negative (P/N) ratios in undiluted cell lysates
[b]Passages 1 and 2 at 30 days
[c]Passages 3–6 at 21 days

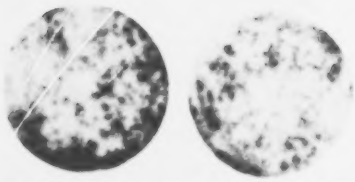

FIGURE 2—Foci Produced in BS-C-1 Cells in the RIFA from HAV Isolated from Concentrated Ground Water in Bartow County, Georgia following Initial Isolation in FRhK-4 Cells. (Panels from left to right represent increasing dilutions of the recovered HAV.)

that low levels of ground water contamination can produce significant outbreaks of hepatitis A.

This outbreak also demonstrated the stability of HAV in untreated water. The water samples from which the virus was isolated were obtained three months after the onset of symptoms in the index case and 28 days after the onset of symptoms in the last trailer park resident (Figure 1). The recovery of HAV suggests that the contaminating virus had continued access and persisted in the aquifer supplying the well although it cannot be ruled out that fourth generation cases (illness onsets from October 4 to 13) who were visitors to the trailer park, may have recontaminated the aquifer. In addition, the water concentrates were stored at 4°C for eight months before they were inoculated into cell culture.

While the incidence of HAV infection has been decreasing in the US over the past 10 years, it continues to be the second leading cause of viral hepatitis. Although the majority of the population utilizes municipal drinking water, it is evident that well water contamination with HAV continues to occur and that this source of infection is not widely appreciated.

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Review Criteria for Schools of Public Health Funding, Project Grants

Final review criteria, funding priority, and special consideration for special project grants to Schools of Public Health were published in the February 8 issue of the *Federal Register*. Among the review criteria to be considered is whether the project contains realistic objectives as described in *The Future of Public Health, Objectives for the Nation* and the *Sixth Report to the President and Congress on the Status of Health Personnel in the United States*.

The funding priority is designed to promote health initiatives that will encourage and increase involvement of underrepresented minority health trainees. Special consideration will be given to applications that address five issues:

- Substance Abuse
- HIV/AIDS
- Geriatrics
- Environmental health
- Training students to work in public health agencies

For further information, contact Blake Crawford, HRSA, US Public Health Service, DHHS, 5600 Fishers Lane, Room 14-43, Rockville, MD 20857; (301) 443-3376.

A Rural Outbreak of Legionnaires' Disease Linked to Visiting a Retail Store

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Abstract: Between May 7 and June 7, 1986, 27 residents of a rural county in Maryland developed legionellosis, and two died. *Legionella pneumophila* serogroup 1 was cultured from the sputum of two patients and identified in lung tissue of a third patient by direct fluorescent antibody staining. An additional 11 patients had four-fold rises in antibody titer to *L. pneumophila*, and 13 had single titers ≥1:256. To determine risk factors for disease, we performed a case-control study. Twelve of 16 case-patients reported visiting store A in the two weeks before onset of illness compared with four of 28 control-patients. A serologic survey of employees showed that employees of store A were 3.63 times more likely than control employees to have titers of antibody to *L. pneumophila* ≥1:256 (95% confidence intervals 0.8, 16.7). Cultures of soil specimens, samples of water from the hot water system of store A and from stagnant ponds near store A collected five weeks after the end of the outbreak were negative for *Legionella* species. Store A was adjacent to a site of excavation and construction during May 1986, when the community was experiencing an extended drought. This investigation suggests that exposure to excavation and construction activity may be a risk factor for legionellosis. (*Am J Public Health* 1990; 80:431–434.)

Introduction

Investigators agree that *Legionella pneumophila* is ubiquitous in nature, but they disagree about the importance of various modes of transmission.[1] Attention has focused on inhalation of organisms from water droplets in aerosols and aspiration of contaminated potable water as predominant modes of transmission. These conventional assumptions of how legionellae are transmitted are inadequate to explain outbreaks associated with construction and excavation when aerosols or potable water sources are not implicated. In this report we describe a community outbreak of legionellosis that was not associated with an aerosol or contaminated potable water. Exposures to excavation and construction were the only identifiable risk factors.

During the third week of May 1986, the staff radiologist of a community-hospital in a rural county of Maryland recognized an unusual cluster of cases of severe community-acquired pneumonia. The Maryland State Health Department and local county health department were notified and an investigation was initiated. Review of chest radiograph reports confirmed an increase in the number of both hospitalized patients and outpatients with radiographic evidence of pneumonia in May 1986 compared with April 1986, April 1985, and May 1985. Cases identified by this review had a wide range of clinical findings, and initial microbiologic studies did not implicate any single pathogen.

Methods

We defined cases of outbreak-associated Legionnaires' disease as follows: onset of symptoms consistent with pneumonia during May 1986, residence in county A, and either isolation of *L. pneumophila* serogroup 1 from a clinical specimen, fluorescent antibody staining of organisms from tissue, four-fold rise in antibody titer to *L. pneumophila* serogroup 1 to a titer ≥128, or a single antibody titer ≥256.

Cases were ascertained by reviewing all reports of chest radiographs performed on inpatients and outpatients during May and June 1986 at the only hospital in the county, clinical histories recorded in charts of patients reported to have pneumonia, and the reports by community physicians of patients with pneumonia. Convalescent-phase serum specimens were obtained on 52 suspect outbreak patients with pneumonia; for 41 patients an acute-phase serum specimen was also obtained.

Isolates of *L. pneumophila* obtained from patients were subtyped by the pattern of binding of a panel of monoclonal antibodies to *L. pneumophila*.[2]

To determine risk factors associated with illness, we performed a case-control study. Since laboratory data were unavailable at the time the case-control study was performed, we used a clinical case definition—temperature ≥101°F, white blood cell count ≥11,000 cells/mm^3, and dense pulmonary infiltrate by chest radiograph. A questionnaire was administered to each case-patient by phoned interview that lasted approximately 10 minutes. We asked whether the patient had visited any of eight specific locations in county A in the two weeks before illness onset and if so, on what date and at what time; whether the patient would have visited these locations during a usual two-week period; underlying medical problems; employment history during the two weeks before illness onset; and use of home air conditioning. At the end of the interview, case-patients were asked to identify two friends of the same sex as themselves and within 10 years of age to serve as control-subjects. Control-subjects were then telephoned, and the same questionnaire was administered. Risk factors were analyzed in a univariate fashion using the Pike-Morrow method for a variable number of controls per case without breaking the match.[3]

Results of the case-control study were used to focus subsequent investigation. Employees working within one-half mile of the retail store associated with cases of Legionnaires' disease, including the store itself, were asked to complete a questionnaire that requested information about recent illnesses and days missed from work. Serum speci-

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mens were obtained from employees working in the implicated area and from control employees of similar age working at a similar retail store located 15 miles from the implicated area who volunteered to be bled. Antibody titers to *L. pneumophila* serogroup 1 were determined by standard methods.[4] Titers were compared by Fishers' exact test.[5]

Water samples were obtained from potential reservoirs of *Legionella* and cultured according to established protocols.[6] Methods used included filter concentration, acid treatment, and use of antimicrobial containing media.Soil samples were also obtained, hydrated in sterile water, and cultured by the same methods.

Records of precipitation and wind direction collected 3.7 miles from the implicated area were obtained from the National Oceanographic and Atmospheric Administration (Asheville, North Carolina), and the environment of the area associated with acquisition of Legionnaires' disease was inspected to determine whether any aerosol generating devices could be identified.

Results

Twenty-seven residents of county A developed Legionnaires' disease during May 1986, and two died. The dates of the onset of symptoms ranged from May 11 to May 28 (Figure 1). *L. pneumophila* serogroup 1 was isolated from two patients' sputa; lung tissue from a third patient, obtained post mortem, gave a positive direct fluorescent antibody stain for

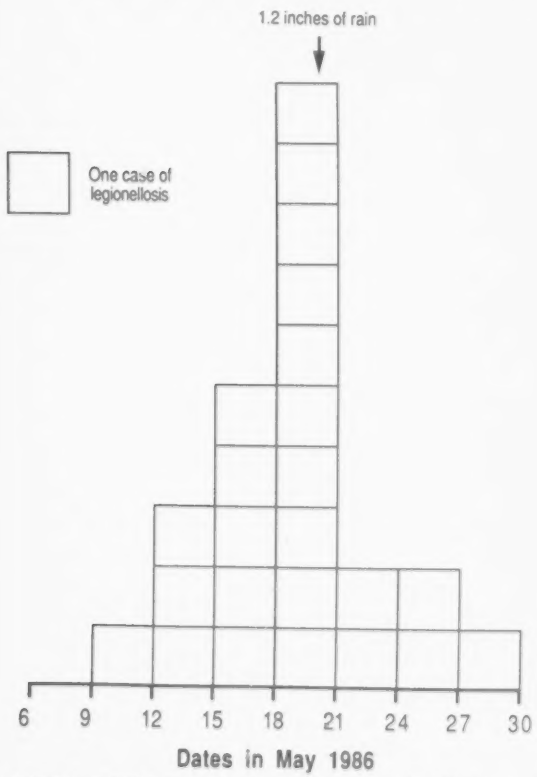

FIGURE 1—Date of Symptom Onset of Cases of Legionnaires' Disease (onset date unknown for 3 persons)

L. pneumophila serogroup 1; 11 patients had four-fold rises in reciprocal antibody titer to *L. pneumophila* serogroup 1, and 13 had reciprocal titers ≥256 (four patients had reciprocal titers of 256). The two isolates of *L. pneumophila* from patients with Legionnaires' disease gave the same monoclonal antibody binding pattern, binding to monoclonal antibodies 1, 2, 5, 7. Patients with Legionnaires' disease ranged in age from 27 to 80 years (median 51). Age-specific attack rates of disease, using 1982 estimates of the county's population, showed that although many young adults were affected, attack rates tended to increase with age (Table 1). Only 17.6 percent of the cases were in women. The locations of patients' residences were spread over a large geographic area, virtually the entire county, without clustering of cases by census tract (Figure 2).

In the case-control study, 16 patients with Legionnaires' disease and 28 controls were interviewed. Of the 27 cases of Legionnaires' disease, 10 did not meet the clinical criteria required for enrollment in the case-control study, and one could not be located. Twelve cases and four controls had visited store A in the two weeks before illness onset (lower bound 95% confidence interval 3.42). No other potential risk factor was significantly more common for the cases.

Store A is a large single room retail store employing approximately 100 employees situated in a sparsely populated area of County A with few nearby residences but some recent commercial development. Several hundred customers visit store A per day. Approximately 100 yards south of Store A, excavation and construction of a shopping mall took place during the month of the epidemic. No cooling towers or wet-heat rejection sytems were present within one-half mile of store A.

The survey of Store A employees revealed that one of 90 had had pneumonia during May. This individual had a reciprocal antibody titer to *L. pneumophila* serogroup 1 of 256 five weeks following the onset of his illness. Greater prevalence of detectable antibody to *Legionella* and higher titers were seen in Store A employees than in persons working in store B and near Store A (Table 2). Store A employees were 3.63 times as likely to have reciprocal titers of 256 or more (95% CI = 0.8, 16.7). Because of the delay between the survey and the end of the outbreak, it was not possible to obtain serum specimens or interviews from persons working at the construction site during May. No *Legionella spp.* were isolated from any of the 35 water and soil samples. The free chlorine level in potable water specimens was 1.0 parts per million.

Only 0.43 inches of rainfall were recorded for County A from May 1 through May 19, compared with a normal rainfall of 2.10 inches. At the peak of the outbreak, 1.2 inches of rainfall were recorded (Figure 1). The predominant wind direction during May (resultant wind vector) was from 241°,

TABLE 1—Attack Rates of Legionellosis in County A by Age*

Age Group (years)	No. of Cases	Attack Rate (per 10,000)
25–34	4	3.1
35–44	7	5.8
45–54	6	8.4
55–64	2	4.1
65–74	4	13.9
75–84	1	8.1

*Age unknown for 3 patients.

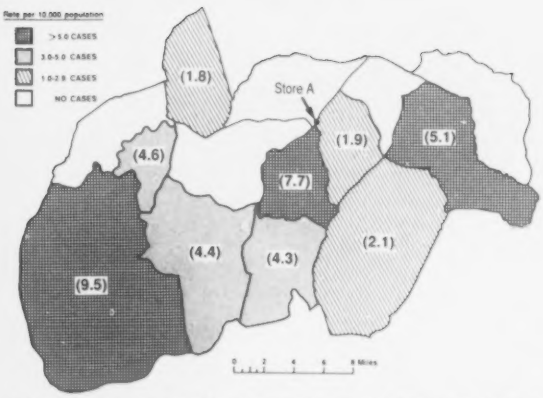

FIGURE 2—Census Tract of Residence for Persons with Legionnaires' Disease in County A, in Maryland, 1986 (numbers in parentheses indicate attack rate per 10,000 population; population per census tract ranged from 2,000 and 15,000)

TABLE 2—Antibody Titers to *Legionella pneumophila* among Employees of Selected Businesses

	Numbers of Employees with Indicated Titers					
Site of Employment	≤64	128	256	512	≥1024	Total
Store A	40	5	3	2	2	52
Store B	14	2	0	1	0	17
Near store A	33	3	1	0	0	37

from the southwest and toward store A from the construction site. Persons working in store A recalled extremely dusty conditions during May 1986, and review of the maintenance records for the ventilation system of store A revealed that frequent changes of the filters for the air intake had been required during the first three weeks of May. Store A was cooled with a freon-based, dry central air conditioning unit; there was no humidification of incoming air.

Discussion

Investigation of this Legionnaires' disease outbreak was complicated by logistic problems in obtaining and testing serum specimens that delayed laboratory confirmation of the diagnosis. We are unaware of previous Legionnaires' disease outbreaks occurring in rural areas despite serologic evidence that exposure is no less frequent in such areas.[7] Legionnaires' disease outbreaks in rural settings may be more difficult to identify because diagnostic facilities for legionellosis are frequently not available. Even in the context of epidemiologic study,[8] many patients with community-acquired pneumonia do not receive a precise etiologic diagnosis. Using review of radiograph reports to identify cases created additional difficulties in our investigation because of the low specificity of this method, unless combined with fever and white blood cell count.

Once Legionnaires' disease was identified as the cause of the outbreak, additional epidemiologic investigation demonstrated an association between visiting store A and illness. Results of the case-control study led to additional surveys to confirm the association of visiting store A and developing legionellosis. The attack rate of 1.5 percent in Store A employees is consistent with attack rates in other epidemics of Legionnaires' disease.[9,10] In addition, elevated antibody titers against *Legionella* were more common and these levels tended to be higher in employees of store A than in control populations.

Negative cultures of all environmental specimens, including water and soil samples, were not entirely unexpected since the specimens were obtained five weeks after the onset of the last case and the end of the outbreak. Since no evironmental legionellae were recovered, no microbiologic proof of the source of *Legionella* organisms was found in this outbreak. The absence of cooling towers at store A and the absence of exposure of case-patients to aerosols of potable water suggest that persons contracting Legionnaires' disease were exposed to some other source of *Legionnella* organisms.

Excavation and construction have been associated with outbreaks[10–12] and sporadic cases[13] of Legionnaires' disease, and excavation and construction of a shopping mall near store A occurred during the month of the epidemic. Although most outbreak investigations in which excavation and construction were associated with disease occurred before methods for recovering *Legionella* were available, in one nosocomial outbreak, *L. bozemanii* was recovered from potable water and from soil at a nearby excavation. *Legionella* was hypothesized to have colonized the potable water system at the time pipe connections linked the new building to the old.[11] Thus, in some outbreaks the link between excavation, construction, and Legionnaires' disease may be potable water.

In the current outbreak investigation cultures of potable water, although obtained five weeks after the end of the outbreak, were negative. High free-chlorine concentrations were found in the municipal water serving store A, so we doubt that *Legionella* could have survived in the potable water. Wind direction and the clogged air filters at store A suggest that airborne particles were blown from the construction site toward store A. Rains occurring on May 20, virtually the first rain of the month, coincided with a cessation of new cases after May 28, within the incubation period of Legionnaires' disease.

This temporal association is similar to that seen at the St. Elizabeth's Hospital outbreak in Washington, DC.[10] In that outbreak, attack rates of legionellosis for patients hospitalized in buildings near excavation were higher than the rates for patients not hospitalized near excavation. Because the etiologic agent was not discovered and methods to culture *Legionella* from the environment were not developed, the reservoir of *Legionella* was not determined. The tight temporal relationship between the onset of illness and excavation suggests that direct exposure to excavation was related to developing legionellosis.

Although legionellae have never been recovered from dry soil, they have been recovered from mud and wet soil.[11, 14–16] Since *Legionella* may be killed by heating or drying,[17] the hypothesis that legionellosis can occur after exposure to dry soil requires the assumption that *Legionella* may exist in an environment that provides protection from heating and drying. Additional environmental reservoirs for *Legionella* may be found in future studies, as were recently identified for *Blastomyces dermatitidis*.[18] The temporal relationship of the rains of May 20, and the cessation of new cases suggest that the rains halted the further transmission of *Legionella*.

This outbreak of Legionnaires' disease was associated with exposure to a retail store near a site of excavation and construction at a time when dry weather conditions prevailed. These results further suggest that, in some outbreaks,

exposure to construction and excavation may be a risk factor for Legionnaires' disease. Future investigations of Legionnaires' disease outbreaks associated with construction and excavation must include expanded searches for environmental legionellae and consideration of novel reservoirs.

ACKNOWLEDGMENTS

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Dr. Robert S. Lawrence Named Editor of *American Journal of Preventive Medicine*

Robert S. Lawrence, MD, has been named editor of the *American Journal of Preventive Medicine*, a bimonthly publication sponsored by the American College of Preventive Medicine (ACPM) and the Association of Teachers of Preventive Medicine. Dr. Lawrence is Charles S. Davidson Associate Professor at Harvard Medical School and Chief of Medicine at Cambridge Hospital.

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A graduate of Harvard College and Harvard Medical School, Dr. Lawrence has also served in the Epidemic Intelligence Service (EIS) of the Centers for Disease Control. On the international level, he has worked as a medical epidemiologist in El Salvador, as coordinator of the Harvard-King Faisal University Project in Saudi Arabia, and as a consultant or visiting professor in Italy, Japan, and Qatar.

Dr. Lawrence serves on many national committees and has been especially concerned with human rights. He is author of various articles, book chapters and books with an emphasis on primary care, disease prevention, health promotion, and medical education.

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Tuberculin Skin Testing in US Navy and Marine Corps Personnel and Recruits, 1980–86

ELEANOR R. CROSS, MS, AND KENNETH C. HYAMS, MD

Abstract: An extensive skin testing program is part of the United States Naval Medical Command's infectious disease control effort. From 1980 to 1986, 2,306,533 skin tests, using five TU PPD, were performed on active-duty Navy and Marine Corps personnel, 0.97 percent of which were positive. A downward trend in positive tests was found with a high of 1.43 percent in 1980 and a low of 0.80 percent in 1983. Since 1984, the percentage of positive tests has remained the same or increased. Shore-based medical facilities around the world reported 1,491,646 skin tests with 1.07 percent positive; Navy ships reported 814,887 skin tests with 0.78 percent positive. PPD-positivity for ships in the Pacific area was higher (0.98 percent) than for ships in the Atlantic (0.62 percent). During this same period, the percentage of positive tests in Navy and Marine Corps recruits ranged from a high of 1.82 percent in 1981 to a low of 1.23 percent in 1986. Since 1984, the percentage of positive tests has remained relatively stable in recruits. The frequency of positive PPD tests found in this study is lower than the percentage positive (1.59 percent) found in active-duty Navy personnel in 1969 and the percentage positive (5.2 percent) found in a study of Navy and Marine Corps recruits between 1958 and 1969. (*Am J Public Health* 1990; 80:435–438.)

Introduction

Since the early 1800s, the Medical Department of the US Navy has recognized the high transmission potential of tuberculosis.[1] Not only is there an increase in risk of exposure from world-wide travel but, as epidemiological studies have demonstrated, environmental conditions aboard ships are ideal for transmission.[2–4] Early attempts at TB control included screening sailors prior to an extended cruise and rejecting "all persons who were believed to be of unsound constitution."[1]

Today, the Navy's tuberculosis control program includes tuberculin skin test screening, contact investigation, and treatment of active cases and selected skin test reactors.[5] The Naval Medical Command directs all personnel whose previous test was negative to be periodically skin tested by the Mantoux method using 5 tuberculin units (TU) Purified Protein Derivative (PPD).[5] Annual screening is done on individuals assigned to operational units and to areas where possible exposure to tuberculosis is high, i.e., members of deployable Navy and Marine Corps units, shipboard personnel, and health care personnel. Sailors in low-risk areas, such as shore-based personnel in the United States, are tested every three years.

A person with induration measuring 10 mm or greater 48 to 72 hours after the administration of a 5 TU skin test is considered a "tuberculin reactor."[5] A new reactor is defined as "a person who has become a reactor within the past three years," and persons who are found to be reactors for the first time but whose prior tuberculin status is unknown. Since earlier studies indicated that a new reactor rate of 1 to 2 percent of personnel tested could be expected in many Navy and Marine Corps settings, the Naval Medical Command recommends a search for active cases of tuberculosis when the rate of new reactors exceeds 2.5 percent in groups of approximately 100 or more personnel.[5]

This report examines tuberculosis skin testing in the Navy from 1980 to 1986. Comparisons are made over time in and recruits Navy and Marine Corps active-duty personnel stationed in various geographic locations.

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Methods

Each month, all outpatient facilities of the Naval Medical Command report the number of PPD tests performed and the number of reactors. Information on age, sex, race, and the exact size of the reaction is not recorded, however. In this study, computerized files of skin test results for the period January 1980 to December 1986 were analyzed.

Reports are received each month from approximately 600 shore-based facilities and from over 500 ships. Approximately 25 percent of the shore-based facilities are located outside of the United States. It is important to note that the location of the reporting facility may not necessarily be the location of the tuberculosis contact. Data from shore-based facilities may include skin test results on individuals from ships during port visits as well as results on individuals transferred to a facility from a different location.

Although these tests are administered and read by a number of different medical personnel, the Naval Medical Command provides very specific instructions on the administration and reading of these tests.[5] Therefore, the overall results of the skin tests from different locations are generally comparable. Tubersol (Connaught, Inc., Swiftwater, PA) was used for the skin testing during the period of this study.

For the 1980 to 1986 study period, the total number of skin tests performed in active-duty personnel fluctuated from a high of 373,747 in 1983 to a low of 287,251 in 1986. Average annual active-duty strength increased from 720,832 in 1980 to 805,128 in 1986. Consequently, approximately 40–50 percent of the total force was tested annually. The mean age of active-duty personnel increased from age 25.0 years in 1980 to age 26.1 years in 1986. The mean sex ratio of Navy and Marine Corps personnel changed from 16:1 (male:female) in 1980 to 11:1 in 1986.

For recruit personnel, approximately 83 percent were between the ages 17 to 21 years during the study period. The percentage of both Black and Hispanic recruits increased during the period 1981 to 1986 from 12 percent to 17 percent for Blacks and from 4 percent to 9 percent for Hispanics.

Results

Between 1980 and 1986, a total of 2,306,533 tests were read for all active-duty Navy and Marine Corps personnel, 0.97 percent of which were positive. The rate of positive tests decreased from a high of 1.43 percent in 1980 to a low of 0.80

percent in 1983. In 1983, this downward trend changed, and from 1984 to 1986 the percentage of positive tests either remained the same or increased (Table 1, Figure 1).

Shore-based Facilities

In shore-based facilities located inside of the continental United States (CONUS), a total of 1,319,610 tuberculin skin tests were read, of which 1.00 percent were positive. The percentage of positive tests dropped from a high of 1.41 percent in 1980 to a low of 0.83 percent in 1983. After 1983, the rate of positive tests either rose or remained the same (Figure 2). The average percentage of positive tests in shore-based facilities located outside the continental United States (OCONUS) was higher (1.61 percent but followed a very similar trend (Figure 2).

By location of shore-based facilities, the highest frequency of positive tests was in the Philippines (3.10 percent), followed by Japan (2.90 percent), Korea (2.00 percent), Guam (1.88 percent), Bahrain (1.66 percent), and Okinawa (1.40 percent). By US Naval District, skin test positivity was highest in the Northwestern United States (1.79 percent) and Northern California (1.77 percent) (Table 1, Figure 3). The lowest rates were found in the North Central area (0.46 percent).

Positive TB Skin Tests Aboard Ships

Of 814,887 skin tests read aboard ships, 0.78 percent were positive. The positive rate was higher in ships based in the Pacific area (0.98 percent) than for ships based in the Atlantic (0.62 percent). As in shore-based facilities, there was a general downward trend in both areas from 1980 to 1982, especially in the Pacific. After 1982, the rates of positive tests remained fairly stable (Table 1, Figure 4).

Navy and Marine Corps Recruits

Between 1980 and 1986, a total of 618,074 skin tests were performed on recruits, 1.45 percent of which were positive (Table 1, Figure 5). The highest percentage of positive tests (1.82 percent) was found in 1981. Since 1984, the percentage of recruits with a positive PPD skin test has remained relatively stable at between 1.28 and 1.23 percent.

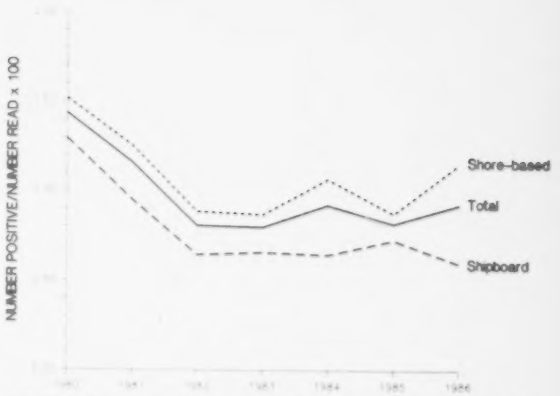

FIGURE 1—Frequency of Positive Tuberculin Tests in all Active-duty US Naval and Marine Corps Personnel, in all Shore-based Naval Medical Facilities, and in all ships, 1980–86

Discussion

The results of this study show a downward trend in the frequency of positive TB skin tests from 1980 to 1983. After 1983, the percentage of positive tests either remained stable or increased. It is noteworthy that these general trends occurred in all population groups of active-duty US Navy and Marine Corps personnel. The reason why different populations tested in different areas of the world exhibited such closely similar patterns of positive tests could not be determined. The data indicate, however, that the threat of TB infection is greatest in the Pacific for both shore-based and shipboard military personnel.

TABLE 1—Percent Positive Tuberculin Reactors in Active-duty US Navy and Marine Corps Personnel and in Recruits, 1980–86

Location	Number Reactors/Number Read × 100 (Reactors/Read)							
	Average 1980–86	1980	1981	1982	1983	1984	1985	1986
Total	0.97 (22388/2308633)	1.43 (4385/305837)	1.16 (3988/318616)	0.81 (2604/323488)	0.80 (2978/373747)	0.92 (3263/356926)	0.82 (2794/340588)	0.92 (2645/287351)
Shore-based								
Total	1.07 (16020/1491646)	1.52 (2908/191658)	1.25 (2750/219710)	0.88 (1927/218103)	0.87 (2158/248741)	1.07 (2481/233717)	0.87 (1847/211232)	1.15 (1939/168486)
CONUS	1.00 (13246/1319610)	1.41 (2363/167518)	1.18 (2355/199209)	0.84 (1622/193872)	0.83 (1860/223659)	1.00 (2068/208074)	0.78 (1402/179946)	1.06 (1578/149433)
OCONUS	1.61 (2772/172036)	2.26 (545/24140)	1.93 (395/20501)	1.26 (305/24231)	1.18 (298/25182)	1.53 (423/27643)	1.42 (445/31287)	1.89 (361/19052)
Atlantic	0.91 (420/46396)	1.33 (89/6698)	0.97 (51/5245)	0.89 (47/5274)	0.80 (44/5526)	1.26 (98/7791)	0.46 (51/11099)	0.83 (40/4793)
Pacific	1.87 (2352/125640)	2.61 (456/17442)	2.25 (344/15256)	1.36 (258/18957)	1.29 (254/19656)	1.64 (325/19852)	1.95 (394/20218)	2.25 (321/14259)
Shipboard								
Total	0.78 (6348/814987)	1.29 (1477/114279)	0.95 (939/98908)	0.64 (677/105385)	0.66 (820/125008)	0.64 (792/123209)	0.72 (937/129356)	0.59 (708/118790)
Atlantic	0.62 (2802/454838)	0.83 (478/57459)	0.77 (421/54535)	0.53 (305/57699)	0.52 (385/73817)	0.60 (420/69837)	0.66 (471/71881)	0.46 (322/69809)
Pacific	0.98 (3546/360051)	1.76 (999/56820)	1.17 (518/44371)	0.78 (372/47666)	0.85 (435/51389)	0.70 (372/53372)	0.81 (466/57475)	0.78 (384/49098)
Recruits	1.45 (8991/618074)	1.76 (1135/64624)	1.82 (1690/92699)	1.31 (1278/97283)	1.57 (1508/95853)	1.28 (1383/108840)	1.27 (1112/87282)	1.23 (907/73793)

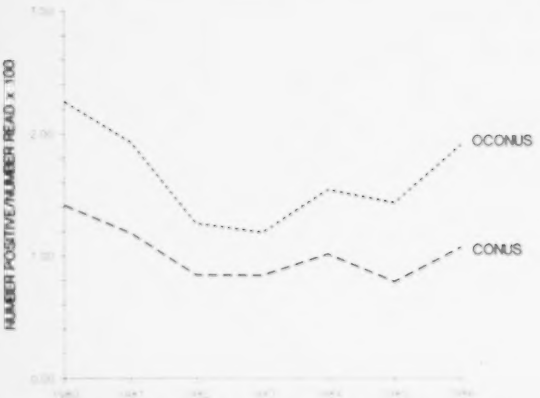

FIGURE 2—Frequency of Positive Tuberculin Tests in Active-duty US Naval and Marine Corps Personnel in Shore-based Facilities within the Continental United States (CONUS) and outside the Continental United States (OCONUS), 1980–86

FIGURE 3—Percentage of Positive Tuberculin Tests in Active-duty US Naval and Marine Corps Personnel by US Naval District, 1980–86

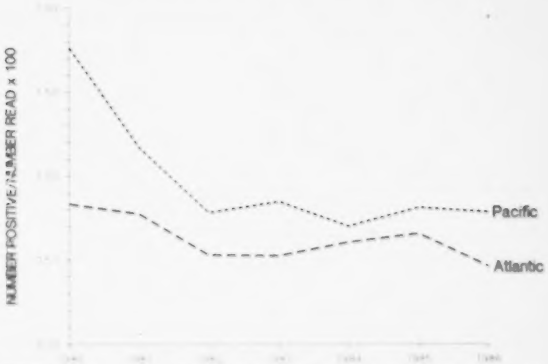

FIGURE 4—Frequency of Positive Tuberculin Tests in Active-duty US Naval and Marine Corps Personnel Assigned to Ships in the Atlantic and the Pacific, 1980–86

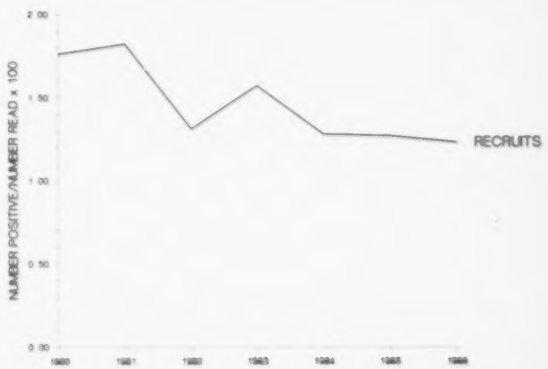

FIGURE 5—Frequency of Positive Tuberculin Tests in US Naval and Marine Corps Recruits, 1980–86

In 1969, 1.59 percent of skin tests were positive overall; 1.72 percent were positive in shore-based facilities in the continental United States; and, 1.08 percent of PPD tests were positive on Navy ships.[6] This level of skin test positivity is higher than for comparable groups during the 1980 to 1986 period of this study and suggests a continuation of the downward trend of tuberculosis infection in the US Navy which was observed prior to 1970.[7]

Between 1958 and 1969, Navy recruits were skin tested with 5 TU PPD by specially trained public health nurses.[7] Tuberculin reactions measuring 10 mm or greater were found in 5.2 percent of the tests. This is substantially higher than the percentage of positive tests (1.45 percent) found in recruits from 1980 to 1986 and corresponds to the lower incidence of tuberculosis in the United States during the period of the current study.

One problem when comparing positive skin test rates in military personnel is that an unknown number of tests are applied but not read, which would lower overall rates. However, there is no reason for this problem to have been greater in any location or any particular year of this study. Also, a person with a positive skin test who is missed on one screening has another chance of being detected on subsequent testing and counted as PPD-positive.

There was a steadily declining incidence rate of tuberculosis disease in the US civilian population until 1984. In 1986, for the first time since 1953, there was a substantial increase in indigenous tuberculosis cases.[8] This reversal has been attributed to an increase in the number of cases of acquired immunodeficiency syndrome (AIDS); to an increase in the number of tuberculosis cases in the homeless, immigrant and other susceptible populations; and, to decreased tuberculosis control efforts.[8–10]

The military population in this study also demonstrated a change in the declining incidence of tuberculosis infection at the time tuberculosis disease was increasing in the civilian population. The military population, however, does not include a large number of individuals who are particularly susceptible to tuberculosis infection, such as the homeless or immigrants, although AIDS cases have been reported in the military.[11,12] Also, the highest rates of positive tests observed in this study occurred in shore-based facilities outside the continental United States, where events occurring in the US may have had less impact.

Factors which could have changed the extent of TB exposure in the military, such as increased deployment to and increased recruitment from endemic regions such as the Philippines, could not be analyzed from the available data.[7,13,14] However, no large shipboard tuberculosis out-

breaks of the magnitude previously reported occurred during this seven-year study period.[2,3]

The "booster effect" is a phenomenon in which increases in the size of the skin test reaction is due to boosting of waned hypersensitivity by sequential testing rather than new infection. Boosting is more common among middle-aged and older persons and when skin testing is done at frequent intervals.[15,16] Because the Naval Medical Command recommends annual testing for personnel in high risk areas and retesting every three years for those personnel at low risk, boosting should be uncommon except in frequently tested groups such as hospital personnel.

ACKNOWLEDGMENTS

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The opinions and assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the US Department of the Navy.

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5th National Conference on Chronic Disease Prevention and Control

The Centers for Disease Control, the Association of State and Territorial Health Officials (ASTHO), and the Directors of State Chronic Disease Programs will cosponsor the fifth national conference on "Chronic Disease Prevention and Control: From 1990 to 2000," October 17–19, 1990 at the Westin Hotel, Renaissance Center, in Detroit, Michigan. The conference is open to the public; there will be a $25 registration fee.

The 1990 conference will include two plenary sessions that address demographic trends:
- Implications for Chronic Disease and the Public's Health
- Tobacco Control: Science and Politics

Six concurrent sessions of invited papers will focus on special populations:
- Minorities, the Elderly, and Rural
- State Highlights—A Look at Michigan
- Quality of Life and Disability—Issues and Impact
- Dietary Fat/Nutrition
- Industry and Chronic Disease Prevention—What Are the Societal Roles and Responsibilities?
- State and Community Tobacco Control Activities

The conference will consider abstracts for oral presentations, poster sessions, or roundtable discussions on relevant chronic disease topics involving: program development and implementation; epidemiologic studies; and social and economic issues.

For additional information, contact: Jack Friel, MPA, Chief, Conference Management Operations, Center for Chronic Disease Prevention and Health Promotion, Mailstop A37, Centers for Disease Control, Atlanta, GA 30333; (404) 639-2249 FAX (404) 639-1563.

Tuberculosis Infection in Urban Adolescents: Results of a School-based Testing Program

M. Anita Barry, MD, MPH, Lillian Shirley, RNC, MPH, Michael T. Grady, MD, Sue Weidhaas Etkind, RN, MS, Carol Almeida, BSN, John Bernardo, MD, and George A. Lamb, MD

Abstract: In 1986–87 a pilot tuberculosis (TB) skin testing program was introduced for seventh and tenth grade students in the Boston (Massachusetts) public schools. The 8.9 percent tuberculin positivity rate in tenth grade students was significantly higher than the 5.1 percent rate found in seventh graders. A majority of those who were skin test positive were born outside the United States. These results suggest that tuberculin testing in an urban school setting may identify a significant number of candidates for TB preventive therapy, particularly among tenth grade students and those who are foreign-born. (*Am J Public Health* 1990; 80:439–441.)

Introduction

Tuberculosis (TB) continues to be an important public health problem in the United States. In 1986, 22,768 cases of tuberculosis were reported nationwide, including 1,214 (5.3 percent) in children younger than 15 years of age.[1] In 1987, nine of 123 verified TB cases in Boston, Massachusetts occurred in children under age 15.[2]

Development of active TB can be largely prevented by isoniazid (INH) therapy in infected persons without disease.[3] However, identification of both adults and children with TB infection is problematic. Current guidelines from the American Academy of Pediatrics for TB testing in children advise annual tuberculin testing in high-risk children, such as those in neighborhoods where the TB case rate is higher than the national average, those from households with one or more cases of TB, American Indian and Alaskan children, and children of parents who have immigrated from Asia, Africa, the Middle East, Latin America, or the Caribbean.[4] In low-risk groups, TB skin testing is recommended at 12–15 months of age, before school entry, and in adolescence. However, no recent published data on the prevalence of tuberculous infection in urban adolescents in the United States are available to assess whether adolescents should be considered at high risk.

Following the diagnosis of TB in a Boston Public School student in the fall of 1986, a TB testing program was introduced for seventh and tenth grade students. Students with positive skin tests were referred for appropriate medical evaluation and preventive therapy. The purpose of this report is to describe the prevalence of tuberculin skin test positivity in seventh and tenth grade students who participated in the program as well as the subsequent course of preventive therapy in those for whom such therapy was indicated.

Methods

The Boston Public School system enrolls approximately 57,000 students. Seventh graders are enrolled in 25 middle schools, and tenth graders in 19 high schools with each school assigned either a full- or part-time nurse. Racial-ethnic breakdown reveals that 47.8 percent of all Boston Public School students are Black, 25.6 percent White, 17.7 percent Hispanic, 8.2 percent Asian, and <1 percent other.

At the time this program was introduced, TB testing was required for new school entrants only. The new policy continued to require TB testing for new school entrants (regardless of grade). In addition, seventh and tenth grade students not previously known to be PPD (purified protein derivative) positive were required to have a TB skin test done during that school year. Due to problems of retention and motivation of urban adolescents in school, however, students were not excluded from classes if they did not comply with the seventh and tenth grade testing policy. Students were encouraged to have tuberculin testing performed by their primary care provider, but arrangements for placement and reading of skin tests in the schools were made. Mantoux testing using intermediate strength PPD was the preferred method for skin testing as suggested by the Centers for Disease Control (CDC) and the American Thoracic Society guidelines.[5] Tubersol (Connaught Laboratories Inc., Swiftwater, PA) was the only PPD product used within the schools. Medical evaluation and a single chest x-ray following a documented positive skin test were mandatory.

Data collected for each tuberculin positive student included information on age, sex, race, country of origin, site of testing, size of PPD induration, site of medical follow-up, and duration of INH therapy. For analysis, chi square testing was used (Professional Database Analysis System; Conceptual Software, Inc., Houston, Texas).

Results

Of 3,642 seventh graders eligible, 2,799 (77 percent) were tested (Table 1). In the tenth grade 2,584/4,353 (59 percent)

TABLE 1—Results of a Tuberculosis (TB) Testing Program, Boston Public Schools, 1986–87

	Grade 7	Grade 10
Number eligible	3,642*	4,353*
Number (percent) tested	2,799 (77)	2,584 (59)
Number (percent)** PPD positive***	145 (5.1)#	233 (8.9)

*This number excludes 34 and 29 known PPD positives in seventh and tenth grades, respectively.
**Numbers include those previously known to be PPD positive.
***PPD positive is defined as induration ≥ 10 mm at 48–72 hours.
#Difference in proportions is 3.8 percent (95% CI = 2.5, 5.2) using test-based confidence interval.

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participated in the program. Participation rates in individual schools ranged from 33 percent to 100 percent in seventh grades and from 16 percent to 100 percent in tenth grades. Of seventh grade program participants, 1,554 (56 percent) were tested in school and 1,245 (44 percent) by their private health care provider. Of tenth graders, 1,881 (73 percent) were tested in school and 703 (27 percent) by a private provider. The rate of tuberculin positivity in seventh graders (5.1 percent) was substantially lower than that in tenth graders (8.9 percent) (see Table 1).

Mean age of seventh graders who tested positive during the program was 13.1 years (range 12 to 15 years); 85 percent were foreign-born. The mean age of tuberculin positive tenth graders was 16.5 years (range 14 to 21 years); 80 percent were foreign-born (Table 2). Country of origin was documented for 295 tuberculin positive students (Table 3). The largest number were from Caribbean countries with Haiti the most common country of origin.

Information on the size of PPD induration was available for 131 of the 315 students with induration ≥ 10 mm. Twenty-four percent had a reading ≥ 20 mm; an additional 13 percent had induration between 16 and 19 mm.

Records to assess compliance with preventive therapy were available for 230 of 314 (73 percent) tuberculin positive students (Table 4). Overall 63 percent of students for whom preventive therapy was recommended completed at least six months of therapy.

An unexpected finding during the program was the discovery of an unsuspected case of active pulmonary TB. The individual was an asymptomatic 20-year-old Haitian male in the tenth grade whose tuberculin test had been negative two years previously. His chest x-ray, done in follow-up to a 25 mm PPD in school, revealed right upper lobe infiltration consistent with active or old TB. Sputum smear was negative. Isoniazid, rifampin, and ethambutol therapy was started. When cultures revealed *M. tuberculosis* sensitive to all antimicrobial agents tested, ethambutol was discontinued. In the high school he attended 41 percent of the tenth grade students participated in the program. Thirty-three (18 percent) of these were tuberculin positive.

TABLE 2—Demographic Characteristics of Students with a Positive* Tuberculin Skin Test during a School-based Testing Program, 1986–87

Characteristics	Grade 7 (N = 111)	Grade 10 (N = 203)
Mean age (years)	13.1	16.5
Sex**		
Male	2.7	5.4
Female	2.5	4.9
Race**		
White	.46	1.4
Black	1.4	4.9
Hispanic	3.2	6.4
Asian	13.8	13.5
Place of Birth		
Foreign-born	95 (85)***	163 (80)
US-born	8 (7)	29 (14)
Unknown	8 (7)	11 (5)

*Positive is defined as induration ≥ 10 mm at 48–72 hours using Mantoux testing.
**Numbers expressed as rate/100 using the total number of students in that category in each grade as denominator.
***Numbers in parentheses denote percentage. May not add up to 100 due to rounding error.

TABLE 3—Birthplace of Tuberculin Positive Students, 1986–87 (N = 295)

Region	Number (%)
Caribbean	
Haiti	51 (17)
Dominican Republic	20 (7)
Puerto Rico	11 (4)
Other	31 (11)
Asia	
China	43 (14)
Vietnam	29 (10)
Cambodia	10 (3)
Other	5 (2)
North America	
United States	37 (13)
Central America	
Other	21 (7)
Africa	
Cape Verde	19 (6)
Other	5 (2)
South America	8 (3)
Europe	5 (2)

*Percentages do not add up to 100 due to rounding error.

TABLE 4—Therapy of Tuberculin Positive Boston Public School students, 1985–87

Results	Grade 7 (N = 111)	Grade 10 (N = 203)	Total (N = 314)
No record	37	40	77
Moved	1	6	7
Records reviewed	73	157	230
Previous Rx	7 (10)*	12 (8)	19 (8)
Rx < 6 mos**	19 (26)	65 (41)	84 (37)
Rx ≥ 6 mos	47	80	127 (55)

*Percentage of those in whom record was available for review.
**Includes five students in whom therapy had to be stopped due to adverse effects.

Discussion

In 1979, data from 13 cities with populations of 250,000 or greater revealed a tuberculin positivity prevalence of 5.6 percent in those ages 5–14 years.[6] In our study, the 8.9 percent rate of tuberculin positivity found in tenth graders whose mean age was 16.5 years was substantially higher than the 5.1 percent found in seventh graders whose mean age was 13.1 years. There are several possible explanations for the higher rate in tenth graders. There may be proportionately more foreign born tenth graders than seventh graders; data to document this possibility are not available. Second, the higher rate may be related to more advanced age and consequently a longer chance to become infected. Third, higher rates in tenth graders may have occurred as a result of spread from the tenth grade student with unsuspected Class III pulmonary TB. However, the case was not smear positive, and excluding results from his school lowers the prevalence rate in tenth graders only to 8.3 percent (200/2405).

Generalizability of tuberculin prevalence rates noted in this study to all urban adolescents is limited by several factors. Rates in students may differ from those in adolescent drop-outs and student non-participants. Although data are available for 59 percent of tenth graders and 77 percent of seventh graders, little is known about those who chose not to

participate. Finally, Boston is a gateway city for refugees; this may result in higher rates of tuberculin positivity than in cities where refugees are less common.

Delivery of adequate preventive therapy, defined as ≥ six months of INH, could be documented in a greater proportion of seventh graders than tenth graders (74 vs 59 percent). These figures are comparable to the 73.4 percent completion of preventive therapy figure noted by CDC among all persons for whom preventive therapy was recommended over a 12-year period in 16 selected regions of the US.[7]

Current TB surveillance practices in the United States vary widely. A 1983 survey of health department officials revealed that only 20 percent of respondents required TB screening in students.[8] The authors concluded that testing of students was non-productive other than "in schools with large numbers of refugees, immigrants, children of indigent parents, etc." The proportion of urban school systems which meet such criteria is unknown, and data from other geographic regions are needed to resolve the issue.

In the United States, TB rates are higher in minorities. In 1986, the TB case rate per 100,000 population was 4.6 in Whites, 17.7 in Hispanics, 27.6 in Blacks, and 50.4 in Asian/Pacific Islanders.[9] Our tuberculin positivity rates reflect this trend with the highest prevalence in Asians, followed by Hispanics and Blacks.

Using data from an analysis done by the Centers for Disease Control in 1986,[10] the cost effectiveness of this program can be demonstrated. Assuming progression to active TB in 10 percent of those not receiving preventive therapy, and $4,983 cost for a single uncomplicated case of disease, the cost of preventable cases detected in this program would be $189,384. In contrast, the actual cost of the screening was $75,465 ($19,000 for a part-time nurse coordinator, $1,057 for screening supplies, and $55,408 for the clinical and pharmaceutical costs related to INH follow-up) for a net savings of $113,919.

Preventable TB cases continue to occur in children in the US.[11] Our data suggest that TB testing in urban school settings comparable to ours is likely to uncover a significant number of adolescents who are tuberculin positive. Because of the relatively high risk of progression to active tuberculosis in adolescents who are infected with TB, identification of and therapy for tuberculin positive children and adolescents is particularly important.[12]

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AAFP Will Publish Family Practice Research Journal

Family Practice Research Journal is now an official journal of the American Academy of Family Physicians (AAFP), signifying the Academy's efforts to support clinical research by family physicians. The Academy recently assumed ownership from Sciences Press, a subsidiary of Plenum Publishing Corporation, New York. *American Family Physician* also is a medical journal of the Academy.

According to the publisher, Clayton Hasser, "*Family Practice Research Journal* aims to complement other family journals by focusing on the stimulation and renovation of clinically oriented, practice-initiated and practice-based research articles, with emphasis on assisting novice family physician researchers and authors to implement and publish their research studies." Currently published twice a year, the journal will become a quarterly publication beginning with the June 1990 issue.

The American Academy of Family Physicians, headquartered at 8880 Ward Parkway, Kansas City, MO 64114, is the national association of family doctors. It is the largest national medical specialty organization with more than 68,000 members. The Academy was founded in 1947 to provide high continuing medical education standards.

Epidemiologic and Clinical Characteristics of Persons with Serologic Evidence of *E. canis* Infection

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Abstract: The results of a serosurvey of Oklahomans for the presence of antibody to *Ehrlichia canis* is reported. Paired serum specimens, from patients lacking the serologic criteria for diagnosis of Rocky Mountain spotted fever (RMSF), were tested. A four-fold increase in *E. canis*-IFA antibody was found in 16/144 (11 percent) of these paired serum samples. Patients with serologic evidence of *E. canis* infection had a mean age of 34 years, 69 percent were male, and 63 percent lived in a town <10,000 population. Signs and symptoms included: fever 94 percent, headache 94 percent, fatigue 94 percent, anorexia 81 percent, nausea 60 percent, and rash 44 percent. When compared to control patients, whose sera were submitted for RMSF testing but did not meet serologic criteria for RMSF or *E. canis*, case-patients were more likely to have had leukopenia (OR = 4.9, 95 percent CI = 1.2, 19.0) and tick exposure (OR = 9.5, 95 percent CI = 1.4, 62.7). The results suggest *E. canis*, or a closely related agent, is a cause of human illness. Ticks are a probable vector. (*Am J Public Health* 1990; 80:442–445.)

Introduction

In each of the years 1982–86, Oklahoma has had the highest rate of Rocky Mountain spotted fever (RMSF) in the United States. For every case of RMSF reported, at least four other persons were tested for RMSF, but were negative for RMSF antibody.[1] We have suspected that these illnesses, which we have called "Oklahoma tick fever," may be caused by other tick-borne agents.[1]

Ehrlichia canis is the causative agent of canine ehrlichiosis, a tick-borne, rickettsial disease of dogs with wide geographic distribution.[2,3] In 1986, the first case of human infection with *E. canis* was described.[4] The patient was bitten by several ticks 12 days before onset of illness and was initially suspected to have RMSF. The diagnosis of *E. canis* infection was made from electron micrographs of leukocytic cytoplasmic inclusions characteristic of *Ehrlichia* species, and the presence of an eight-fold fall in serum antibody titer to *E. canis* during the seven weeks following onset of illness. Since discovery of the first case, additional patients have been identified, by serologic testing, from at least 11 states.[5–9] Although *Ehrlichia canis* has not been isolated under natural conditions from species other than *Canidae*, current evidence supports the role of *E. canis*, or a closely related agent, as a cause of illness among human beings.[2]

We undertook a case-control study to link clinical characteristics and risk factors with serologic evidence of *E. canis* infection.

Methods

Case Surveillance

To identify patients with serologic evidence of *E. canis* infection, we tested paired (acute- and convalescent-phase) sera from 144 persons. The survey included all available paired serum specimens that were tested for *R. rickettsii* in Oklahoma during 1986 and found negative. We obtained sera from the Oklahoma State Department of Health (OSDH) and a commercial hospital laboratory located in Oklahoma City. Using an indirect fluorescent antibody test (IFA), the convalescent-phase specimen for each pair was screened for *E. canis* antibody at 1:16 dilution. For patients whose sera was positive at 1:16, the acute- and convalescent-phase specimens were tested in consecutive two-fold dilutions until an end point titer was reached. A positive test result was defined as a four-fold or greater increase in antibody titer to *E. canis* antigen, and a peak convalescent-phase titer of ≥1:64.

Sera from patients with positive tests for *E. canis* were also tested for antibodies to *Rickettsia typhi* and *Coxiella burnetti* by microagglutination test at the Rocky Mountain Laboratories, Hamilton, Montana.[10] All available blood smears from case-patients, made during their illness, were examined for leukocytic intracytoplasmic inclusions. Blood specimens obtained from dogs belonging to case-patient households were tested for *E. canis* antibody at the Oklahoma Animal Disease Diagnostic Laboratory, Oklahoma State University, Stillwater.[11] Three ten-fold serum dilutions beginning at 1:10 were tested for each dog specimen.

Case-Control Study

We compared the epidemiologic and clinical characteristics of the 16 patients with positive tests for *E. canis* with 32 controls randomly selected from the 128 RMSF-IFA negative patient serum pairs remaining after case-patients had been identified. Controls were matched by laboratory from which the case-specimen originated, and consisted of patients whose acute- and convalescent-phase serum was <1:64 for *R. rickettsia* and convalescent-phase serum <1:16 for *E. canis*.

Efforts were made to conduct personal interviews for case-patients and controls. Substitute control-patients were randomly chosen from the remaining controls when it was not possible to complete interviews with the initially selected controls. Interviews began with open-end questions and concluded with questions designed to elicit specific clinical and demographic information and animal and arthropod exposure histories. All available medical records were reviewed for cases and controls. Odds ratios (OR) and 95%

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confidence intervals (CI) were calculated using the program developed by Rothman and Boice.[12]

Laboratory Procedure

A standard IFA procedure for detecting antibody to *R. rickettsii* had been used by both laboratories from which RMSF-negative serum was obtained.[13] All testing of human serum for *E. canis* antibody was done at the Oklahoma State Department of Health (OSDH) laboratory using a commercially available *E. canis* antigen (ProtaTek International Inc., St. Paul, Minnesota*), and according to a published procedure[11] with the following exceptions: human serum was substituted for canine serum, dilutions started at 1:16, a quantitative control was used, and goat anti-human conjugate (Clinical Sciences, Wippany, New Jersey*) was substituted for anti-dog conjugate. A random sample of case-patient sera was blindly retested for *E. canis* antibody by OSDH laboratory personnel. Agreement was confirmed if the second test result was within one dilution of the first result.

Results

Case Surveillance

Of 144 serum pairs tested, 16 (11 percent) had a four-fold increase in antibody titer to *E. canis* antigen (Table 1). The geometric mean titer for case-patients increased during each successive week for the first month after onset. An agreement of 80 percent between the first and second test result was recorded when 10 of the serum specimens were blindly retested for *E. canis* antibody.

Case Description

The mean age of case-patients was 34 years (range 2–68), and more than two-thirds were male (Table 2). Most case-patients lived in a town of less than 10,000 population or a rural unincorporated area, and all but one case-patient recalled finding one or more ticks on their body during the 30 days before onset of illness.

Signs and symptoms most frequently experienced by case-patients included fever, headache, fatigue, anorexia, and nausea (Table 3). Rash was present in 7/16 (44 percent) patients, and was described as petechial (three patients), maculopapular (three patients), or vesicular (one patient). When present, rash was limited to the trunk and proximal extremities; no case-patient had rash involving hands or feet.

Results of 43 total leukocyte counts performed on 15/16 (94 percent) case-patients were evaluated. Leukopenia, (WBC <4,800/mm^3), was observed in 10/15 (67 percent) case-patients. Excluding patient #4, whose onset date is uncertain, the median day after onset of clinical signs for the lowest total leukocyte count was day 4 (range 0–11 days) for the remaining nine case-patients who had leukopenia. Of 35 total lymphocyte counts available for 14 case-patients, lymphopenia, (<1,500/mm^3), occurred in 9/14 (64 percent), and

TABLE 2—Demographic Characteristics and Exposures for Patients with Serologic Evidence of *E. canis* infection, Oklahoma, 1986

	Cases No./Total (%)	Controls† No./Total (%)	Odds Ratio	95% Confidence Interval
Age X = 34 (range 2-68) X = 28 (range 2-80)				
Sex (male)	11/16(69)	19/32(59)	1.5	(0.4, 5.4)
Reside in town <10,000	10/16(63)	13/32(41)	2.4	(0.7, 8.4)
Arthropod exposure, Ticks on body				
Attached	12/16(75)	16/31(52)	2.8	(0.6, 13.3)
Attached or crawling*	15/16(94)	19/31(61)	9.5	(1.4, 62.7)
Chiggers	7/16(44)	10/30(33)	1.6	(0.4, 5.5)
Fleas	0/16(00)	9/30(30)		
Mosquitoes	8/16(50)	18/30(60)	0.7	(2.3, 0.2)
Dog contact	14/16(88)	23/31(74)	2.4	(0.5, 12.9)
Two or more dogs	6/16(38)	6/31(19)	2.5	(0.7, 9.6)
Other animals	5/16(31)	10/29(34)	0.9	(3.2, 0.2)

†Denominator may be less than 32 due to missing information.

*Use of trade names is for identification only and does not imply endorsement by the United States Department of Health and Human Services, or the United States Public Health Service.

TABLE 1—Laboratory Findings for Patients with Serologic Evidence of *E. canis* Infection, Oklahoma, 1986

Patient Number	Age (years)	Sex	Total WBC*	Total Lymphocytes	Total Platelets*	AST† (SI)	Titer 1	Titer 2	Titer 3
1	02	M	1,600	800	31,000	23.50	<16 (03)**	2,560 (19)**	128 (216)**
2	59	M	2,500	1025	NORMAL	–	16 (03)	64 (22)	128 (189)
3	59	F	2,100	630	68,000	0.48	16 (14)	64 (22)	32 (93)
4	46	M	4,000	1040	357,000	0.70	<16 (84)	64 (99)	128 (140)
5	36	F	6,300	1827	–	–	<16 (01)	64 (13)	128 (204)
6	10	F	4,500	2610	NORMAL	–	32 (04)	128 (12)	128 (69)
7	28	M	9,700	1455	275,000	–	<16 (03)	64 (34)	128 (121)
8††	12	M	3,400	1836	173,000	11.50	<16 (02)	128 (16)	128 (116)
9	40	M	1,600	464	71,000	3.43	16 (04)	256 (10)	64 (278)
10	09	M	–	–	–	–	16 (03)	128 (18)	32 (233)
11††	39	M	10,400	2496	NORMAL	–	16 (15)	256 (29)	32 (152)
12	07	M	2,800	1456	–	–	32 (03)	128 (18)	32 (225)
13	66	F	1,900	570	33,000	6.45	16 (05)	128 (24)	256 (207)
14	37	M	10,900	6758	153,000	–	32 (15)	128 (29)	– –
15	26	M	5,500	2214	–	–	64 (15)	256 (24)	128 (236)
16	68	F	4,300	645	380,000	0.48	32 (01)	64 (11)	128 (128)

*Lowest value recorded.
†Highest value recorded, reference range 0–0.58 ukat/L.
**Days after onset of illness.
††Patient had other laboratory confirmed diagnosis.

TABLE 3—Clinical Characteristics of Patients with Serologic Evidence of *E. canis* Infection, Oklahoma, 1986

	Cases* No./Total (%)	Controls† No./Total (%)	Odds Ratio	95% Confidence Interval
Signs and Symptoms				
Fever	15/16(94)	28/32 (88)	2.1	(0.2, 20.5)
Headache	15/16 (94)	22/32 (69)	6.8	(1.0, 48.3)
Fatigue	15/16 (94)	30/32 (94)	1.0	(1.0, 1.0)
Anorexia	13/16 (81)	24/30 (80)	1.1	(0.2, 5.1)
Nausea	9/15 (60)	19/32 (59)	1.0	(0.3, 3.5)
Myalgia	7/15 (47)	17/32 (53)	0.8	(0.2 2.7)
Vomiting	7/16 (44)	17/32 (53)	0.7	(0.2, 2.3)
Rash	7/16 (44)	22/32 (70)	0.4	(0.1, 1.2)
Adenopathy	3/16 (19)	6/32 (19)	1.0	(1.0, 1.0)
Abdominal pain	3/16 (19)	6/32 (19)	1.0	(1.0, 1.0)
Confusion	2/16 (12)	4/32 (12)	1.0	(1.0, 1.0)
Hospitalized	6/16 (38)	10/32 (31)	1.3	(0.4, 4.7)
Treatment with chloramphenicol or tetracycline	14/16 (94)	28/31 (88)	0.8	(0.1, 12.3)
WBC <4,800	10/15 (67)	7/24 (29)	4.9	(1.2, 19.0)
Lymphocytes <1,500	9/14 (64)	11/23 (48)	2.0	(0.4, 9.7)
Platelets <150,000	5/13 (38)	4/18 (22)	2.2	(0.4, 14.1)

*Denominator may be less than 16 due to missing information.
†Denominator may be less than 32 due to missing information.

excluding patient #4, the median day after onset of illness for the lowest lymphocyte count was 6.5 (range 0–15 days). Platelets were evaluated in 13 case-patients, five (38 percent) of which were thrombocytopenic, (<150,000/mm^3). A total of 21 platelet counts were performed on the five thrombocytopenic case-patients, and the median day after onset for the lowest platelet count was 6 (range 3–16 days).

Of seven case-patients for whom data were available, five (71 percent), had serum aspartate aminotransferase (AST) values above the reference range (0–0.58 ukat/L). Serum from six of these patients was also tested for total bilirubin, and in two patients (Nos. 8,9 Table 1) the values were 6.6 and 2.2 mg/dL, respectively.

Sufficient paired serum was available from 13/16 (81 percent) case-patients to test for antibodies to *Rickettsia typhi* and *Coxiella burnetti*. No significant titers (>1:8) were detected to either antigen. Microscopic examination of peripheral blood smears from three case-patients and a buffy coat smear from a fourth, made during clinical illness, did not reveal inclusions characteristic of *Ehrlichia* spp.

Illness resulted in hospitalization for 6/16 (37 percent) case-patients, and tetracycline or chloramphenicol was prescribed for all but two. There were no case-patient deaths.

Two case-patients were diagnosed with other illness. One 12-year-old boy (No. 8) had vomiting, jaundice, petechial rash, and a positive IgM-specific anti-HAV test diagnostic of recent hepatitis A infection; the other, a 39-year-old male (No. 11), had fever, fatigue, posterior cervical adenopathy, and a four-fold decrease in antibody titer to Epstein-Barr virus supporting a diagnosis of mononucleosis. A third case-patient (No. 4) developed a chronic illness with fatigue, myalgia, and a vesicular rash on his arms, head and mouth two to four weeks after removing a tick from his abdomen. One month after onset he received a 10-day course of tetracycline 250 mg QID. Serum specimens drawn 84, 99, and 140 days after onset revealed no antibodies to *R. rickettsii* but elevated titers to *E. canis*. The cause of illness in the remaining 13 case-patients was either unknown or clinically thought to be RMSF despite negative serologic tests.

Of 25 dogs in 14 case-patient households, we tested 16 (64 percent), from nine households, for *E. canis* infection and none had antibody titers, ≥1:100, suggestive of recent infection.

Case Control Study

All cases, and 27/32 (84 percent) controls were interviewed in person by one or two of the authors (BWR and JRH). Cases and controls were informed of serologic results prior to interview. Of 32 original controls, two were deceased, two had moved out of state, and one could not be contacted. Information for the two deceased controls was obtained from medical records, and telephone interviews were conducted for three others, two of whom had moved out of state and one for whom personal interview was impractical due to constraints of time and distance.

The median interval of time from onset of disease to collection of acute- and convalescent-phase serum was similar for cases (median = 4 and 18.5 days, respectively), and controls (median = 3 and 17 days, respectively). Case-patients were slightly older, more often male, and somewhat more likely to live in a town of <10,000 population or a rural unincorporated area than were controls (Table 2).

Signs and symptoms in case-patients were similar to those of controls except for an increased frequency of headache reported by case-patients. Case-patients were more likely to have developed leukopenia (WBC <4,800/mm^3) and to have reported finding a tick on their body in the month before onset of illness both of which were statistically significant (Table 2). Exposure to bloodsucking arthropods other than ticks was similar for case-patients and controls except that controls were more likely to report flea bites.

Discussion

Our findings support the role of *E. canis*, or an antigenically similar agent, as a cause of clinical illness among humans in Oklahoma. We have demonstrated serologic evidence of *E. canis* infection in a subgroup of Oklahomans whose serum was submitted for RMSF testing, but that did not meet the criteria for serologic confirmation. Laboratory findings of leukopenia, lymphopenia, and elevated hepatic enzymes in both canine [14] and human[4–9] patients are consistent with the hypothesis of a common agent. The similarity of clinical characteristics among case-patients and the differences between case-patients and controls support the hypothesis that the case-patient syndrome is a distinct clinical entity.

E. canis is reported to have a limited host range,[2] and the only recognized vector, *Rhipicephalus sanguineus*, feeds infrequently on humans.[15] This suggests the possibility of an antigenically similar, but distinct, agent as the cause of illness in case-patients, or an unrecognized mechanism of transmission for *E. canis*.

The proportion of *E. canis* positive serum pairs in the sample tested (16/144, 11 percent) when applied to the total number of serum specimens found negative for RMSF in Oklahoma each year implies that the incidence of human ehrlichiosis may be as much as half that of RMSF. The frequency with which evidence of infection was found in Oklahomans and the wide distribution of *E. canis* in the United States suggests that other states should consider

surveillance to determine the presence and distribution of infection.

Further studies are necessary to assess the sensitivity, specificity, and reliability of the canine IFA test for humans. Our results, on a limited sample, demonstrated an 80 percent reliability. The two case-patients with confirmed diagnoses of hepatitis A and mononucleosis may reflect on the specificity of this test. Based on the antibody response of all other case-patients, the rise in titer for patient No. 4 between days 88 and 94 after the reported onset of illness suggests the possibility of concomitant illness and a more recent exposure to *E. canis* antigen.

Clinical characteristics of case-patients were markedly similar to patients with RMSF and to controls. Oklahomans with symptoms of fever, headache, rash, and a history of tick-bite during the period April through August will prompt submission of samples for RMSF testing. Clinical characteristics among case-patients could be biased as a result of selection for RMSF and may not be representative of the disease as it exists in the general population. Differences such as the increased frequency of low white blood count in case-patients, when compared to controls, are likely to be valid. Rash occurred less frequently in case-patients than in controls, and when present was not of the typical pattern described for RMSF.

The results of this study implicate ticks as vectors for transmission of infection to case-patients. In spite of a selection bias for increased frequency of tick exposure in the population from which both case-patients and controls were drawn, a significantly higher proportion of case-patients reported finding ticks on their body than did controls. Previous reports indicate 50 percent to 100 percent of patients with serologic evidence of *E. canis* infection had tick exposure.[5-9] The recognized vector for *E. canis* in dogs in the United States, *R. sanguineus* (the brown dog tick), is reported to feed on humans infrequently suggesting that this species is of questionable importance in transmission of infection to humans. When the agent of human ehrlichiosis is isolated from a human patient, further studies of other tick vectors are indicated. If the agent is identified as *E. canis*, then the possibility of chronic infection of humans should be investigated, as chronic *E. canis* infection has been documented in dogs.[16]

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Self-Rated Health and Mortality in the NHANES-I Epidemiologic Follow-up Study

ELLEN L. IDLER, PHD, AND RONALD J. ANGEL, PHD

Abstract: The ability of self-rated health status to predict mortality was tested with data from the National Health and Nutrition Examination Survey (NHANES-I) Epidemiologic Follow-Up Study (NHEFS), conducted from 1971–84. The sample consists of adult NHANES-I respondents ages 25–74 years (N = 6,440) for whom data from a comprehensive physical examination at the initial interview and survival status at follow-up are available. Self-rated health consists of the response to the single item, "Would you say your health in general is excellent, very good, good, fair, or poor?" Proportional hazards analyses indicated that, net of its association with medical diagnoses given in the physical examination, demographic factors, and health related behaviors, self-rated health at Time 1 is associated with mortality over the 12-year follow-up period among middle-aged males, but not among elderly males or females of any age. (*Am J Public Health* 1990; 80:446–452.)

Introduction

A rather intriguing and consistent finding concerning the predictors of mortality has been reported by several researchers in recent years. Studies based on four different sets of data indicate that a simple, one item self-report of health status is as powerful a predictor of mortality as more detailed health status indicators.[1–3] In these studies the response to some variant of the question, "Would you say your health is excellent, very good, good, fair, or poor?" is significantly associated with the risk of mortality over periods of four to nine years, even when prior information about health and all other major known risk factors are accounted for. Without good baseline health indicators, this finding would not be particularly surprising. But the varieties of methods used and the consistency of the findings in these studies leads to the rather profound realization that what we learn from this simple question carries a great deal of clinically relevant information, making it as powerful a predictor of mortality as simultaneous reports of serious chronic disease or functional disability.

The first of these studies was published in the *American Journal of Public Health* in 1982.[1] In it, Mossey and Shapiro reported that in a large elderly Canadian sample of the province of Manitoba, respondents' self-ratings of health outweighed the extensive health care utilization and medical history data available in predicting survival over a seven-year period. The editor of the Journal, realizing the importance of the finding, highlighted it in an accompanying editorial.[4]

Subsequent studies in California, Connecticut, and Iowa have replicated the finding. Adults in Alameda County, California, who assessed their health as poor had increased relative risks of mortality of approximately 1.95 compared to those who said they were in excellent health, even when self-reports of chronic and acute illnesses, sex, age, income, education, health practices, and social networks were controlled.[2] Elderly adults in New Haven, Connecticut, and Iowa and Washington Counties, Iowa, had increasing risks of mortality for every level of self-assessed health other than excellent, despite controls for chronic conditions, disability, pain, medications, demographic factors, and health practices.[3] Together, these studies suggest that individuals have access to very important internal information with which they are able to make informed predictions of their probability of survival.

The primary methodological problem that makes attempts to assess the medical significance of self-reported health status difficult arises from the inability to unambiguously control for actual physical health status. In models in which self-assessments are used as predictors of some outcome, their impact may in fact be due to inadequately measured physical health status. In other words, it is difficult to establish that self-assessed health has an influence above and beyond that it shares with actual physical health. The studies mentioned above all took different approaches to isolating the net effect of self-assessed health, but all of these controls for physical health relied heavily on the self-reports of respondents and, as a result, none could be considered ideal controls for "objective" health status. Even the Canadian study, which depended to a smaller extent on respondent information, substituted records of medical care utilization which the authors acknowledged were subject to biases of their own.[1,4] No population-based study of self-assessed health and mortality has had as its physical health status control the results of a comprehensive, standardized physical examination carried out by trained physicians. Such an examination could be considered objective because it would consist of observations made by an external and impartial observer, and because a single standard would be applied to all participants in the study. Only this form of measurement would have the possibility of detecting untreated, undiagnosed, and otherwise unreported conditions.

Methods

Such a methodology is precisely the one employed by the National Center for Health Statistics's National Health and Nutrition Examination Survey (NHANES-I),[5] conducted from 1971 to 1975, and the NHANES-I Epidemiologic Follow-up Study (NHEFS),[6] conducted from 1982 to 1984. Specifically, the NHANES-I provides data from a detailed physical examination of a 20 percent subsample of the first-stage NHANES-I sample (N = 3,854), and an additional sample which is called the augmentation component (N = 3,059); together these make up a baseline sample of 6,913 adults ages 25 to 74 years who constitute a probability sample of the United States population. The first-stage NHANES-I sample deliberately oversampled certain population subgroups believed to be at risk for nutrition-related health

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problems: persons living in poverty areas, women of childbearing age, and the elderly. This sample is thus simultaneously very heterogeneous and nationally representative.

The follow-up to the NHANES-I, the NHEFS, successfully traced 92.9 percent of the original NHANES-I cohort;[7] for the subsample analyzed here the proportion traced was slightly higher, 93.2 percent. Those who were lost to follow-up tended to be younger, especially Black males and White females.[7] Respondents are included in the present analysis if they were successfully traced (by being contacted in person or having a proxy and/or a death certificate to verify their death), regardless of whether or not they completed the follow-up interview. This yields a usable sample size of 6,440. We might note that tracing over 90 percent of the sample 10 years after the initial survey is a truly commendable feat and it makes these data one of the best sources available for studying the predictors of mortality. The death rate for the subsample was 12.9 percent, somewhat lower than the 15.1 percent reported for the total sample.[7] This is likely due to the fact that the augmentation component was added to the sample at the end of the NHANES-I data collection period, thus making follow-up periods shorter for a substantial portion of our sample. The outcome variable used in this study is survival time following the initial interview.

The NHANES-I and the NHEFS are ideal for our purposes because they contain a fully objective source of information on the respondent's medical diagnoses at Time 1, as well as the respondents' self-ratings of health. The physicians made direct physical examinations of the respondents, including standing and sitting blood pressure and pulse, examination of the ears, head, eyes, mouth, neck, abdomen, major and minor joints, and skin, percussion of the liver, and auscultation of the heart. Based on positive responses to screening questions, the physician then administered supplemental questionnaires for arthritis, respiratory, and cardiovascular diseases. In addition, the physicians had immediately available to them the results of the following laboratory and other tests: hemoglobin, hematocrit, red and white blood cell counts, height and weight, body and skinfold measurements, audiometry, spirometry, electrocardiogram, single breath diffusing capacity, goniometry, and X-rays of the chest, hand and wrist, hips and knees.[5] The examination is as detailed as one could reasonably expect in a population survey of this scale. The physicians recorded their findings by coding any abnormalities they found in terms of the International Classification of Diseases Revision Eight (ICD-8) categories[8] and judged the severity of these conditions on a three-point scale as either minimum, moderate, or severe.[9] To create a set of variables which would summarize this information we grouped the ICD diagnosis codes by the ICD's own 15 broad categories: infectious diseases, cancers and neoplasms, metabolic disorders, blood diseases, mental disorders, diseases of the nervous, circulatory, respiratory, digestive, and genitourinary systems, diseases of the skin and musculoskeletal systems, congenital anomalies, symptoms of ill-defined conditions, and accidents.*

An individual received a score of one for that category if they had one or more diagnoses present; the score was then weighted by one, two, or three for the most severe grading the physician had given that individual for any diagnosis in that ICD category. Respondents were scored zero if they received no diagnosis in a category. In addition, to account for the additive effects of comorbidity, we created a variable that consists of the sum of unweighted separate diagnoses each respondent received. This complete set of variables is introduced first into all models as a control for medical diagnoses. It represents a distinct alternative to the methods used in previous studies in that it distills a large amount of information from a comprehensive physical examination that was conducted by a small team of physicians in a structured and standardized manner, and it incorporates a weight for the clinical severity of the condition, a feature which even the most accurate or extensive self-report data cannot claim.

Certainly a physical examination of the sort administered in the NHANES-I is not unerring. In a single cursory examination, the physician is likely to miss subtle conditions or syndromes in their prodromal state. Diagnosis is a complex task that depends on the physician's judgment and any single assessment of a person's state of health is always subject to observer variability.[10] The examination represents no more than the physician's informed clinical judgment arrived at after a brief encounter with a new patient. However, because the examination was performed by an external, independent, medically trained observer, and because these observers relied on standardized criteria for assessing the presence or absence of specific conditions, these diagnoses avoid confounding the impact of self-rated health on mortality with that of self-reported controls for health.

The independent variable of primary interest in this study is the response to the single item, "Would you say your health in general is excellent, very good, good, fair, or poor?" The question wording used here, and the labels for the five response categories differ slightly from those used in other studies;[1-3] in some cases the categories are excellent, good, fair, poor, and bad; sometimes only four categories are included and "poor" is omitted; sometimes the eliciting question contains a relativizing phrase such as "compared to other people your age . . ." The significance of these alternate forms in unknown; however, to date no two published studies have used the same form, although all have reported similar effects. Because of low frequencies in the "poor" category, the "poor" and "fair" categories were combined in this analysis, reducing the five response categories to four; the responses are treated as a set of n-1 dummy variables with the omitted category being "excellent" health.

The following sociodemographic variables are included in the analysis as controls: age (treated as a continuous variable); and dummy variables for race, 9 to 12 years of education or 13 or more years (compared with 8 or fewer), marital status, income less than $5000, income less than $15,000, and being currently employed. Health behavior variables included dummy variables for current smoking and past smoking (compared with never having smoked); an index of alcohol use based on the frequency of consumption multiplied by the number of drinks usually consumed, weighted by their alcohol content; the physician's indication of the presence of obesity; and a measure of inactivity based on frequency of exercise and other physical activity.

Proportional hazards models based on the SAS statistical procedure PHGLM[11,12] are used to estimate the effect of self-rated health on the probability of survival during the follow-up period. This procedure was used because of the censored nature of the data and because we wish to identify the predictors of survival time over the entire follow-up period. A complete, parallel set of logistic regression analy-

*Two additional categories, for pregnancy complications, and perinatal morbidity and mortality, were omitted as irrelevant and of negligible frequency.

ses on the dichotomous mortality outcome was also performed, which in effect measured the probability of survival at just one time point, the end of the follow-up period. The results of these logistic regressions were nearly identical to those from the survival analyses, hence we present only the latter. Odds ratios are calculated from the hazard models by taking the antilog of the estimated coefficients.[13] Three models are estimated, one containing just the physician's examination variables and self-rated health, one which adds sociodemographic variables in a block, and a third which adds health behavior variables. In this way, any changes in the relation between self-assessed health and mortality can be attributed to the various control variables.

Results

Figures 1 and 2 show the unadjusted relationship between self-assessed health at the time of the first interview and the probability of survival over the 12-year follow-up period for men and women. These figures show that: 1) mortality risks increase for individuals with progressively poorer self-perceptions of health; and 2) the risks are apparently greater for males than for females. Only 60 percent of males with poor self-assessed health survive to the end of the 12-year period, while nearly 80 percent of the women do. Further, the lines are more straight than curved indicating that the increased risk of mortality is spread throughout the follow-up period. Because of the differences in the risk of mortality, all analyses are performed separately by sex.

The full proportional hazards models are presented in Tables 1 and 2. In the first models, which contain only physician examination variables as controls, self-assessed health explains a significant amount of the remaining unexplained variation in survival time; odds ratios for the extreme category are 3.3 for men and 2.1 for women. There are also progressively elevated risks for the intermediate categories for men, but not for women.

The models which add sociodemographic variables reveal a diminished effect of self-assessed health on mortality for men, with lower odds ratios of 1.7 for poor/fair health and 1.5 for good health. Among the women all effects of self-rated health disappear. Stepwise logistic regressions not shown here reveal that age accounts for most of the association between self-assessed health and mortality for men and for all of the association for women. In this sample older people were more at risk for both poor self-assessments of health and mortality. Among the males, some of the apparent initial

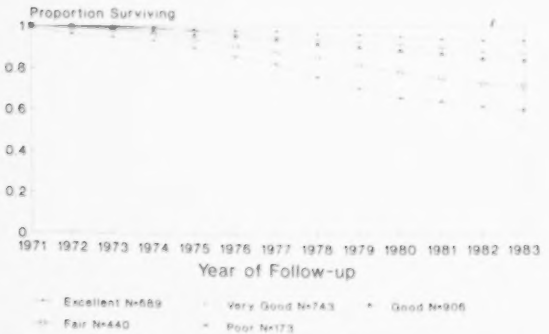

FIGURE 1—Male Mortality in NHANES-I Epidemiologic Follow-up Study by Levels of Self-rated Health

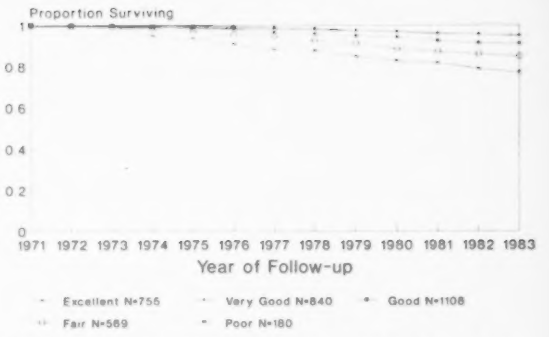

FIGURE 2—Female Mortality in NHANES-I Epidemiologic Follow-up Study by Levels of Self-rated Health

effects of self-assessed health on mortality were also due to confounding effects of employment, marital status, and race. Taking all these variables into account reduces the odds ratio for the extreme category by half.

It should be noted that the model with sociodemographic factors taken into account fits the data much better than the model with health variables alone. The models' likelihood ratio chi squares (G^2) reported at the bottom of Tables 1 and 2 double when these variables are added, and the difference in G^2 is highly significant (588.2, 27 df - 285.9, 19 df = 302.3, 8 df, $p < .000$ for men; 429.0 27 df - 211.8, 19 df = 217.2, 8 df, $p < .000$ for women).

The third model introduces variables for smoking, alcohol use, obesity, and lack of exercise. The inclusion of these factors does not alter the already nonexistent relationship for women. It further reduces the association for men, so that only the extreme category contrast remains. A second stepwise procedure (not presented) revealed that the addition of current smoking to the model had the biggest impact on the association; males who smoke were both more likely to give poor self-assessments of health and to die. The final adjusted odds ratio for the poor/fair category in the men's hazard model is 1.5 (95% CI = 1.1, 2.2).

These models too have a substantially better fit than the sociodemographic models which preceded them. Adding the measures of health behaviors adds 54.1, 5 df ($p < .000$) to the G^2 for men, and 12.3, 5 df ($p < .05$) for women.

Given the literature on the ways in which self-ratings of health vary according to the respondent's social location, certain interactions were considered. We have already seen that self-rated health predicts mortality marginally for men, but not at all for women. But does it predict mortality better for some men (or women) than others? The issue of age is especially salient given that so many of the previous studies have been done with elderly samples. To test an age-self-rated health interaction, we divided the males and females into three groups, those ages 25 to 44, 45 to 64, and 65 to 74. Because deaths were extremely rare (31 males and 27 females) in the under-45 age group, making parameter estimates from these models unreliable, we do not report the results from the youngest group.

The results can be seen in Tables 1 and 2. A substantial difference emerges between the middle-aged and elderly men. The relation between self-rated health and mortality for the male sample as a whole disappears altogether for elderly men, and emerges very strongly for middle-aged men. In the

TABLE 1—Proportional Hazards General Linear Model Coefficients for Survival Time in the NHANES-I Epidemiologic Follow-Up Study (NHEFS) by Levels of Self-Rated Health, Males

	Model 1 Physician Controls	Model 2 Sociodemographics	Model 3 Health Behaviors	Model 3 By Age Group 45–64	Model 3 By Age Group 65–74
ICD-9 Categories					
Infection	−.331 (.220)	−.214 (.222)	−.218 (.216)	−.554 (.418)	−.109 (.304)
Neoplasm	.249 (.105)	.108 (.114)	.125 (.115)	.228 (.213)	.131 (.154)
Endocrine	−.208 (.097)	−.111 (.100)	−.155 (.111)	.012 (.149)	−.309 (.182)
Blood	.050 (.417)	−.055 (.546)	−.141 (.572)	−6.160 (27.3)	.145 (.758)
Mental	−.024 (.165)	.047 (.159)	−.064 (.159)	−.072 (.206)	−.117 (.280)
Nervous	.007 (.072)	−.038 (.073)	−.004 (.073)	.070 (.114)	−.120 (.103)
Circulatory	.242 (.054)	.120 (.059)	.137 (.059)	.202 (.091)	.058 (.086)
Respiratory	.154 (.069)	.190 (.068)	.167 (.070)	.134 (.124)	.201 (.094)
Digestive	.170 (.098)	.112 (.104)	.144 (.109)	.142 (.160)	.084 (.155)
Genital	−.852 (.730)	−.574 (.692)	−.431 (.681)	.084 (.699)	−1.859 (10.2)
Skin	−.050 (.106)	−.122 (.117)	−.161 (.119)	.036 (.185)	−.278 (.163)
Muscle	−.011 (.061)	−.119 (.064)	−.114 (.064)	−.038 (.101)	−.161 (.090)
Congenital	−.249 (.188)	−.280 (.206)	−.369 (.224)	−.822 (.586)	−.334 (.275)
Illness Symptoms	.207 (.082)	.278 (.087)	.260 (.088)	.326 (.127)	.184 (.140)
Accident	−.270 (.136)	−.156 (.131)	−.225 (.132)	.081 (.167)	−.916 (.444)
Sum of conditions	.147 (.042)	.089 (.047)	.068 (.047)	−.046 (.075)	.159 (.066)
Self-rated Health					
Poor/Fair	1.203 (.167)	.552 (.184)	.436 (.189)	1.026 (.330)	−.071 (.258)
Good	.748 (.166)	.396 (.178)	.292 (.182)	.793 (.322)	−.144 (.247)
Very good	.543 (.176)	.287 (.187)	.251 (.190)	.646 (.342)	−.248 (.259)
Sociodemographics					
Age		.059 (.005)	.066 (.006)	.060 (.014)	.083 (.025)
White		−.327 (.127)	−.309 (.129)	−.487 (.195)	−.215 (.191)
High school education		.136 (.115)	.086 (.116)	.077 (.176)	.044 (.176)
College education		.194 (.158)	.158 (.160)	.131 (.241)	.405 (.236)
Married		−.387 (.116)	−.305 (.118)	−.409 (.184)	−.083 (.178)
Income < $5000		.451 (.180)	.384 (.181)	.464 (.269)	.260 (.307)
Income < $15000		.273 (.157)	.210 (.157)	.255 (.213)	.076 (.295)
Working		−.460 (.117)	−.460 (.118)	−.370 (.181)	−.444 (.180)
Health Behaviors					
Present smoker			.616 (.135)	.835 (.230)	.551 (.191)
Past smoker			.027 (.141)	.426 (.244)	−.222 (.187)
Alcohol use			.001 (.000)	.001 (.000)	−.000 (.000)
Obesity			.209 (.124)	.156 (.186)	.220 (.183)
Inactivity			.090 (.044)	.139 (.069)	.090 (.065)
N	2931	2796	2772	1225	514
G^2	285.9	588.2	642.3	186.6	95.3
degrees of freedom	19	27	32	32	32

latter group, odds ratios for the poor/fair, good, and very good categories are 2.8 (95% CI = 1.5, 5.3), 2.2 (95% CI = 1.2, 4.1), and 1.9 (95% CI = 1.0, 3.7), respectively. Self-rated health does appear to predict mortality very strongly for middle-aged men and it predicts better for them than for any other group.

Because we were interested in the relative contributions of the self-rated health and physician examination variables to the explained variance in mortality, we compared the fit of the full models (Model 3 in Tables 1 and 2) with the fit of models from which we had alternately removed the variables for self-assessed health and physician-assessed health. This would allow us to assess the relative contribution of the two health status measures by their contribution to the G^2 for the full model. We estimated these models for the whole sample, and also by age group. Our findings varied, both by sex and by age, and were somewhat surprising: for the women in the sample, neither the physicians' diagnoses nor self-assessed health make any significant contribution to the model G^2 when sociodemographic and health behavior variables are controlled. While some of the individual physician measures were significant predictors of female mortality (notably diagnoses of circulatory and mental disorders), the removal of the entire set of diagnoses from the model failed to reduce the G^2 by a significant amount. For the males, on the other hand, the physician measures (diagnoses of circulatory and respiratory disorders, and ill-defined illness symptoms in particular) did contribute to G^2, both for the male sample as a whole (642.3, 32 df - 606.2, 16 df = 36.3, 16 df, $p < .01$) and for the elderly men (95.3, 32 df - 57.5, 16 df = 37.8, 16 df, $p < .01$). In none of these groups did the block of self-assessed health variables have an impact on G^2. Among the middle-aged men, however, where the effect of self-assessed health on mortality was strong, self-assessed health does alter G^2 (186.6, 32 df - 174.8, 29 df = 11.8, 3 df, $p < .01$). The effect of the physician-assessed measures within this group is marginal (186.6, 32 df - 162.0, 16 df = 24.6, 16 df, $.10 > p > .05$). These comparisons corroborate the findings regarding the importance of self-assessments of health among middle-aged men, and show the findings from the physicians' examinations to be better predictors of male mortality than female mortality.

To focus a little more closely on the role of self-assessed health vis-a-vis the other important predictors of mortality, we compared the odds ratios and partial Rs for the various levels of self-assessed health with those for other variables in

TABLE 2—Proportional Hazards General Linear Model Coefficients for Survival Time in the NHANES-I Epidemiologic Follow-Up Study (NHEFS) by Levels of Self-Rated Health, Females

	Model 1 Physician Controls	Model 2 Sociodemographics	Model 3 Health Behaviors	Model 3 By Age Group	
				45–64	65–74
ICD-9 Categories					
Infection	−.198 (.223)	.209 (.223)	.163 (.223)	.544 (.274)	.071 (.553)
Neoplasm	.171 (.203)	.023 (.211)	.037 (.210)	.038 (.538)	−.069 (.272)
Endocrine	.081 (.079)	.109 (.080)	.142 (.085)	.047 (.149)	.201 (.113)
Blood	.022 (.553)	−.039 (.534)	−.021 (.531)	−7.601 (86.3)	.171 (.483)
Mental	.189 (.183)	.440 (.183)	.419 (.187)	.038 (.372)	.861 (.281)
Nervous	.042 (.102)	−.036 (.105)	−.045 (.105)	.186 (.183)	−.146 (.137)
Circulatory	.443 (.067)	.241 (.071)	.253 (.072)	.360 (.131)	.235 (.093)
Respiratory	−.144 (.148)	.010 (.141)	−.034 (.141)	.100 (.242)	.002 (.181)
Digestive	.051 (.186)	.156 (.183)	.105 (.191)	.180 (.325)	.015 (.268)
Genital	−.197 (.353)	.008 (.352)	.028 (.354)	−6.614 (62.4)	.221 (.426)
Skin	.002 (.190)	.102 (.192)	.088 (.195)	.147 (.307)	.136 (.277)
Muscle	.268 (.071)	.012 (.073)	.020 (.073)	.074 (.145)	.029 (.089)
Congenital	−.401 (.305)	−.183 (.299)	−.236 (.293)	−6.458 (29.3)	.008 (.290)
Illness Symptoms	.112 (.104)	.000 (.106)	.012 (.107)	.064 (.204)	−.068 (.148)
Accident	−.164 (.327)	.056 (.315)	.051 (.315)	−.080 (.527)	.393 (.668)
Sum of conditions	.061 (.046)	−.009 (.049)	−.012 (.049)	−.096 (.105)	.021 (.060)
Self-rated Health					
Poor/Fair	.735 (.188)	.184 (.196)	.204 (.202)	.256 (.354)	.084 (.287)
Good	.324 (.187)	−.029 (.191)	−.010 (.194)	−.009 (.330)	−.037 (.278)
Very good	−.129 (.219)	−.321 (.222)	−.246 (.224)	−.195 (.372)	−.456 (.339)
Sociodemographics					
Age		.067 (.006)	.071 (.007)	.049 (.019)	.049 (.029)
White		−.458 (.151)	−.466 (.152)	−.485 (.259)	−.341 (.214)
High school education		−.022 (.138)	−.056 (.139)	.195 (.248)	−.097 (.185)
College education		−.020 (.191)	−.064 (.193)	−.051 (.377)	−.387 (.269)
Married		−.391 (.128)	−.378 (.129)	−.322 (.229)	−.304 (.170)
Income < $5000		.271 (.228)	.301 (.230)	.553 (.356)	.085 (.395)
Income < $15000		.114 (.213)	.123 (.214)	.046 (.328)	.202 (.387)
Working		−.380 (.164)	−.381 (.165)	.053 (.227)	−.813 (.400)
Health Behaviors					
Present smoker			.384 (.145)	.596 (.222)	.455 (.225)
Past smoker			.332 (.172)	.206 (.330)	.335 (.227)
Alcohol use			.000 (.000)	.000 (.000)	.000 (.001)
Obesity			−.175 (.132)	.158 (.234)	−.305 (.176)
Inactivity			.040 (.057)	.059 (.104)	.015 (.074)
N	3417	3267	3242	1358	557
G^2	211.8	429.0	441.3	74.2	61.4
degrees of freedom	19	27	32	32	32

the full models. For the male sample as a whole, age is the most important predictor of mortality, followed (in this order) by smoking, unemployment, alcohol use, illness symptoms, marital status, race, respiratory diagnoses, poor self-assessed health, circulatory diagnoses, low income, and lack of exercise. Among the middle-aged men, poor self-assessed health ranks fourth as a predictor, behind (in this order) alcohol use, age, and smoking. Among the women the order is age, circulatory diagnoses, race, marital status, smoking, unemployment, and mental disorders. We conclude that health behaviors (especially smoking and high levels of alcohol use) and demographic variables (particularly employment) are more important predictors of mortality in age-adjusted models than either physician- or self-assessed health. Among the physician measures, however, we would note that ill-defined illness symptoms, and diagnoses of respiratory and circulatory problems are the best predictors of mortality for men, and diagnoses of circulatory and mental disorders are most important for women.

In understanding the impact of self-rated health on mortality, one of the other concerns has been that poor self-assessments of health may simply be reflecting the greater severity level or better respondent knowledge of conditions which are leading to imminent mortality.[1] If people with poor self-assessed health only survive for a short follow-up period then the effects of self-assessed health are more easily explained as inadequate controls for physical health status than as indicators of some more subtle process.

To test this possibility, we identified deaths which occurred in three "early mortality" periods of two, three, and five years following the year in which the respondent entered the NHANES-I study, and compared them with the deaths that occurred in the remaining years. We then performed logistic regression analyses with the full set of variables on just the subset of deaths to see if self-assessments of health predicted early mortality better than late mortality. The results (not shown) revealed that, among both males and females, self-rated health did not predict early mortality better than late. This confirms the findings of Mossey and Shapiro,[1] who concluded that ". . . while objective health status may change over time, self-ratings of health represent a relatively stable perception held by the individual." (p 804) This suggests that the effects of self-assessed health on mortality cannot be explained by superior respondent knowledge of the poor prognoses for existing conditions that are leading to imminent mortality.

Discussion

These analyses have shown that the ability of self-rated health to predict mortality in this national sample is in large part explained by the sociodemographic characteristics, health risk behaviors, and the medical diagnoses of the respondents. Only among middle-aged men was there any substantial independent increased risk of death from self-assessments of health after these other factors were taken into account. In this NHANES-I sample, older people, smokers, alcohol abusers, the unmarried, the unemployed, and Blacks were initially less likely to give positive assessments of their health and subsequently less likely to survive, and these factors, by and large, account for the initial association of self-rated health with mortality.

Our findings, then, do not fully corroborate the findings of the earlier studies reviewed above in which self-assessments remained strongly significant predictors of mortality in full samples, in spite of controls for physical health indicators and other factors. Our analysis differs from these earlier studies in two major respects: 1) the NHANES-I provides an alternate indicator of physical health, medical diagnoses; and 2) the analysis is based on a large and heterogeneous national sample. The previous studies were of localized and/or elderly samples, and employed controls for physical health status that were at least in part based on self-reports. We attribute the differences between our results and those of previous studies to these differences in study design.

No other population-based prospective study of self-assessed health has had medical diagnoses derived from a standardized medical examination as statistical controls for physical health status. The NHANES-I detailed examination could potentially yield information on previously undiagnosed as well as treated and self-reported illness. Indeed, this was a primary purpose of the NHANES-I: ". . . to obtain information on unrecognized and undiagnosed conditions—in some cases, even nonsymptomatic conditions."[5] (p 3) In studies which depend on self-reports of conditions for their measure of physical health status, the same optimistic or pessimistic response bias may influence specific reports of conditions as well as global assessments. The current study is unique in basing its controls for physical status on information gathered from an external observer and hence applies the same standard to all respondents, regardless of their race, income status, education level, or access to medical care.

Having said this, it should be noted that however theoretically advantageous these controls for medical diagnoses are, they do not on their own eliminate the association of self-assessed health with mortality in this sample. In fact, they explain very little of the observed variation in survival. Perhaps this should not be surprising; this was a fundamentally healthy young and middle-aged sample and few cases of serious illness were diagnosed. Moreover, in making their diagnoses, the physicians were not asked to consider longevity as a criteria. In any case, among both men and women, self-assessed health remained a strong predictor of survival when the diagnoses alone were controlled. In this analysis, the relationship of self-assessed health to mortality was better explained by age (primarily) and, to a lesser extent, by smoking, alcohol abuse, unemployment, race, and marital status than it was by its confounding with physical health status as judged by these physician examinations.

The second major difference between the present study and those which have preceded it concerns the sample. The NHANES-I sample is large and heterogeneous, quite unlike the community, county, or even provincial samples discussed earlier. One concrete example of the differences is in the role that age plays in the analyses. In the Connecticut and Iowa samples,[3] which contained only elderly respondents, age acted as a suppressor variable on the relation between self-assessed health and mortality; that is, because older (elderly) respondents gave better self-assessments of health at any given level of objective health status than younger (elderly) respondents, controlling for age in the analysis *increased* rather than decreased the effect of self-assessments on mortality. In the NHANES-I sample of young and middle-aged adults and only relatively young elderly, the effect of age is very much the opposite: adding a main effect of age to the models sharply curtails or eliminates the association of self-assessed health with mortality.

Only in one age/sex group do we see the hypothesized effect. In a sense, however, this is the group for which the public health significance of the findings is potentially greatest, since male mortality in this age group greatly exceeds female mortality, and since most deaths which occurred in this group could be considered premature and conceivably preventable. The limited findings in this study may provide some clue to the origins and meaning of these subjective perceptions. One might speculate that the apparently accurate predictions of survival made by the middle-aged male respondents in this study could be derived in part from a knowledge of their family's history. Having had a close family member who suffered a premature death from heart disease or cancer could lend ominous significance to symptoms such as a persistent cough or a mildly high cholesterol level which might appear unimportant to someone from a long-lived family. One might also observe that the strength of the association in this age group underscores the fact that, in our society, "excellent" health is the norm for the nonelderly, and any deviation from the norm indicates a potentially serious risk.

Even in comparison with the urban and nonelderly Alameda County sample with which it has more in common, however, the NHANES-I sample is diffuse and geographically unbounded. It encompasses the complex ethnic and cultural diversity of the nation. The cognitive processes by which individuals make global judgments about internal states are by no means well understood, but there is strong evidence that they somewhat consistently vary from one social, ethnic, or cultural setting to another.[14–17] Self-assessments appear to be made by some process of reference-group comparison, in which the individual chooses an appropriate group and judges the state of his or her own health by the health levels typical of the group in question. In other words, these judgments have no absolute reference point. The national sample in a sense removes these judgments from the cultural and community contexts in which they were formed, causing the variation in mortality to be explained primarily by the more objective and also more diverse physical health, demographic and health behavior factors. This analysis should remind us that demographic and health risk behavior factors remain potent predictors of mortality.

Self-assessments of health should continue to be of interest as mortality predictors, not only within homogeneous community samples, but also in large national samples where their impact appears more limited. Self-ratings of health are inexpensive, easy to obtain, and already available in nearly all health surveys. Even if they act as sensitive indicators of mortality risk only for certain subgroups in the population, they should remain valuable tools for health researchers.

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Lowell Levin to Edit New International Journal

Lowell S. Levin, EdD, MPH, professor of public health at the Yale University School of Medicine, has been named editor of the new *International Journal of Iatrogenic Complications*. The quarterly journal, associated with the Copenhagen-based International Society for the Prevention of Iatrogenic Diseases, will begin publishing in February 1991.

Dr. Levin explains that although "iatrogenic" translates literally as "physician-produced," the study of such problems has come to emcompass the negative consequences brought on by any segment of the increasingly complex health care system. When a part of that system harms a patient either by accident, negligence or imcompetence, the ramifications go beyond the medical, to the legal, social and economic. Hence, each article submitted to the new periodical will be reviewed by a jury of three readers from various disciplines, rather than the two readers used by many journals. In addition to physicians, the reviewer roster will include attorneys, economists, social scientists, epidemiologists and others, drawn from a broad geopolitical spectrum.

Dr. Levin is an internationally recognized authority in the field of self-care, which emphasizes education to empower the public in health matters and to prevent illness. He has helped pioneer this concept in both the developed and developing worlds, and has served on various World Health Organization (WHO) committees, including the WHO Committee on Information and Education for Health.

Scholars or other researchers who would like to submit articles to the *International Journal of Iatrogenic Complications* may contact Dr. Levin at (203) 785-2849.

The Economic Impact of Injuries: A Major Source of Medical Costs

LINDA C. HARLAN, PHD, WILLIAM R. HARLAN, MD, AND P. ELLEN PARSONS, PHD

Abstract: Data from the 1980 National Medical Care Utilization and Expenditure Survey were analyzed to place the costs for injuries in the context of all medical costs and to describe the distribution by demographic and diagnostic groups. For the non-institutionalized population, injuries, which include intentional and unintentional, were the second leading cause of direct medical costs, accounting for $16,745 million in medical care expenditures and a major contributor to work loss and disability in the US. For the working-age population (17–64 years) injuries were the leading cost category ($11,341 million) and the third most costly category for persons 65 years of age and over ($3,479 million). The preponderance of costs were attributable to hospital-based care. Direct medical costs were disproportionately greater for males, White and other persons, and for those with household incomes less than $5,000. Injury morbidity also accounts for major indirect costs. Fractures accounted for the highest direct medical costs, greatest per capita charges (based on those with charges), and largest number of restricted activity days. These national estimates document the economic importance of injuries and direct public attention to policy imperatives related to research and prevention. (*Am J Public Health* 1990; 80:453–459.)

Introduction

Injuries are the most common cause of death in the United States for persons under the age of 44.[1] In 1986, unintentional injuries ranked as the leading cause of years of potential life lost before age 65.[2] This consideration and the high incidence of injuries and consequent disability have directed public attention to this problem.[1-3] Despite the frequency of accidental injury, there is a relative paucity of national data on medical costs and morbidity. There appears to be a major disparity between health costs (both direct and indirect) and research investment for prevention and management.[1] However, valid estimates of the national economic impact have not been available until recently[4,5] and these total estimates do not provide a full understanding of the impact of accidents and injuries in various age-sex groups. Information on direct medical costs and indirect costs from disability could provide an economic perspective that would buttress recommendations for greater public investment in research in this field.[1] Moreover, the distribution of costs across demographic and diagnostic categories affords a background understanding of utilization that could lead to focused efforts at prevention and cost containment.

To provide a picture of the national expenditures for injuries, we have analyzed data from the 1980 National Medical Care Utilization and Expenditure Survey (NMCUES). This survey provides the most recent national data on the costs of injuries, which include both intentional and unintentional injuries.

Methods

NMCUES, national survey of health expenditures and use of personal health services, was conducted by the National Center for Health Statistics and the Health Care Financing Administration during 1980 on a national sample of the civilian, non-institutionalized population of the United States. The survey included persons living in 6,600 households. The sample design was a multi-stage probability sample drawn from 135 primary sampling units representing the 50 states and the District of Columbia. Data on 17,123 persons were collected from February 1980 through April 1981. Data were analyzed for Whites and others in one racial category and Blacks in another. The "other" category included American Indians, Alaskan Natives, Asians, and Pacific Islanders. These individuals accounted for approximately 2.5 percent of the "White and other" category. The effect of "others" in the "White and other" category would be expected to be very small. Approximately 7 percent of the NMCUES sample reported themselves as Hispanic. The sample weight was constructed based on race, not ethnicity. The Hispanics are distributed across the racial categories, most being in the White racial category. The estimates obtained if Hispanics were analyzed separately would be non-representative.

Interviews were conducted with each household at approximately three-month intervals. The first two interviews were conducted during a personal household visit by an interviewer; the next two were conducted by telephone; and the final interview was a personal household visit. At the first interview, the respondent was given a diary and asked to record occurrences, conditions, and functional impairments. In addition, the respondent was to record all charges for illnesses and injuries irrespective of who paid these charges, i.e., Medicare, Medicaid, private insurance, or out-of-pocket expenses. Subsequent telephone and personal interviews focused on these diaries. The final response rate was 87.9 percent when attrition over the course of interviewing was taken into account. Detailed information on the survey design, sampling bias, instruments, and statistical analyses are available in several monographs.[5-8]

The public use files of NMCUES were used for these analyses after edits were performed as outlined in the NCHS methodological monograph.[7] Conditions were coded using the International Classification of Diseases, ninth revision, ICD-9[9] and special NCHS codes[10] and grouped into 18 broad diagnostic categories. This report focuses on the "injuries and poisonings" category. In this report, these are referred to as "injuries." The injury category comprised the diagnostic groupings and the relevant ICD codes shown in the Appendix.[9] Both intentional and unintentional injuries are included in these analyses. It was not possible to determine from the data the cause of the injury.

Direct costs for these analyses were defined as charges incurred and reported for inpatient and outpatient care,

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physician and allied health services, prescribed medication and selected medical equipment and other services. The reports of costs were not verified so it is likely that there was an underreporting of actual charges. Some groups probably underreported their charges more than others. Individuals covered by Medicaid, General Assistance or Veterans Administration benefits may not be informed by the payer of the actual charges and may thereby underestimate charges more than other groups.[5] In addition, these data include information only on civilian, non-institutionalized individuals. Costs related to individuals in hospitals, nursing homes, and other institutions or the military would not be represented here.

Disability attributable to these diagnostic groups was categorized into restricted activity (usual activity restricted by illness), bed disability (confined to bed more than half a day), and work loss (lost work because of illness). Restricted-activity days is the most inclusive category and include bed-disability days. Bed-disability days include all hospital days, even if a bed was not occupied. The complex sample design, sampling and respondent biases, and entry or attrition from the survey during the survey year were taken into account in the analysis using procedures described in a methodological monograph.[7] Use of these procedures permits national estimates to be made.

Results

Direct Costs

Direct medical costs for injuries comprised the second largest source of expenditures for medical care in the United States during 1980 (Figure 1). The estimated $16.8 billion for injuries represents 12 percent of all direct costs. This amount was exceeded only by an estimated $20 billion in charges for diseases of the circulatory system.[5]

The distribution of medical costs for the four leading condition categories is presented by age groups in Figure 2. Injury and poisoning ranked among the top cost categories for three of the four age groups (<17 years, 17 to 44 years, and >65 years). Injury costs were the greatest ($9 billion) for young adults (17 to 44 years). This diagnostic category was the third largest contributor to medical costs for older Americans (65 years and over) and was exceeded only by costs related to circulatory conditions and neoplasms. When only the working-age population (17–64 years) was considered, full-time or part-time workers had injuries and poisonings as their major source of medical costs. In contrast, persons not employed had greater charges from other diagnostic categories, generally chronic diseases.[6]

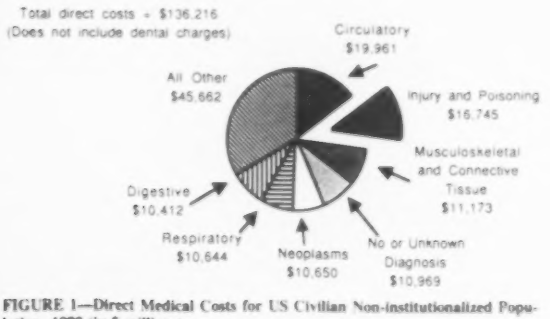

FIGURE 1—Direct Medical Costs for US Civilian Non-institutionalized Population, 1980 (in $ millions)
SOURCE: National Medical Care Utilization and Expenditure Survey

The proportion of medical costs attributable to injuries are presented by demographic characteristics in Figure 3. Medical costs for injuries are presented as the proportion of total direct medical costs for each referent group; the denominator is the total direct cost for all conditions for that group. Injuries accounted for a greater proportion of medical costs for males than for females. White and other persons also had proportionately greater medical costs attributable to injuries than did Black persons. The distribution of injury costs across income groups indicates that persons with family incomes less than $5,000 had a greater proportion of medical costs attributable to injuries than did those in the other income groups. The lower income group (<$5,000) comprised 7.3 percent of the total population, but their total injury costs accounted for 10.3 percent of all injury costs. Thus, the lowest income group had disproportionately greater costs for injuries and disproportionately higher health care expenditures.

The injury category was separated into 11 diagnostic groups which followed the ICD-9 diagnostic categories. The direct costs were determined for each of these groups. The costs attributable to each diagnostic group are presented in Table 1, as are the distributions by age group, sex, and race. Fractures, which includes skull fractures, were responsible for the greatest expenditures and generated $6.4 billion in charges or almost 40 percent of total costs for injuries. The early treatment of trauma ($2.6 billion) and management of the late effects of trauma ($1.7 billion) were also major sources of costs. Sprains and dislocations, wounds (including blood vessel injuries) and complications of medical and surgical care were the only other diagnostic groups leading to over $1 billion per year.

There were important variations in diagnostic group costs by age, sex, and race (Table 1). The costs attributable to fractures, the most costly category, varied by age group from 27 percent (<17 years) to 43 percent (>65 years). Fractures accounted for more costs for females (42 percent) than for males (35 percent). Black persons had 44 percent of injury costs attributable to fractures in contrast to 38 percent for White and other persons. The costs associated with early treatment of trauma were highest for the 45 to 64 year old group, for females, and for Black persons. This grouping includes multiple traumatic injuries, but excludes specific traumatic diagnoses such as fractures and burns. Costs associated with later effects of trauma were greatest for those over age 65 years, for females, and for White and other persons. Sprains and dislocations generated relatively low charges for those over 65 and for females. Wounds accounted for 21 percent of costs for those under 17 years and wounds and fractures together accounted for almost half (48 percent) of costs in this age group. Poisoning costs were overwhelmingly associated with females and were highest in the 17 to 64 year old group. Costs for burns were higher for males than for females and for those 45 to 64 years.

The immediate cause of the injury (e.g., vehicular accident, fall, attempted suicide, etc.) and contributing causes (e.g., alcohol ingestion, speeding, etc.) were not recorded in NMCUES. Therefore, only indirect inferences can be made about causation. The distribution of injuries suggest a relatively greater cost impact of trauma, i.e., sprains, wounds and head injuries, from leisure and work activity on young males and greater cost impact on older women from fractures and late sequelae of trauma.

These aggregate direct costs do not disclose the costs on a per occurrence or per person basis or the allocation of costs

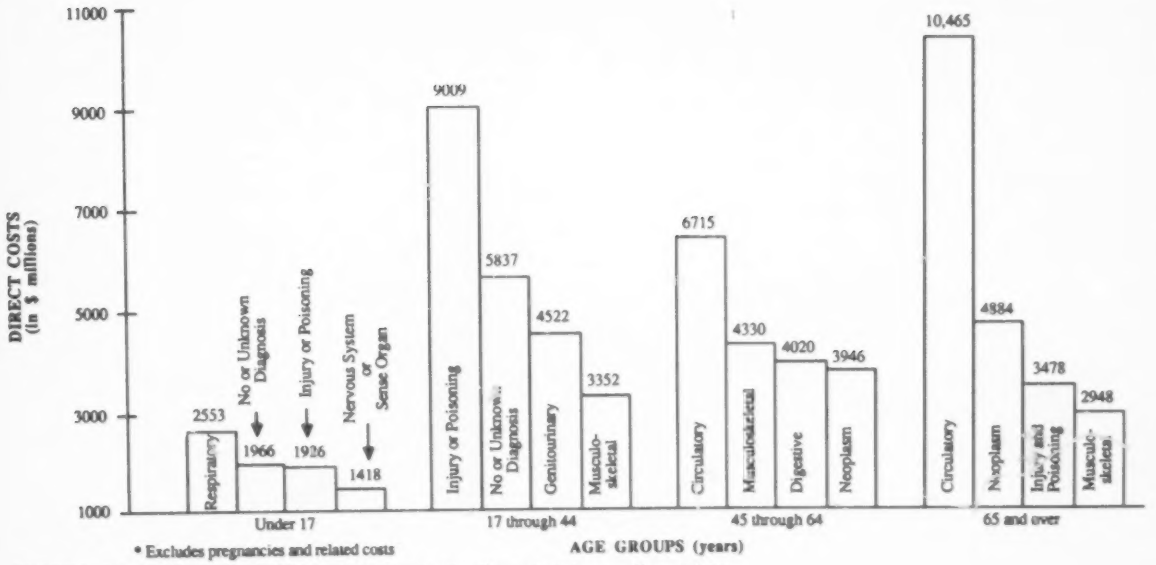

FIGURE 2—Direct Medical Costs for All Persons by Four Leading Diagnostic Categories, Cost, and Age

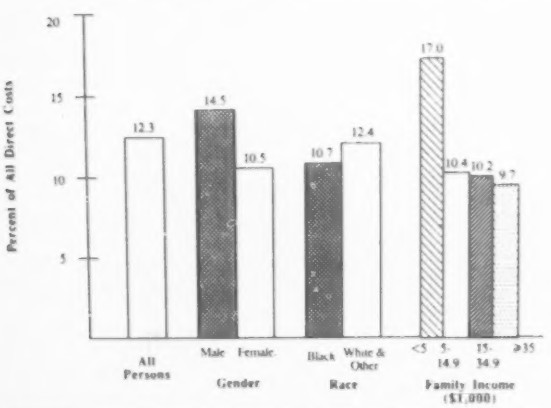

FIGURE 3—Proportion of Total Direct Medical Costs Attributable to Injuries by Demographic Groups
SOURCE: National Medical Care Utilization and Expenditure Survey

across components of care. Therefore, the per capita charges were determined for the diagnostic categories and divided into the charges for four types of health services: hospital care (including emergency room), ambulatory medical care (including outpatient physician charges), hospital physician charges, and medication charges (Table 2). The per capita charges are based on mean cost per individual having charges under each diagnostic and health service category. On a per capita basis, the highest charges were for management of the late effects of trauma and for fractures. For the other diagnostic groups, the rank order of per capita charges differed somewhat from the rank order of total direct charges (Table 1). Sprains had relatively low per capita costs but high aggregate costs because of greater frequency. On the other hand, burns had high per capita costs but low aggregate costs. The most striking finding regarding per capita charges is that the preponderance of charges for each diagnostic code (except sensory impairment) were generated by hospital care, which includes emergency room services. However, for the diagnostic categories of foreign bodies, burns, and sensory impairment, the estimates are unstable because fewer than 10 individuals in NMCUES were hospitalized in each category during the survey. The next highest category of charges were for physicians' hospital services. Only small charges ranging from $12 to $71 were reported for medications and from $57 to $224 for ambulatory care. Therefore, over 90 percent of per capita costs for most injury categories were related to hospital-based care.

Disability

Disability and work loss are indirect morbidity costs of medical conditions. Although total disability, work loss, and restricted-activity days were collected on each individual, a single day could be assigned to several conditions. Therefore, days lost for each condition were calculated by multiplying the weighted proportion of days lost due to the condition of interest by the total days lost. A national estimate of 932,300 person years of restricted activity and 292,300 person years of bed disability were attributable to injuries. These days represent about 10 percent of days attributable to all conditions during 1980 and exceed the estimates of disability for circulatory conditions for the same period.[11]

Restricted-activity and bed-disability days are presented for the diagnostic groups in Figure 4. Fractures were responsible for the greatest mean number of restricted-activity and bed-disability days at all ages. For persons age 65 years and older, the mean number of days approximately doubles. Similarly, two- to three-fold greater disability occurred for older adults with head and internal injuries, late effects of trauma, and complications of medical and surgical care. For sprains, burns, and early effects of trauma, there were no differences by age. On the other hand, poisonings, sensory impairment, and wounds had lower mean days of restricted activity or bed disability for older persons, but the frequency

TABLE 1—Direct Medical Costs for Diagnostic Codes by Age Group, Gender and Race: National Medical Care Utilization and Expenditure Survey, 1980

Diagnostic Category	Total Charges* (in $millions)		Age Group				Gender		Race	
	$	(%)	<17	17–44	45–64	65+	Male	Female	White & Other	Black
Fractures	$ 6421	(38.3)	524	3678	709	1510	3071	3350	5763	658
Early Complications of Trauma	2602	(15.5)	280	1171	510	641	1103	1499	2317	285
Late Effects of Trauma	1694	(10.1)	222	730	103	638	695	998	1668	26
Sprains, Dislocations	1599	(9.5)	240	944	295	120	1048	551	1438	161
Wounds (including blood vessels)	1565	(9.3)	406	849	150	160	1089	476	1456	109
Iatrogenic†	1152	(6.9)	87	586	185	295	634	519	1104	49
Head Injuries	816	(4.9)	53	680	55	29	633	183	692	124
Poisonings	383	(2.3)	30	226	80	47	26	357	348	35
Burns	281	(1.7)	36	51	190	4	225	55	264	17
Foreign Body Entry	132	(0.8)	26	54	30	23	70	62	107	25
Impairment of Senses	100	(0.6)	22	40	25	12	80	19	96	4
Total**	16745	(99.9)	1926	9009	2332	3479	8674	8069	15253	1493
(proportion of Total Costs)			(11.5)	(53.8)	(13.9)	(20.8)	(51.8)	(48.2)	(91.1)	(8.9)

*Does not include charges for dental services.
**Do not necessarily add to $16745 due to rounding.
†Complications of medical and surgical care.

TABLE 2—Per Capita Medical Charge for Individuals with Charges by Diagnostic Codes, National Medical Care Utilization and Expenditure Survey, 1980

Diagnostic Category	Hospital	Ambulatory	Hospital Physician	Prescription & Other Service
Fractures	4548	194	986	39
Late Effects of Trauma	3818	104	1005	33
Early Complications of Trauma	2198	87	112	23
Iatrogenic†	2138	113	1017	27
Burns	2104	67	985	18
Poisonings	1763	57	196	12
Wounds (including blood vessels)	1279	90	776	16
Sprains, Dislocations	1271	102	483	18
Head Injuries	1260	224	449	53
Foreign Body Entry	669	74	263	17
Impairment of Senses	205	175	1068	71
All Injuries	2879	123	817	27

†Complications of medical and surgical care.

was low and the estimates unstable. In general, bed-disability days comprise less than half of restricted activity except for those over age 65 years who had fractures, head and internal injuries, and late effects of trauma. Although not reported here, the distributions of disability by gender and race were similar to the distributions of direct and per capita costs.

Work-loss days were estimated for the working age-population (17 to 64 years), both men and women. The total work loss attributable to injuries was 247,900 person years. This represents 14 percent of all work-loss days attributable to illness. The mean work-loss days by diagnostic groups are plotted in Figure 5. Fractures were responsible for the highest mean work-loss days. Sensory impairment, head and internal injuries, sprains, burns, and the early and late effects of trauma are the other injuries that resulted in a mean work loss of five days or more. There were important differences between men and women for several of the condition groups. Men had more work-loss days than women from all conditions except sensory impairment and fractures. Although not shown, there were no important trends by race, except for greater mean work-loss days for Black persons attributable to early and late treatment of trauma.

Discussion

These analyses confirm that injuries are a major source of medical costs, both direct medical charges and indirect costs of illness, as measured by disability days. Injuries are the second leading cause for direct medical costs in the civilian, non-institutionalized population. This cost burden is spread across all ages. Injuries are the leading contributor to medical costs for persons 17 to 44 years of age. Importantly, injuries also account for a considerable proportion of medical costs for Americans, age 65 years and over. For this older group, only circulatory conditions and neoplasms are responsible for higher costs. The general perception that medical expenses in older, non-institutionalized persons result primarily from chronic diseases and infections contrasts with these national findings.

These survey data underestimate the actual economic impact on the elderly because NMCUES did not survey institutionalized persons who represent an important proportion (about 10 percent) of those over age 65 years. As such, NMCUES does not address the cost burden related to institutionalized individuals. Hodgson and Kopstein have reported that the costs for this group are different than those living in the community.[4] Using a variety of data sources, they estimated that per capita expenditures for the elderly institutionalized and non-institutionalized population rank injuries as the 4th and 8th, respectively for women and men. These authors also found that although only 11 percent of the population is over the age of 65, they consume about 31 percent of the personal health care dollars. Therefore, the total cost estimates for the US population would be greater than estimated from this survey. Rice, *et al*, reported that when institutionalized and non-institutionalized elderly are included that diseases of the circulatory system account for 31 percent of the total direct costs.[12] Because the elderly contribute disproportionately to total costs and because chronic disease is responsible for a greater proportion of the cost when institutionalized persons are included, injuries would account for a somewhat lower proportion of medical costs for the elderly.

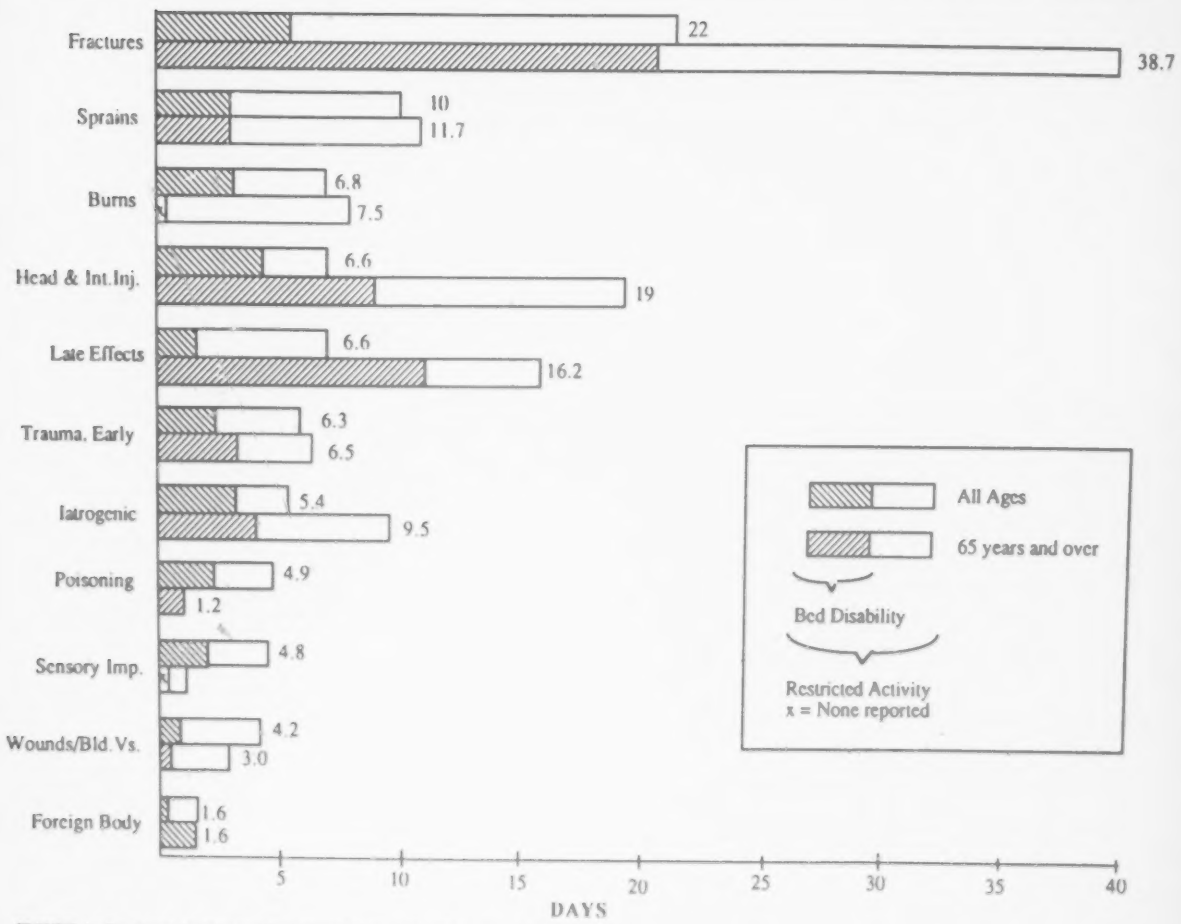

FIGURE 4—Restricted Activity and Bed Disability Days for All Ages and for Age 65+

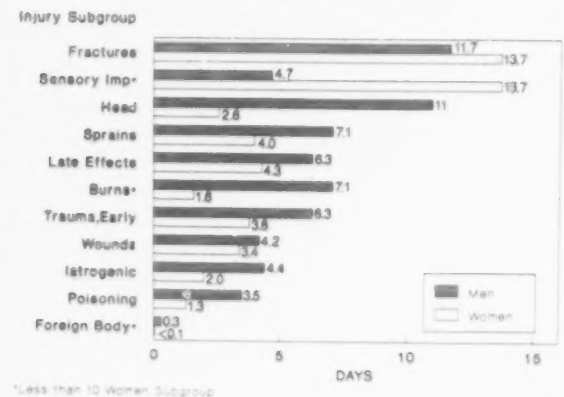

FIGURE 5—Mean Work Loss Days by Injury Subgroup and Gender

The burden of medical costs from injuries are also disproportionately greater for males, persons other than Black, and those with family incomes less than $5,000 (in 1980 dollars). Each of these three groups reports a greater proportion of health care charges attributable to acute injuries and their sequelae than do their counterparts. This maldistribution of costs may reflect differential exposure to potentially injurious situations in the workplace or community. Medical costs for the category of "White and other" might be slightly higher because of the inclusion of "others" in the category. However, the sample weights were computed for "Whites and others" as one category. The "others" category is small and therefore the effect on this category would be minimal.

The economic impact and the demographic pattern of expenditures are concordant with epidemiologic estimates of the health impact of injuries. The potential years of preretirement work life lost as a result of premature mortality from intentional and unintentional injuries was over 4 million years in 1982. This toll was roughly twice the years of potential work life lost from either neoplasms or heart disease.[2] In the National Household Interview Survey of 1980–81, a very high incidence of intentional and unintentional injuries were reported; about 75 million injuries were

reported by 70 million persons.[13] The injury rates were higher for males than for females (40 million versus 30 million), with the greatest number (21 million) occurring in the 17 to 44 year old age group. Both the NHIS and the NMCUES agree on the high occurrence rate in the US population and on the gender and age distributions. These morbidity estimates also correspond to the patterns of national mortality from injuries.[14] Therefore, the mortality, morbidity, and cost data confirm the remarkable impact of injuries.

The economic burden of disease includes the loss of economic productivity to society and income resulting from work loss. These measures of lost productivity were high, reflecting the frequent occurrence of injuries among the younger, working-age population. Injuries accounted for approximately 10 percent of all restricted-activity and bed-disability days during the survey year and about 17 percent of all work-loss days. These estimates exceed the disability and work loss attributable to acute respiratory conditions, which are the most common acute conditions in the general population.[15] These findings agree generally with data from the larger National Health Interview Survey conducted in 1980–81.[13] These findings do not address additional mortality costs to society. Depending on the discount rate used, the mortality costs from injuries are reported to be six to eight times the morbidity costs.[12]

There are considerable social costs in terms of work loss, restricted activity, and bed disability; these costs are distributed across the entire age range. The duration of disability and the mean direct costs are greater for older persons, but the impact of total direct costs and work loss is nevertheless severe for younger adults. MacKenzie, *et al*, examined the functional recovery and medical costs of trauma in patients admitted to two trauma centers and discharged to extended care or rehabilitation facilities or home.[16] Of 262 individuals who were working before their accident, only 57 percent had returned to full-time work within one year.

Examination of total and per capita charges for diagnostic groupings provides further insights into the high costs of injuries. Hospital-based services, including physician fees, dominated the costs for all injury groups. This close linkage of injury care to hospital services indicates that costs for these conditions will parallel the general inflation of hospital operating costs. Because those with the least ability to pay—younger persons and those with low family incomes—have disproportionately higher costs for injuries, it is likely that increasing financial pressures may force many hospitals, and especially those with a large indigent or uninsured clientele, to reassess their ability to provide injury services.[17] A number of hospitals have already terminated emergency care services as a result of cost considerations. In a study of nonaccidental injuries, assaults and suicides, 2,451 patients were admitted to a trauma center.[18] The average charges for the victims of assaults were over $5,000 per patient while the suicide victim's charges were nearly $13,000. Six months after the study had been completed only about 70 percent of the hospital charges had been reimbursed. The authors noted that even if all of the hospital bills were covered the profit margin would only be 1 percent.

The charges associated with specific diagnoses identify conditions and characteristics of persons responsible for generating costs. Fractures accounted for the greatest total direct costs, and per capita costs were also high for this diagnosis. Moreover, fractures were responsible for greater mean disability- and work-loss days than were other diagnoses. Although fractures occurred more frequently in young adults (17 to 44 years), the cost and disability impact was greatest for those over age 65 years. However, fractures occurring in the older population are more likely to result from osteoporosis and hip fractures.[19,20] The diminished resiliency, more frequent requirement for surgical management, and the complicating comorbidity of chronic disease contribute to the greater burden for older persons. For similar reasons, head injuries and late effects of trauma also had a greater effect on older persons. These findings argue that injury research and preventive efforts should not be directed exclusively to the younger adult and employed population, who are generally considered the primary target for these efforts. Prevention and more efficient management of these injuries in the elderly could have considerable economic and health impact.

For conditions other than fractures, the aggregate charges and per capita charges were frequently discordant. For sprains, the per capita charges were relatively low but the high incidence in the population contributed to relatively high total charges. On the other hand, burns occurred less frequently but had higher per capita charges.

The diversity of the conditions included as injuries and the differences among them with respect to per capita costs and disability have implications for public policy. The most effective medical and cost reduction strategy would be prevention. Prevention would affect not only the less common and more expensive conditions but also the total costs of less expensive and commonly occurring conditions by decreasing the incidence of both. Prevention requires specification of the site and circumstances of injury. In NMCUES, there was no inquiry about the causes for the injuries or the sites of occurrence. However, in the contemporaneous National Health Interview Survey, vehicular accidents, industrial accidents, and the home were the most frequent sites of injury.[13] Examining type of accident or manner of injury as the cause of death for 1981, motor vehicle accidents were by far the leading cause of death,[21] followed by falls, fires, and drowning. Major cost containment strategies should be directed toward prevention at all potential sites as well as toward cost efficient urgent and emergency care. These data from NMCUES document the cost of post-emergency care for fractures, trauma, and burns and indicate the potential value of developing more cost-efficient care.

The remarkable impact of injuries raises important public policy issues. Compared with the considerable attention directed toward heart disease, cancer, and recently AIDS (acquired immunodeficiency syndrome), the health professionals and the public appear less concerned with injury research and prevention. The federal research investment in prevention and treatment of injuries was estimated to be $112 million in 1983 or about one-tenth of the research commitment to cancer and one-sixth of that to heart disease and stroke.[1] Moreover, only about one-fourth of federal research investment in injuries was directed toward prevention with the remainder devoted to injury mechanisms, acute care, and rehabilitation.[1] In the context of high medical care costs, this research investment is extremely small. The national costs for injury care exceed those for cancer and are nearly as great as for heart disease. The years of life lost and indirect costs from disability and work loss exceed those for either of these disease categories. The extremely small research commitment to injuries contrasts sharply with the commitment to cancer and heart research. From the perspective of research investment as a proportion of health care

costs, injury had less than 1 percent (actually 0.7 percent) of the care cost invested in research. The comparable proportions for cancer and heart disease were 9 percent and 3 percent, respectively.[1]

Several philosophical, political, and financial reasons can be cited for the public oversight. Importantly, injuries may still be considered to result from random and unalterable events and thus be unpredictable and unpreventable. Even the relatively sparse epidemiologic data from investigation of accidents, injuries, and outcomes indicate that this is not true.[22] Further investigation should improve prediction and afford major decreases in injury rates and injury costs.[23] The lack of research monies probably relates to the absence of a focus for organizing a professional and public constituency. Without a focus of responsibility for defining and reporting injuries, funding research, and monitoring change, there is little political action to reassess priorities and generate new and useful knowledge. From the economic as well as the health perspective, the need warrants greater attention.

APPENDIX
Diagnostic Categories and ICD Codes for Accidents and Injuries

Diagnostic Category	ICD Code
Fractures	800–829
Sprains, strains, dislocations	830–839 840–848
Intracranial, internal injury	850–869, 950–957 special NCHS codes
Open wounds, injury to blood vessels	870–879, 880–887, 890–899, 900–904
Late effects of injuries, poisonings, toxic effects of external cause	905–909
Other early complication of trauma	910–919, 920–924 925–929, 958–959, 990–995
Effects of foreign body entering through orifice	930–939
Burns	940–949
Poisonings, toxic effects	960–979, 980–989
Complications of medical, surgical care	996–999
Partial impairment of senses or other special impairment from accident or injury	special NCHS codes for sensory impairment

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Public Health Briefs

Evaluation of Active versus Passive AIDS Surveillance in Oregon

Steven K. Modesitt, RN, MPH, Sharon Hulman, RN, BSN, and David Fleming, MD

Abstract: We searched for unreported AIDS cases in Oregon through death certificate and medical record review, and enhanced infection control practitioner and physician surveillance. Fifty-six AIDS cases diagnosed between February 1, 1986 and January 31, 1987 were reported passively. Twenty-nine additional cases diagnosed during this time were retrospectively identified by active methods. Ninety percent of those 29 cases were diagnosed by physicians and cared for in hospitals that had previously reported cases. Completeness of reporting under the passive system was 64 percent. (*Am J Public Health* 1990; 80:463–464.)

Introduction

Since the acquired immunodeficiency syndrome (AIDS) was first described in 1981, surveillance for AIDS cases has served to define the modes of transmission and to characterize the populations most affected. To date, there have been little data to assess the need for these active surveillance systems, that seek AIDS case reports through a variety of methods rather than passively depending on physician reporting. This study assesses the need for active AIDS surveillance systems. The HIV (human immunodeficiency virus) program of the Oregon Health Division initiated prospective active surveillance for AIDS in February 1987. We applied the methods used for active surveillance to retrospectively determine the number and characteristics of AIDS cases that had gone unreported under the previous passive system. We report here the results of that evaluation.

Methods

We used four active surveillance methods to identify unreported cases diagnosed between February 1, 1986 to January 31, 1987, the year before active surveillance began.

- All physicians in Oregon were sent a letter reviewing AIDS reporting requirements and soliciting case reports. Physicians who had previously reported at least one AIDS case were provided with a list of all these cases for review and asked to report additional unreported cases under their care.
- Infection control practitioners at hospitals where previous AIDS cases had been diagnosed were asked to compile a list of previous AIDS admissions and maintain a log of subsequent admissions for comparison with our central AIDS registry. Those from hospitals without reported cases were asked every six months to confirm that no AIDS-related admissions had occurred.
- At all hospitals statewide where at least one case had been diagnosed, medical records of all potential AIDS admissions were reviewed. Admissions were pulled if they contained any of five International Classification of Diseases, Volume 9 (ICD-9) codes (042, 043, 279.19, 136.3, 173.9) previously shown to be reliable in identifying persons with an AIDS diagnosis.[1] Cases were matched with reported cases in the central registry and physicians of possible unreported cases were queried.
- Death certificates designating AIDS or listing a cause of death potentially AIDS-related (including all deaths related to known opportunistic conditions and deaths from infectious or immunosuppressive causes in persons under age 60) were reviewed. Physicians of potential unreported cases were queried.

A case diagnosed during the year before active surveillance began was considered to be discovered by **active surveillance** if it was reported by any reporting source after the start of active surveillance and more than 6 months after diagnosis. All other cases were classified as reported by passive surveillance. All cases met the 1985 CDC surveillance case definition.[2]

Results

Completeness of Reporting

Fifty-six AIDS cases diagnosed between February 1, 1986 and January 31, 1987 were reported under the passive system. Using active surveillance methods an additional 29 cases diagnosed during this time were found retrospectively. The completeness of reporting before active surveillance began was 64 percent (95% confidence interval = 54, 74). For cases reported under the passive system, the median time between diagnosis and report, or lag time, was one month. Lag time ranged from 7 to 19 months for cases found by active surveillance only (median = eight months).

Characteristics of Cases and Reporting Sources

Cases reported by passive surveillance did not significantly differ from those identified by active surveillance by age, race, county of residence, risk factors, disease at diagnosis or mortality status at time of report, although our power to detect significant differences was limited by the relatively small sample size (Table 1).

Characteristics of physicians who reported cases by passive surveillance did not differ from physicians whose cases were identified under active surveillance by specialty,

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TABLE 1—Characteristics of Cases Reported by Passive or Active Surveillance

Characteristics	Passive (n = 56)		Active (n = 29)	
Age (mean)	35.6yrs		35.2yrs	
Race	No.	%	No.	%
White	53	94.6	28	96.6
Non-White	3	5.4	1	3.4
County of residence				
Multnomah (urban Portland)	38	67.9	23	79.3
Other	18	32.1	6	20.7
Mortality status				
Alive at report	38	67.9	19	65.5
Dead at report	18	32.1	10	40.0
Disease at diagnosis				
PCP	33	59.1	20	68.5
Other disease w/o PCP	14	25.2	3	10.3
KS alone	9	16.1	6	20.4
Mode of transmission				
Homosexual/bisexual men	45	80.4	22	75.9
Homosexual/bisexual IVDA	5	8.9	5	17.2
Hemophiliac (adult)	3	5.4	1	4.0
Other	3	5.4	1	4.0

PCP = Pneumonocystis carinii pneumonia; KS = Kaposi's sarcoma; IVDA = Intravenous Drug Abuse

TABLE 2—Characteristics of Physicians Reporting by Passive or Active Surveillance

Physician Characteristics	Passive		Active	
Specialty	No.	%	No.	%
Internal Medicine	29	51.8	10	36.0
Infectious Disease	10	17.9	13	44.8
Allergy and Immunology	9	16.1	1	3.4
Other	6	10.7	5	17.2
Unknown	2	3.6	0	0
City of practice				
Portland	39	69.6	24	82.8
Not Portland	17	30.4	5	17.2
Previous AIDS Report				
Yes	42	75.0	26	89.7
No	13	23.2	3	10.3
Unknown	1	1.8	0	0

city of practice, or previous reporting history. Ninety percent (26/29) of cases identified by active surveillance were cared for by physicians who had reported at least one case before the start of active surveillance.

Characteristics of hospitals from which cases were reported passively did not differ from hospitals where cases were identified under active surveillance by previous reporting history, bed size, or location. Ninety percent (26/29) of cases identified by active surveillance were diagnosed in hospitals that had cared for at least one case before the start of active surveillance.

Efficacy of Active Surveillance Components

We compared the relative effectiveness of each component of active surveillance in identifying previously unreported cases. The initial reporting source for cases identified actively was physicians for 14 percent, infection control practitioners 31 percent, medical record review 31 percent, and death certificate review 24 percent. We estimated the completeness of the surveillance system using the capture-recapture methodology (Chandra Sekar and Deming method).[3] Our surveillance system was divided into two independent reporting components: unsolicited (physician or infection control practitioner reporting) and solicited (medical record review or death certificate review). The percentage of cases found by unsolicited means was 80 percent. The percentage of solicited reports was 95 percent. Seventy-seven percent of cases were found by both methods. The estimated completeness of reporting was 99 percent of the estimated case population.

Discussion

Relative to active AIDS surveillance, the completeness of passive AIDS surveillance in Oregon in 1986–87 was only 64 percent, similar to preliminary findings from South Carolina[4] and less than an earlier New York study.[5] Passive AIDS surveillance, even in relatively low incidence areas, is incomplete. Projections based on passive surveillance would underestimate future incidence proportionately and could result in poor public health planning and resource distribution decisions.

Because almost all cases during this time in Oregon were occurring in urban gay and bisexual men, we recognized at the outset that we would not be able to assess differences in reporting based on case risk factor.

Infection control practitioners, medical record review, and death certificate review were all useful in identifying cases unreported more than 90 days after diagnosis. Each of the multiple reporting components independently identified cases that would otherwise have gone unreported.

Physicians and hospitals with previous reporting history appear to be the reservoir for the largest number of unreported cases and are the best targets for enhanced surveillance activities. As the epidemic continues, the ability to maintain effective active surveillance will include using resources efficiently, combating "reporting fatigue," and assuring timely reporting as clinical practice shifts diagnosis to outpatient settings. As the AIDS epidemic progresses and physicians and hospitals gain more experience with this disease, active surveillance may become more rather than less important.

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The Prevalence of High-Risk Sexual Behavior in Male Intravenous Drug Users with Steady Female Partners

Diane K. Lewis, PhD, MPH, John K. Watters, PhD, and Patricia Case, BA

Abstract: A sample of 149 (70 White, 79 Black) male intravenous drug users with steady female sexual partners was interviewed in 1987, in treatment and street settings, about sexual practices during the previous five years. Eighty-three percent had multiple partners, 15 percent reported male sexual contact, 38 percent reported heterosexual anal intercourse, and 73 percent never used condoms. Thirty-seven White and 30 Black subjects reported bisexuality or heterosexual anal intercourse or both. (*Am J Public Health* 1990; 80:465–466.)

Introduction

Research on AIDS (acquired immunodeficiency syndrome) prevention among intravenous (IV) drug users has focused primarily on needle sharing,[1,2] with few studies to date on high-risk sexual behavior.[1,3] Since sexual behavior may prove more difficult to change than needle behavior,[2,4,*] more information is needed to design effective AIDS education programs. This paper presents the prevalence of high-risk sexual behavior in a sample of male IV drug users in San Francisco.

Methods

Data on sexual behavior were collected in early 1987 from 149 male IV drug users with stable female sexual partners. The men were a sub-set of a larger study of risk behaviors for human immunodeficiency virus (HIV) transmission among 632 male and female IV drug users. Of the 411 male subjects, 34 had exclusively male sexual contacts during the previous five years and 377 had sexual contact with females during that period. Of these 377, 134 were White, 171 were Black, and 82 were Hispanic or other ethnic backgrounds. Participants were asked: "Do you have a steady sexual partner (or partners)?" If they answered yes, they were asked whether the partner(s) was male, female, or both. Of the 377 males, 201 reported a steady partner or partners; 184 had a steady female partner, 14 had a steady male partner, and three had both a male and a female partner. One White and one Black male reported both a female and a male as steady partners. Information on length of relationship was not obtained. Men with no steady sexual partners, those whose partners were exclusively male, and those of Hispanic or other ethnic backgrounds were not included in this study.

Subjects were recruited from a 21-day methadone clinic and a symptomatic (nonmethadone) clinic, and from three street settings. In-treatment subjects had a documented history of opiate addiction (the methadone clinic), or were identified by staff as having injected drugs during the three weeks prior to admission (the symptomatic clinic). Of those contacted in the two clinics, 86 percent agreed to participate. Out-of-treatment subjects, recruited via word of mouth through a targeted sampling technique,[5] reported needle use within three weeks of interview and showed signs of recent venipuncture. The refusal rate for street subjects is unknown because of the chain-referral recruitment method.

Structured 45 minute interviews were conducted by trained interviewers who questioned subjects about demographic characteristics, medical history, drug use patterns, and AIDS awareness, as well as sexual behavior. Subjects were asked their total number of female sexual partners during the previous five years; if they had sexual contact with males; if they ever engaged in heterosexual anal intercourse, and the percent of time of condom use.

Results

Of the 149 subjects, 70 were White and 79 were Black; less than one-fifth (18 percent) of the subjects were currently married. Age ranged from 20 to 66 years; the median age of Whites was 33.5 years, of Blacks 36 years. Eighty-eight (59 percent) of the men were interviewed out-of-treatment, 27 (39 percent) of the White subjects and 61 (77 percent) of the Black subjects.

High Risk Sexual Activities

Over four-fifths (83 percent) of the men reported more than one female sexual partner during the previous five years, 60 percent had five or more, and 39 percent had 10 or more. Whites had a median of 7.5 partners and Blacks a median of five partners.

Twenty-two (15 percent) of the 149 men reported sexual contact with a male (13 percent of the Whites and 17 percent of the Blacks). White subjects who reported same-sex activity had a median of 20 female partners and three male partners. Black subjects who reported bisexual activity had a median of 10 female and three male partners.

Heterosexual anal intercourse was reported by 56 (38 percent) of the men (46 percent of the Whites and 30 percent of the Blacks). Almost three-fourths (73 percent) of the men said they never used condoms.

Table 1 shows the distribution of subjects engaged in high-risk sex by treatment status and by ethnicity. Twenty-seven of 61 in-treatment subjects and 29 of 88 out-of-treatment subjects reported either bisexuality or heterosexual anal intercourse. In all, 33 White and 23 Black subjects reported either male sexual contact or heterosexual anal intercourse; seven Black and four White subjects engaged in both activities (see Table 1).

The majority of men engaged in high-risk sex reported non-use of condoms (Table 1). Fourteen of 22 men who had male sexual contact, and 41 of 56 men who engaged in

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*Watters JK, Huang KH, Cheng Y-T, Lorvick J, Carlson J: HIV seroepidemiology and behavior change in intravenous drug users: Progress report on the effectiveness of street-based prevention. Presented at the IV International Conference on AIDS, Stockholm, 1988.

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TABLE 1—High-Risk Sexual Behaviors in 149 Intravenous Drug Users According to Treatment Status and Ethnicity

	(1)	(2)	(3)		(4)
	Bisexual	Heterosexual Anal	(1)+(2)	Either Heterosexual Anal or Bisexual	Never Use Condoms
In-Treatment (N)					
Black (18)	1	9	1	8	16
White (43)	3	20	2	19	33*
Total (61)	4	29	3	27	49*
Out-of-Treatment (N)					
Black (61)	12	15	6	15	38
White (27)	6	12	2	14	21
Total (88)	18	27	8	29	59
Total (N)					
Black (79)	13	24	7	23	54
White (70)	9	32	4	33	54
Total (149)	22	56	11	56	108

*One subject with a missing answer.

heterosexual anal intercourse said they never used condoms (data not shown).

Discussion

Overwhelmingly research on IV drug users is conducted in treatment programs.[5,6] In this study, the majority of subjects were recruited out-of-treatment. Because this was not a random sample, the results may not be generalizable. Other limitations are a lack of information on duration of partner relationships and the problem of bias in recalling and accurately portraying past sexual activity.

Due to a lack of comparable studies, it is unknown whether the range and level of sexual activity reported by these subjects is similar to other IV drug users. The sexual risk taking in this sample may be connected with recent increases in cocaine injection and smoking.[7,8] Because cocaine has been linked with heightened sexual activity in some studies,[9,10] the impact of it and other drugs on high-risk sexual behavior merits study.

The numerous sexual partners and the high-risk practices reported by a majority of these IV drug users indicate the potential for widespread secondary transmission of HIV. The difficulty in reducing sexual risk behavior is shown by the high proportion of subjects in this study who reported non-use of condoms, despite out-reach risk reduction programs, initiated in 1986, which included widespread distribution of free condoms to IV drug users in San Francisco.[4,**]

Further research is required on ways to overcome barriers to condom use and to reach heterosexually identified male IV drug users engaged in sexual activity with males. Continued aggressive culturally sensitive out-reach sexual risk reduction strategies are needed, since many IV drug users may not be enrolled in treatment programs.[11–13]

**Watters JK: Preventing human immunodeficiency virus contagion among intravenous drug users: The impact of street-based education on risk-behavior. Presented at the III International Conference on AIDS, Washington, DC, 1987.

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Are Patients Talking to Their Physicians about AIDS?

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Abstract: We conducted a nationwide telephone survey of a random sample of United States adults in summer 1988 (n = 2000, response rate = 75 percent) to find out if physicians were providing education and counseling to the public about AIDS and AIDS prevention. Within the previous five years, 94 percent had seen a physician but only 15 percent had discussed AIDS even though most said they would not object to discussing the topic. AIDS-related conversations are not commonplace in physician's offices and in most cases (72 percent) patients are the initiators of such conversations. (*Am J Public Health* 1990; 80:467–468.)

Introduction

Physicians have been encouraged to discuss AIDS (acquired immunodeficiency syndrome) risk reduction with all of their patients and to take sexual histories to help all patients identify and modify risky behavior;[1,2] physicians are well placed to provide this service, as approximately 76 percent of Americans see a physician annually.[3] Moreover, 60 percent of respondents to a National Center for Health Statistics survey[4] said that they would ask a physician if they wanted specific information about AIDS.

Methods

Telephone interviews were completed with a representative sample of 2000 civilian, non-institutionalized United States adults (75 percent response rate) between July 5 and August 19, 1988. In selecting the sample, the country was divided into two strata by AIDS prevalence and subjects were drawn using a random-digit-dial procedure. There were 1000 respondents from each stratum.

The data reported here are based on weighted estimates for the two strata combined. Given the stratification design effect (the ratio of the variance of design used to that of a simple random sample of the same size), the effective sample size for population estimates was 1320.[5] The confidence intervals in Table 1 reflect this design effect.

Subjects were selected randomly within households. To partially correct for known biases in the selection of respondents resulting from random digit dialing,[6] responses were weighted by the number of persons in the household and the inverse of the number of telephone lines in the household. To correct for small differences between the demographic characteristics of the sample and census estimates, data were also weighted by race, sex, and age.

The interview contained 70 items concerning public perceptions about AIDS and health care. Items about physician counseling are presented in the Appendix. Ninety-four percent of respondents recalled seeing a physician at least once in the past five years; 76 percent of respondents had visited a physician in the past year. Data reported here are from respondents who had seen a physician in the past five years, referred to later in this paper as "patients." Further details of the methods have been reported in earlier papers.[7–9]

TABLE 1—**Percent of Patient Respondents Who Had Discussed AIDS with their Physician by Demographic Characteristics (with 95% confidence intervals)**

	N*	%	95% Confidence interval
Total	1876	15.4	12.9, 17.9
Sex			
Male	865	14.1	11.2, 17.0
Female	1011	16.4	13.6, 19.2
Race			
White (non-Hispanic)	1496	14.5	12.3, 16.7
Black	180	22.2	14.7, 29.7
Age (years)			
18–29	511	17.2	13.2, 21.2
30–49	789	17.5	14.2, 20.8
50+	576	10.8	7.7, 13.9
Income (household per annum)			
< $20,000	478	13.0	9.3, 16.7
$20,000–34,999	535	13.8	10.2, 17.4
$35,000–49,999	320	16.3	11.3, 21.3
$50,000+	220	20.9	14.3, 27.5
AIDS Prevalence in SMSA			
High	943	19.4	16.9, 21.9
Low	933	11.3	9.3, 13.3
What are your chances of getting the AIDS virus?			
High/Medium	96	27.1	16.2, 38.0
Low	660	17.4	13.8, 21.0
None	1090	12.8	10.4, 15.2
Knows HIV-infected person	372	26.9	21.4, 32.4
Does not know HIV-infected person	1474	12.4	10.3, 14.5
Knows risk group member	761	23.4	19.7, 27.1
Does not know risk group member	1050	10.0	7.8, 12.2

*Subtotals which do not add to 1876 exclude "don't know" and "no answer" respondents for those items.

Results

Only 15 percent of patients recalled ever talking to their physicians about AIDS. The demographic groups more likely to have discussed AIDS were young people, Blacks, people with a high household income, or people who lived in an SMSA with high AIDS prevalence (Table 1). In addition, those for whom AIDS was personally most salient, for example those who knew someone with AIDS or HIV (human immunodeficiency virus) infection, were more likely to have talked to their physician about the disease (Table 1). Of the 240 patients who had talked to their physician about AIDS, physicians initiated the conversations in only 28 percent of the cases.

Patients had discussed a variety of AIDS-related issues with their physicians; sexual behavior and safe sex were discussed most frequently. Risk of transmission from blood transfusions

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and other health care-related risks were also discussed frequently, as were concerns about casual transmission.

Among patients who had not yet spoken with their physician about AIDS, 56 percent did not express a preference for or against talking to their doctor about AIDS, only 17 percent said they would not want to discuss the topic with their doctor, and 27 percent wanted to do so. Younger respondents (32 percent) and Blacks (37 percent) were more likely to want to talk to their physician about AIDS.

Since many AIDS-related concerns have to do with sex, patient-physician interaction regarding sexuality may influence the frequency of discussions regarding AIDS. Most patients (78 percent) did not recall having been asked questions about their sexual behavior at their physician's offices. Only 7 percent of respondents reported they would be unwilling to answer a physician's questions about their sexual behavior; 59 percent said they would be glad for the opportunity to talk to their physician about sex. Most patients (67 percent) said they would be very comfortable talking with their physician about AIDS.

The AIDS-related topics which patients said they would like to discuss are listed in Table 2. Transmission of AIDS, especially by casual contact and in medical settings, heads the list. Although 59 percent of patients said that they would go to their physician or HMO (health maintenance organization) for more specific information on AIDS, respondents reported getting most of their information from television (mentioned by 67 percent), newspapers (56 percent), and magazines (36 percent).

Discussion

Relatively few patients had discussed AIDS with their physician. This is consistent with previous reports based on physician surveys[10] and more limited patient surveys.[11] Previous studies[12] have also documented the infrequency of sexual history taking. It should be noted, however, that the denominator for the history taking and AIDS counseling items includes all patients who have had any physician contact over the past five years. Many of these contacts may not have been appropriate situations for these preventive practices. Although fallibility of patient recall may also lead patients to underestimate counseling by physicians, if patients do not remember having discussed AIDS, it is unlikely that the counseling was effective. Although the methodology of this survey may yield conservative estimates of physicians' actual counseling efforts, the evidence remains that AIDS-related discussions are not commonplace in physicians' offices.

TABLE 2—AIDS Topics People Wanted to Discuss with Their Physician according to the 419 Patient-respondents (27%) Who Said They Wanted to Talk to Their Physician about AIDS but Had Not Yet Done so

Topics for Discussion	%
AIDS transmission—general information	23.9
Risk from casual contact	21.0
Contagion in medical settings	11.7
Sexual behavior, safe sex	9.5
Prevention/avoid contracting AIDS	7.9
Treatment for AIDS	7.6
Infection control	6.7
Blood transfusions	5.7
Symptoms of disease	5.7
If doctor knew of anything not widely reported	5.3
Antibody testing	3.8
If physician was treating people with AIDS	3.6
Scientific questions	3.3
Drug use	3.1
Distrust of mass media	2.9

APPENDIX

Survey Items Concerning Patients' Experience and Opinions about AIDS Education by Physicians

- Have you ever talked to your medical doctor about AIDS?
- Who started the conversation . . . you or the doctor?
- What was it about AIDS that you and your doctor discussed? *(open-ended)*
- Would you want to talk to your doctor about AIDS, would you prefer not to discuss this with your doctor, or do you not care?
- What is it about AIDS that you would like to discuss with your doctor? *(open-ended)*
- If you were to talk with your doctor about AIDS, should they start the conversation, or would you rather start it yourself?
- Have you ever been asked questions at the doctor's office about your sexual behavior?
- It has been suggested that medical doctors should ask their patients about their sexual behavior as part of a routine health history taking. If your doctor asked you about *your* sexual behavior, would you be glad for the opportunity to share this information with your doctor, be somewhat uncomfortable but answer the questions anyway, or would you be unwilling to talk about these matters with your doctor?
- How comfortable would you feel . . . *(insert a through c)* . . . very comfortable, mostly comfortable, somewhat uncomfortable, or very uncomfortable?
  a. if you had a conversation with your doctor about AIDS
  b. if your doctor asked you questions about your sexual behavior
  c. if your doctor asked you questions about your use of intravenous drugs
- According to medical authorities, the following groups of adults are at highest risk for AIDS: people who received blood transfusions between 1977 and 1985, intravenous drug users, men who have sex with other men, and anyone who has had many sex partners. Do you know anyone that might be a member of these groups?
- Have you ever personally known anyone with AIDS or the AIDS virus?

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Screening for Chlamydial Cervicitis in a Sexually Active University Population

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Abstract: Enzyme-linked immunoabsorbent assays to detect chlamydial cervicitis were performed on samples from 1,320 sexually active university women. Seventy-five (prevalence 5.7 percent) had positive tests. Demographic, history, symptom, and physical examination variables were insufficient to predict infection accurately. We conclude that screening during routine visits with this population is cost-effective. (*Am J Public Health* 1990; 80:469–471.)

Introduction

Chlamydia trachomatis is the most prevalent sexually transmitted disease in the United States.[1] Until recently, diagnosis of chlamydial infection was handicapped by the lack of an inexpensive diagnostic test. *Chlamydia* is easily treated with antibiotics, but left untreated can persist for an indefinite time, and result in severe sequelae. While symptomatic women have a higher prevalence rate, most women with cervical infections have few or no symptoms.[2]

Methods

Routine screening for chlamydial infection of all sexually active students receiving physical examinations at the University of California-Los Angeles, Student Health Service Women's Clinic was implemented in June 1986. Some women were seen for initial or yearly examinations (routine visit group) and some women were seen for symptoms, partner symptoms, or previous problem follow-up (symptom/problem group).

Specimens were tested using Chlamydiazyme™ kits run according to the manufacturer's directions. Women with positive tests were typically tested with doxycycline 100 milligrams twice daily for seven days. Sexual abstinence was encouraged during treatment; partners were referred for treatment. Test of cure using culture was requested approximately three weeks after treatment.

A total of 1,320 women were screened between June 24 and October 24, 1986. Data were abstracted from the medical records of all but 19 of these women. Data collected included: age, marital status, race/ethnicity, reason for the visit, gynecologic history, present contraceptive use and level of current sexual activity, current symptoms, partner symptoms, physical findings, test results, and clinician diagnosis.

Chlamydiazyme test result was cross tabulated with these variables; prevalence odds ratios (POR) with 95% confidence intervals (CI) were calculated. All variables found to have POR with a lower limit on the 95% CI greater than one were subsequently used in a logistic regression (BMDPLR).[3]

Results

Of the 1,320 women tested, 75 (5.7 percent) had positive Chlamydiazyme tests. Thirty-six (5.0 percent) of the 724 routine visit group women had positive Chlamydiazyme tests while 39 (6.8 percent) of the 577 symptom/problem group women had positive tests.

Seventy-four of the 75 women with positive tests at their first study period visit returned for treatment; 65 of the 74 treated women returned for a culture test of cure. All 65 were successfully treated.

Table 1 lists those variables which were related to Chlamydiazyme result for the total sample. In the logistic regression, only five of the 13 variables were independently related to a positive test.

Recognizing that a logistic regression equation is likely to be too complicated for most clinicians to apply, a risk-factor index similar to those used by others[4,5] was constructed by summing presence of positive findings on each of the five independently related variables. Thus, women with none of the findings were assigned a score of 0, and women with all five of the findings were assigned a score of five. Table 2 lists sensitivity, specificity, and predictive values using different scores on the index as cutpoints.

Parallel analyses to those conducted for the total sample were conducted separately for women who were seen for routine visits, and those who were seen for problem/symptom visits. Results were not substantially different, but are available upon request

Discussion

Prevalence rates of chlamydial infection among female college students have ranged between less than 5 percent to nearly 10 percent.[6–14] The 5.7 percent prevalence of cervical infection we observed is at the low end of this range. However, our prevalence among women age 22 years and younger in our study was 8.7 percent; the relatively large number of older students in our study may account for the lower rate.

The literature is divided over whether medical history and examination are adequate to identify infection.[12,14] In our study, if only women who had one or more of the risk factors found to be independently related to a positive test had been screened, 694 women would not have been screened at a significant savings in resources, and 81 percent of the women

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TABLE 1—Rates of Positive Chlamydiazyme Tests Related to Selected Demographic, History, Symptom, and Examination Variables

Variables	All Subjects N	Percent Positive	Prevalence Odds Ratio (95% CI)
Marital status			
Not married	1096	6.1	2.7
Married	129	2.3	(0.8,8.8)
Age (years)			
22 or younger	462	10.0	3.1
23 or older	839	3.5	(1.9,5.0)**
Sexually active past 3 months			
No or not noted	494	3.6	2.0
Yes	807	7.1	(1.2,3.5)
New partner past 3 months			
No or not noted	1163	5.0	2.7
Yes	138	12.3	(1.5,4.9)**
Multiple partners past 3 months			
No or not noted	1229	5.2	3.3
Yes	72	15.3	(1.7,6.6)
Taking oral contraceptive			
No	831	4.7	1.7
Yes	470	7.7	(1.1,2.7)
Using condoms			
No	1164	6.2	2.9
Yes	137	2.2	(0.9,9.4)
Partner present symptoms			
No or not noted	1240	5.1	4.6
Yes	61	19.7	(2.3,9.1)
Cervix normal on examination			
No	77	15.6	3.4
Yes	1218	5.2	(1.7,6.8)
Cervix bleeds easily			
No	1263	5.3	6.0
Yes	32	25.0	(2.6,13.7)**
Cervix red			
No	1267	5.5	3.9
Yes	27	18.5	(1.4,10.6)
Wet mount—yeast			
No	1090	6.3	2.3
Yes	209	2.9	(1.0,5.4)
Wet mount—bacterial vaginosis			
No	1217	4.8	5.2
Yes	82	20.7	(2.9, 9.4)**
Wet mount—white blood cells			
No	1254	5.2	5.2
Yes	45	22.2	(2.5,10.7)
Diagnosis—normal examination			
No	621	7.7	2.0
Yes	678	4.0	(1.2,3.2)
Diagnosis—possible chlamydia			
No	1265	5.0	10.4
Yes	34	35.3	(5.0,21.5)**

**Variables independently related to Chlamydiazyme result in logistic regression

with a positive test would have been tested. However, 14 (19 percent) of the 75 women with positive tests would have been missed. In our sample, 27 (36 percent) of the 75 women with positive tests had entirely normal physical examinations. Thus it appears while signs, symptoms, history, and demographic variables would be helpful in determining whether to test for infection when laboratory resources are limited, these variables are insufficient to predict infection *accurately*.

Seven guidelines for determining whether a screening program is likely to be effective have been proposed.[15] If a randomized clinical trial has shown the effectiveness of a screening program, the assessment is relatively easy. We are unaware of any randomized trials of screening for chlamydial infection. However, we believe a screening program in our female population satisfactorily satisfies the other six guidelines.

For example, several tests are available which could be used to screen for *chlamydia*. We chose to use Chlamydiazyme, which has compared favorably with culture in our population.[10] The importance of the screening test and the prevalence of the condition is especially important when considering possible negative effects of a screening program. Using the specificity (99.5 percent) and prevalence (4.9 percent) found in our previous studies,[10] less than one out of 10 women who are told they are infected will be erroneously told so.

The costs of alternative strategies for screening during routine gynecologic visits were recently analyzed.[16] A strategy using no screening was compared with a strategy involving the routine use of culture, and a strategy using a less costly but less sensitive and specific alternative test. The analysis indicated that the alternative test would need to cost about $10 per test at a prevalence of 5 percent for the screening strategy to be cost effective. Because our screening test is both more sensitive and specific than the estimates used in that analysis, and our cost per test is less than $8, it appears that screening among our population can be supported on economic as well as clinical grounds.

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TABLE 2—Prediction of Positive Chlamydiazyme Result Using Different Levels of Risk Factor Index—All Subjects

Risk Factor Index Score	N	% Positive	% Sensitivity*	% Specificity*	% Positive Predictive Value*	% Negative Predictive Value*
0	694	2.0				
1 or More	599	10.2	81.3	55.8	10.2	98.0
0 or 1	1172	4.3				
2 or More	121	20.7	33.3	92.1	20.7	95.7
2 or Less	1270	5.0				
3 or More	23	47.8	14.7	99.0	47.8	95.0

*Compared to Chlamydiazyme result—predictive values based on observed prevalence

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Prevalence of Markers for Hepatitis B and Hepatitis D in a Municipal House of Correction

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Abstract: Following an outbreak of hepatitis B (HBV) in a municipal house of correction, HBV markers were detected in 173/406 (43 percent) inmates and 10/129 (8 percent) staff. Of the 173 HBV-infected inmates, 14 (8 percent) had hepatitis D (HDV) markers compared to 0/10 staff members. Intravenous drugs use (IVDU) was most strongly associated with HBV marker presence. Increasing duration of imprisonment, history of hepatitis B and especially IVDU were associated with the prevalence of HDV markers. (*Am J Public Health* 1990; 80:471–473.)

Introduction

Hepatitis D (HDV) is caused by an incomplete RNA virus dependent on hepatitis B virus (HBV) for survival.[1,2] Estimates of its prevalence range from 0–10 percent in chronic HBV carriers to 30–50 percent in persons with fulminant hepatitis B.[3] Most data suggest that in the United States intravenous drug users and hemophiliacs are at increased risk for HDV.[4] Although higher prevalence of HBV markers in prisoners or inmates of correctional facilities when compared to the general population has been described in several studies, similar data on HDV in incarcerated populations are not available.[5–10]

In January 1985, the Boston Department of Health and Hospitals was notified that five inmates in the Suffolk County House of Correction had an acute onset of hepatitis B. One inmate was coinfected with HDV and died of fulminant hepatitis. All inmates and employees were offered HBV screening and subsequent vaccination if seronegative as part of an outbreak control plan. While providing epidemiological assistance, we had the opportunity to evaluate the HDV status in those with HBV markers.

Methods

The Suffolk County (Massachusetts) House of Correction at Deer Island, operated by the City of Boston Penal Institution Department, accommodates 460–480 inmates, who serve an average sentence of 11 months. There are approximately 210 staff members employed at the facility including two registered nurses and one part time physician who staff an on-site infirmary.

In February 1985, a fact sheet on HBV and HDV was distributed to all inmates and staff, and participation in an HBV screening and vaccination program was solicited. Participants were questioned by one of the investigators regarding age, duration of involvement with the prison system, history of prior hepatitis, history of intravenous drug use (IVDU), location within the prison facility, and primary city of residence.

Serum was obtained to determine presence of HBV core antibody (anti-HBc; CORAB, Abbott Laboratories, N. Chicago, Illinois). Sera with borderline anti-HBc results were also tested for hepatitis B surface antigen (HBsAg) and antibody (anti-HBs) (AUSRIA and AUSAB, respectively, Abbott Laboratories). All anti-HBc positive specimens were also analyzed for HDV antibody (anti-delta; Abbott Laboratories). An interpretation of test results was given to each participant and HBV vaccination offered to those without HBV markers. This protocol was approved by the Boston Department of Health and Hospitals Institutional Review Board.

The magnitude of relative effect was assessed for several risk factors of HBV positivity. Univariate and multivariate

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TABLE 1—Characteristics of Participants in a Hepatitis Screening Program in Prison, Boston, 1985

Characteristics	Inmates (N = 406) (%)	Employees (N = 129) (%)
Mean age (years)	27.8	33.9
Mean duration of prison involvement (years)	2.2	5.7
History of hepatitis, any type	45 (11)	5 (4)
History of hepatitis B	17 (4)	0 (0)
History of homosexual activity	12 (3)	0 (0)
Intravenous drug use	136 (33)	2 (2)
Anti-HBc positive	173 (43)	10 (8)

analyses were conducted using SAS procedures to calculate odds ratios, test-based confidence intervals,[11] and logistic regression coefficients. Crude odds ratios (OR) and their exact 95% confidence intervals (CI) for risk factors in the 14 cases of HDV were calculated using the method described by Gart.[12]

Results

Of 463 inmates offered screening, 409 (88 percent) agreed to participate. Of these 409, three had equivocal anti-HBc results and are not included in further analysis. Characteristics of participants are shown in Table 1. There was no discernible pattern in findings related to city of residence.

Of the 142 employees invited to participate, 129 (91 percent) agreed. Their characteristics are shown in Table 1.

By univariate analysis, risk factors for HBV among prisoners included age greater than 30 years, prior history of HBV, and IVDU. In the multivariate model prior history of hepatitis was also included. Tests for interaction of age and IVDU and for other potential interactions were not significant. By multivariate analysis, IVDU was most strongly associated with HBV marker presence (OR = 6.0, 95% CI = 3.6, 10.2). Increasing duration of incarceration, independent of age or IVDU, was also related to the presence of HBV markers (Table 2).

Of the 173 specimens from inmates with anti-HBc, 14 (8 percent of all inmates) contained anti-delta. Relative odds of IVDU, imprisonment for one to four years and for five years or more, history of hepatitis B, and age over 30 were elevated. All prisoners positive for HDV reported that they had had some type of hepatitis in the past, and 12 of the 14 reported both a history of hepatitis and IVDU. The small numbers and correspondingly wide intervals, and the inability to control joint confounding in this data set, prevents further interpretation of the data.

Discussion

Several studies have documented a high prevalence of HBV markers in prison populations.[5–10] Reported figures are comparable to the proportion reported here of 43 percent. As in other studies, IVDU was found to be strongly associated with evidence of HBV infection. One-third of our inmate participants gave a history of intravenous drug use. This figure may not be reflective of correctional institutions as a whole, and thus the prevalence of HBV markers may differ in other facilities.

Less information is available on the prevalence of HDV infection in inmates of correctional facilities. A previous study revealed that four of 19 prisoners (21.1 percent) known

TABLE 2—Prevalence of Hepatitis B (HBV) and Hepatitis D (HDV) Markers According to Risk Factors and Relative Odds of Marker Positivity with 95% Confidence Intervals

Risk Factor	N	HBV		HDV	
		Positive N (%)	Adjusted* OR (95% CI)	Positive N (%)	Crude OR (95% CI)
Total	406	173 (43)	—	14 (8)**	—
Age (years)					
≤30	261	91 (35)	1.0	6 (7)	1.0
>30	144	81 (56)	1.8 (1.1–3.0)	8 (10)	1.6 (0.5–5.7)
Unknown	1	1		0	
Prison involvement (years)					
<1	166	46 (28)	1.0	1 (2)	1.0
1–4	178	81 (46)	1.8 (1.3–2.6)	8 (10)	4.9 (0.6–224)
≥5	58	42 (72)	3.3 (2.3–4.8)	5 (12)	6.1 (0.6–294)
Unknown	6	4 —	—	0	
IV drug use					
No	264	72 (27)	1.0	2 (3)	1.0
Yes	136	101 (74)	6.0 (3.6–10.2)	12 (12)	4.7 (1.0–44)
Unknown	6	0 —	—	0	
History of HBV					
No	358	137 (38)	1.0	6 (4)	1.0
Yes	17	14 (82)	5.4 (1.3–22.0)	2 (14)	3.6 (0.3–23)
Unknown	31†	22 —	—	6† —	—
Homosexual activity					
No	387	166 (43)	1.0	13 (8)	1.0
Yes	12	6 (50)	0.6 (0.1–2.2)	0 (0)	
Unknown	7	1 —	—	1	

*A multivariate model included age, years involved in prison, intravenous drug use, history of HBV, and homosexual activity.
**Denominator is number HBV positive in each risk category.
†Includes people with a history of hepatitis, type unknown, and people who refused to answer the question.

to be infected with HBV in a county jail had markers for HDV, two with antigen and two with antibody.[4] In the current study all participants were healthy at the time of screening, and the anti-delta positive results presumably represent past infection. Although one of the five cases of HBV in the outbreak that precipitated the screening/vaccination program contained IgM anti-delta, specimens from persons in this program were not evaluated for the presence of HDV antigen or IgM anti-delta. Thus some acute HDV infections could have been missed. The proportion of specimens containing anti-delta (14/173, 8 percent) from inmates in this study, however, approximates the proportion of HDV antibody noted in inmates by the study by Jacobson, *et al.*[4] As with HBV, IVDU was strongly associated with the presence of anti-delta; of the 14 inmates with markers for HDV, 12 (86 percent) gave a history of IVDU. Ponzetto, *et al.* previously reported that 10 percent of drug addicts participating in a Veterans Administration (VA) cooperative study had evidence of infection with HDV.[13]

Measures that protect against HBV infection, such as use of hepatitis B vaccine, are also effective in preventing HDV infection. Implementation of HBV preventive guidelines in corrections facilities should reduce the risk of HDV.

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Influenza Epidemics and Anencephaly

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Abstract: To explore the postulated association between maternal influenza and congenital defects of the central nervous system, 14 virologically verified epidemics in Finland, 1969–82, were studied. Mothers of 248 anencephalic children were grouped into those whose first trimester had occurred during an epidemic period and those whose pregnancy had commenced during a non-epidemic period. No significant differences in prevalence of anencephaly were noted in these groups. (*Am J Public Health* 1990; 80:473–475.)

Introduction

There is ample evidence that certain virus infections can be transmitted to the embryo during pregnancy and cause impaired fetal development expressed as congenital defects.[1] Among these potential teratogens, influenza virus has been frequently mentioned, but evidence for or against such harmful effects has remained inconclusive.[2,3] We have conducted a population study based on recorded influenza epidemics and one particular marker defect, anencephaly. This defect has several advantages for investigative purposes: its detection rate is high in stillbirths and in newborn infants, the diagnostic means have not changed during our 15-year-study period, notification rate of this severe defect is high, and finally, as it is a lethal condition, the reports to the Register of Congenital Malformations can be confirmed and completed from the compulsory death certificates. Moreover, defects of the central nervous system (CNS) have often been mentioned in the context of maternal influenza.[2–6]

Methods

The material was collected from the files of the Finnish Register of Congenital Malformations operating since 1963[7,8] and completed with data from the death certificates obtained from the Central Statistical Bureau. These sources cover the entire population of 4.9 million; the present analysis includes all mothers whose last menstruation occurred between October 1, 1968 and March 31, 1982, comprising 858,917 deliveries. Altogether 248 cases of anencephaly were recorded in children weighing 600 g or over at birth. Of these, 201 cases were single defects and, in 47, additional malformations were reported.

Influenza activity in the Finnish population has been monitored by the Central Public Health Laboratory since 1969 with virus isolation and serodiagnostics of acute respiratory infections. Our study covered 14 epidemic periods during which influenza etiology was confirmed. Excluding sporadic cases and small local outbreaks, the epidemic time

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TABLE 1—Occurrence of influenza epidemics in Finland and serological infection percentages in samples of serum pairs from pregnant women.

Epidemic periods	Number of serum pairs (a)	Influenza A		Influenza B
		H1N1 (%)	H3N2 (%)	(%)
January 69–February 69		–	(b)	–
December 69–February 70		–	(b)	–
April 71–May 71		–	–	(b)
November 71–December 71	137	–	27	–
January 73–February 73	91	–	20	–
January 74–April 74	106	–	23	18
January 75–March 75	144	–	17	–
January 76–March 76	397	–	15	–
March 77–April 77	636	–	14	7
January 78–March 78	651	1	8	–
January 79–April 79	549	3	3	2
January 80–February 80	576	–	25	–
January 81–March 81	674	10	2	4
February 82–June 82	291	1	1	1

(a) The pairs consisted of pre-epidemic and postepidemic specimens.
(b) Epidemic activity recorded, but infection frequency among pregnant women not studied.

was 37 months of the total 162 months monitored during the study period. To create a "non-epidemic" control material, the month preceding and following each epidemic was excluded because of timing uncertainties. Altogether, 26 such borderline months with 46 anencephalic children were thus excluded.

Since the end of 1971, the influenza surveillance program has included annual monitoring of serological infection frequencies among pregnant women in Finland.[9] Serum pairs of pre-epidemic and post-epidemic specimens from a total of 4,252 women were studied for haemagglutination-inhibiting antibodies to influenza A and B viruses including the epidemic variant, and, for detection of anamnestic antibody response, the serotypes of the near past (Table 1). A fourfold or higher rise in antibody titers was considered indicative of serological influenza infection.[9]

For the occurrence of anencephaly in infants, mothers whose first trimester (14 weeks from the start of the mother's previous menstruation) had coincided wholly or in part with a virologically verified influenza epidemic (407,200 mothers) and mothers whose first trimester had occurred completely during a non-epidemic period (314,841 mothers) were compared after exclusion of the borderline months. Power calculations were performed according to Schlesselman.[10] The heterogeneity test was performed and confidence limits for rate ratio estimates were calculated as suggested by Miettinen.[11] Trend analysis for the proportion of rates of anencephaly was performed according to Armitage.[12]

Results

Epidemics caused by influenza A and B viruses, and serological infection frequencies in the follow-up samples of pregnant women are listed in Table 1. Figure 1 shows the rates of anencephaly when the first trimester had occurred during the influenza epidemics or during the non-epidemic periods. In the epidemic group, 117 anencephalics were reported and in the non-epidemic group, 85; prevalence 0.29 per mil and 0.27 per mil, respectively.

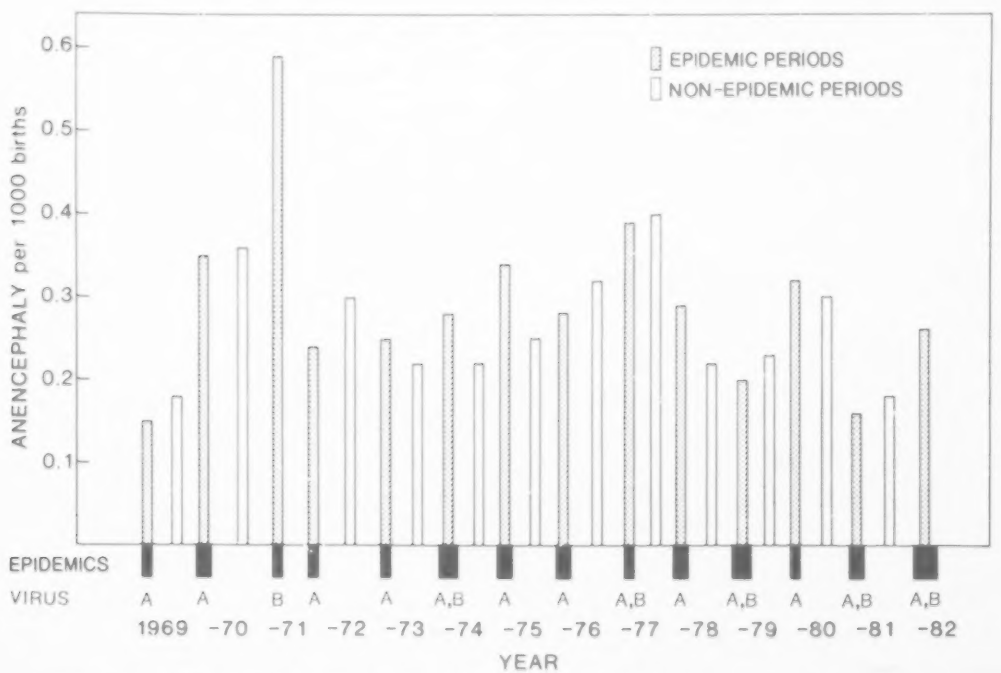

FIGURE 1—Prevalence of Anencephaly in Children Whose first 12 Weeks of Development Coincided either with an Influenza Epidemic or with the Non-Epidemic Periods

When the rates of anencephaly were examined in relation to the serologically verified influenza epidemics, the April-May 1971 B-influenza epidemic showed an apparently high rate in comparison with other periods (Figure 1). However, the heterogeneity test indicated that within the study period of 1969–82, none of the observed rates showed statistically significant elevation ($p = 0.75$). It was evident that influenza epidemics in general were not associated with the occurring of anencephaly. The rate ratio point estimate for anencephaly was 1.0 (95% confidence limits 0.8, 1.3), when the infants of mothers whose first trimester had coincided with an influenza epidemic were compared with the infants whose sensitive period of development had occurred during a non-epidemic period. Trend analysis showed no change in the rate of anencephaly for the period of 1969–82 (regression slope −0.0000028, SE 0.0000026).

Discussion

Two previous studies in the same population suggest, as we do here, that an influenza virus infection of the mother might not be a major cause of neural tube defects: serological screening of mothers to defective children showed no elevated titers against influenza virus antibodies as compared with matched-pair controls,[13] and CNS defects showed no seasonal variations in 1965–73 despite the definite winter excess of influenza.[14] The latter finding confirms similar observations in other populations.[15]

An exception to the negative findings listed above seems to be provided by the Asian influenza pandemic in 1957 caused by a new subtype (H2N2) of the influenza A virus. Several independent studies reported an association of this epidemic and an increased risk of defects of the CNS, anencephaly included.[14,16–20] Caused by a new subtype of the virus, the epidemic attacked a highly susceptible population, which might explain the association. In the present material, a similar effect could not be found for the 1968/69 pandemic in which the new subtype H3N2 was involved. The present analysis did not reveal any association between an increased risk of anencephaly and maternal influenza infection. The power of the study was about 80 percent to detect a rate ratio of 1.4 for all epidemics combined and almost 100 percent to detect rate ratios of 1.7 or greater ($\alpha = 0.05$, one-sided).

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Rabies Vaccine Adsorbed: Neutralizing Antibody Titers after Three-Dose Pre-exposure Vaccination

BYRON S. BERLIN, MD

Abstract: Field trials at several schools of veterinary medicine showed that three-dose pre-exposure rabies vaccination with Rabies Vaccine Adsorbed developed by the Michigan Department of Health elicited neutralization antibody in practically all recipients two to three weeks after immunization. Titers declined during the first six months after vaccination. However, by 18 to 24 months, 98 percent of recipients still had titers equal or greater than a 1:5 dilution of serum. (*Am J Public Health* 1990; 80:476–478.)

Introduction

Initial clinical trials with Rabies Vaccine Adsorbed (RVA), a new tissue culture derived rabies vaccine developed by the Michigan Department of Public Health,[1] were done with a two-dose pre-exposure immunization schedule.[2] The studies showed that this new vaccine induced satisfactory antibody titers two to three weeks after vaccination and prepared recipients for a vigorous response to booster vaccination. Additional clinical trials, summarized in the present study, were conducted to ascertain antibody responses after administration of this vaccine by pre-exposure immunization schedules recommended by the Immunization Practices Advisory Committee (ACIP), Centers for Disease Control, i.e., three doses of rabies vaccine of tissue culture origin, one dose each day on days 0, 7, and 21 or 28 days.[3]

Methods

Vaccine

The vaccine used in the present study was previously described.[1] Briefly, RVA contains non-infectious, B-propiolactone-treated rabies virus which is adsorbed onto aluminum phosphate. The virus was grown on diploid fetal rhesus monkey lung fibroblasts (FRh1-2), the rhesus analogue of the WI-38 cell. The antigenic potency of each lot of RVA exceeds 2.5 International Units (IU).

Subjects

The study consisted of 24 separate clinical subtrials with a combined enrollment of 1,735 students and staffs from five schools of veterinary medicine: 1,647 with no prior vaccination against rabies and 88 who had not responded to Duck Embryo Rabies Vaccine. The number of volunteers in individual subtrials varied from 16 to 127 and the median size was between 68 and 74 persons. Each person received three intramuscular injections of RVA, 1.0 mL per dose, at 0, 7, and 21 or 28 days and then was bled two weeks after the final dose of vaccine and, depending on the subtrial in which they were included, at six months and nine to 14 months. Volunteers remaining on the campuses were bled 18 to 24 months after vaccination (Table 1). Serum samples were sent to the Michigan Department of Public Health laboratories for antibody determination. Each volunteer provided a brief medical history and signed an informed consent before vaccination.

Serology

Serum neutralization titers were ascertained by the Rapid Fluorescent Focus Inhibition Test (RFFIT)[4] beginning with a 1:5 dilution of the serum and ending with a 1:625 dilution using five-fold dilutions. RFFIT titers, given as International Units (IU), were computed by dividing the reciprocal of the dilution titer of the subject's serum by the reciprocal of the dilution titer of a standard serum, designated as 1.0 IU, tested at the same time. RFFIT titers less than 1:5 dilution of serum, approximately 0.1 IU were considered below the minimally acceptable titer recommended by the ACIP.[3] Titers from 0.2 to 0.5 IU were classed as acceptable because they fell between 0.1 IU and the minimally acceptable initial RFFIT response to post-exposure immunization recommended by the World Health Organization (0.5 IU).[5] Titers above 25 IU were grouped together because the maximum dilution for testing the sera was 1:625.

Results

Ninety-six percent (1,673/1,735) of the vaccinees were bled two weeks after the last dose of the vaccine series (Table 1). Nineteen (1 percent) had either withdrawn voluntarily or were dismissed from the study because of illness or reaction to the vaccine. Forty-three (2 percent) failed to return for bleeding. All but one of the 1,673 who were bled at two weeks had rabies neutralizing antibody titers greater than 0.1 IU.

Subsequently, 91 percent (419/461) of volunteers initially enrolled in five of the 23 subtrials were bled six months after the vaccine series (Table 1) and, of these, 415 (99 percent) had RFFIT titers greater than 0.1 IU. Nine to 14 months after completing the vaccine series, blood samples were obtained from 761/984 (77 percent) volunteers enrolled in 13 subtrials. Of these, 748 (98%) also had RFFIT titers equal or greater than 0.1 IU. Finally, 308 persons (52 percent of the initial

TABLE 1—Rabies Vaccine Study Plan

Number of Subtrials	Number of Volunteers	Time and Number of Persons Bled			
		Two Weeks	6 mos	9–14 mos	18–24 mos
7	360	348			
1	101	99	84		
3	251	249	234	179	
6	431	413	N.D.*	301	
1	109	109	101	N.D.	98
4	366	350	N.D.	281	133
2	117	105	N.D.	N.D.	77
Total (24)	(1,735)	(1,673)	(419)	(761)	(308)
Percent 0.1 IU		1	4	13	5
Number of initial volunteers		1,735	461	984	592
Percent of initial volunteers bled		96	91	77	52

*Not Done.

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TABLE 2—Persistence of RFFIT Titers after Pre-exposure Vaccination with Rhesus Diploid Rabies Vaccine

Number Enrolled	Antigen content*	Schedule Days**	Median Titer***	Bleeding Time	RFFIT Titer Range***							
					<0.2	0.2–0.5	0.6–1.0	1.1–5.0	5.1–15.0	15.1–24.9	>25	Number
109	2.6	0-7-28	6.4	2 wks	0	0	0	25	52	12	5	94
			2.2	6 mos	0	2	23	57	9	2	1	94
			1.2	18 mos	1	23	22	46	2	0	0	94
74	2.6	0-7-21	5.4	2 wks	0	0	1	9	33	9	3	55
			0.3	22 mos	2	26	13	8	5	1	0	55
88	3.0(a)	0-7-28	5.4	2 wks	0	0	1	3	21	7	2	34
			0.4	2 yrs	7	12	10	3	1	0	1	34

*Antigenic Potency expressed as International Units (IU).
**Days of Injection.
***RFFIT Titer expressed as International Units (IU).
(a)Vaccine contained 1:10,000 thiomerosal as a preservative.

volunteers in seven subtrials) volunteered to be bled from 18 to 24 months after vaccination. Ninety-eight percent (303/308) still had titers equal to or greater than 0.1 IU.

The findings in three representative clinical subtrials in which neutralizing antibody titers are presented only for persons from whom successive blood samples were obtained at each indicated time are shown in Table 2. Successive blood samples were obtained from 86 percent (94/109), 74 percent (55/74), and 39 percent (34/88) of volunteers initially enrolled in the respective trials. Two weeks after the third and final injection of RVA, median RFFIT titers in the three subtrials were 6.4, 5.4 and 5.4 IU, respectively. All recipients in these subtrials had neutralizing antibody at two weeks and, in those tested, at six months after vaccination. However, neutralizing antibody titers six months after vaccination were lower than those at two weeks. Eighteen months to two years post-vaccination, median RFFIT titers were from to 0.3 to 1.2 IU and 261/271 (96 percent) of the volunteers had titers equal to or greater than 0.1 IU.

Discussion

The findings summarized in the present study show that the seroconversion rate after pre-exposure vaccination with RVA, given by the currently recommended three dose schedules, approximated 100 percent. However, the duration of the antibody response after initial pre-exposure rabies immunization is as important a concern as the seroconversion rate in evaluating immune responses to rabies vaccines because neutralizing antibody, actively induced by vaccination or passively acquired, is a key factor in protection against rabies infection.[6–9] Yet, determination of the duration of responses to vaccination in sizable populations is complicated by reason of attrition of volunteers. Attrition rates at six months and at nine to 14 months in this study were 9 percent and 23 percent, respectively; these rates probably have little effect on the interpretation of the results in view of the finding that 94 percent of recipients or more had titers equal to or greater than 0.1 IU. On the other hand, even though the attrition rate for those bled 18 months to two years after vaccination was 48 percent, the percent of volunteers showing neutralizing antibody at that time is considered a good approximation of persistence of antibody because titers at two weeks in those who were bled later did not appear different than those in persons who were not bled at the later time.

Although RVA and human diploid cell culture vaccine (HDCV) have not been compared to each other in concurrent clinical trials, the seroconversion rate, persistence of rabies neutralizing antibody, and adverse reactions after pre-exposure immunization with RVA appear practically the same as those obtained with HDCV.[2,10–19]

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Health Effects of a Thorium Waste Disposal Site

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Abstract: A case-control study of 112 households residing in the vicinity of a thorium waste disposal site found a higher prevalence of birth defects (RR 2.1) and liver diseases (RR 2.3) among exposed than the unexposed group. The numbers were quite small and the confidence intervals wide, however, so that no definite conclusions can be drawn from these data. (*Am J Public Health* 1990; 80:478–480.)

Introduction

Several studies have appeared in the medical literature analyzing possible adverse health effects for persons living in the vicinity of hazardous waste disposal sites.[1–3] To our knowledge there is no published study in which the health effects of a thorium waste disposal site (TWDS) on the general population have been investigated, except for a community-based study in China.[4] That study found no deleterious health effects from exposure to thorium, but had very low statistical power.

The main concern about the possible biological effects of thorium (Th) with a half life of 1.4×10^{10} years is based on its radioactive characteristics and isotopes.[5] Thorium^{232} is the parent of various radio-isotopes including ^{228}Ra (radium), ^{228}Ac (actinium), ^{228}Th, ^{224}Ra, and ^{220}Rn (radon)[5]. Current interest is based in part on increased use of thorium as an energy source, e.g., in reactors using ^{232}Th to produce ^{233}U.[6]

Industrial and mining exposure to thorium have resulted in an increased incidence of lung cancer, pancreatic cancer, colorectal cancers, chronic respiratory diseases, liver damage, and other serious illnesses.[7–9] Skeletal sarcomas, thorotrastomas, and sarcomas of the reticuloendo-thelial system have been reported from a study of patients receiving intravenous injections of thorotrast, a collodial solution of thorium oxide that was used as a contrast medium for diagnostic radiographic studies from the 1930s to the 1950s.[10]

The purpose of this study was to conduct a survey of health effects of a thorium waste disposal site among members of the general population of Wayne, New Jersey, USA.

Thorium Waste Disposal Site

The site is located on a 6.5 acre tract of land in Wayne Township, Passaic County in northern New Jersey, USA.* The population density in Wayne is 1,867.2 per square miles and annual per capita income is $7,725.[11] The thorium waste burial site lies within 100 feet of the sidewalk. Commercial businesses, a nursing home, a school bus maintenance yard, and residential homes are adjacent to the site. A drainage ditch lies at the northern and eastern borders, and drains into a brook at the northeast corner of the burial field. The brook (a tributory to the Pompton River) flows through residential areas (copy of map available on request). Fresh water wetlands lie approximately 1,800 feet to the west of the thorium waste disposal site.* From 1948 until 1971, a company extracted thorium and rare earths from monazite ore, for usage by the Atomic Energy Commission.* During its active years, the plant waste was dumped in backyard sludge piles. The wastes have been buried only in the last few years, after spending over 20 years above the ground.**

Surveys of the New Jersey Department of Environmental Protection showed readings of 40-1491 uR/hour (uR = micro-rad, micro being 10^{-6}) in various regions classified as contaminated areas; Sheffield Brook contamination occurs within 10 meters of both sides. Soil samples revealed Thorium (^{232}Th), Radium (^{238}Ra) and Uranium (^{238}Ur) in the contaminated regions. Water samples from Sheffield Brook and the drainage ditch showed mainly alpha and beta activity.

Most of the air concentrations (ambient and indoor) were within the background radiation levels (approximately 150 mrem/year) The US Environmental Protection Agency (EPA) standard for home concentration is below 4 pCi/liter.***

The residents of the households in the vicinity of the thorium waste disposal site and in the vicinity of contaminated Sheffield Brook have not been tested for radioactive materials uptake.

Methods

The exposed population included 76 households within three blocks adjoining the contaminated areas. The 76 comparison (unexposed) households included all houses located on about 9th and 10th blocks distance from the thorium waste disposal site and contaminated Sheffield Brook. Households were accepted for inclusion in the study if they had resided at the present address for at least five years.

Eleven and 10 households of the exposed and unexposed groups, respectively, did not meet the criteria for eligibility, 10 and nine households, respectively, did not participate in the study due primarily to members not being at home at the time of scheduled interviews or having moved out without leaving forwarding addresses.

Face-to-face interviews of all 112 households (362 members), using a structured questionnaire, were carried out by one interviewer who did not know exposure status; 32 household members who did not agree to a face-to-face

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*New Jersey Department of Environmental Protection. Site Descriptions for Hazardous Waste Sites in New Jersey. Division of Waste Management, Hazardous Site Mitigation Administration, 1984/85.

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**New Jersey Department of Environmental Protection. Unpublished data.

***US Environmental Protection Agency, Office of Radiation Programs, Washington, DC 20406.

Further details on measurements of exposed areas are available on request to author (Najem).

TABLE 1—Demographic Variables among Exposed and Comparison Groups

Variables	Exposed Group	Comparison Group
# of Persons studied	171	191
# of Households studied	55	57
% of Persons Age 50 & over	29	32
% of Persons Age 18 & under	27	26
Duration of Residence		
Mean duration*	13.3 Years	17.2 Years
% Under 10 Years	42	23
% 10–20 Years	23	9
% Above 20 Years	35	68
Annual Family Income		
% Less than $15,000	9	9
% $15,000–$49,000	65	63
% $50,000–$59,000	20	21
% $60,000+	6	7

*Mean duration difference = 3.9 years (95% CI = −.71, −7.23)

interview were interviewed by telephone. For each disease or condition reported by the participants, we asked respondents whether they obtained the diagnostic information directly from their physicians; we did not obtain or review medical records.

The risk ratios associated with the disease were estimated by calculating relative risk ratios.[12] Cornfield's 95% confidence intervals (CI) and Miettinen's 95% CI were computed by using True Epistat computer software.[13] The Mantel-Haenszel method of controlling for confounding variables was used by using the SAS (Statistical Analysis System) computer software.[14]

Results and Discussion

There were no important differences in age, sex, race, income, marital status, or religious affiliation of the exposed and comparison groups, but the comparison group had resided longer in the community (Table 1). There were no differences in smoking history, alcohol consumption, frequency of eating local produce, and history of working in a chemical industry, or in reported history of cancer between the two groups (Table 2).

Although there was a higher prevalence of birth defects and liver diseases among the exposed than the control group, numbers are small and confidence intervals wide (Table 3). After adjusting for duration of residency and alcohol consumption, the estimated risk ratios were very similar, except for 1.1 (95% CI = .05, 78.4) for low birth weight and 2.9 (95% CI = .12, 168.24) for liver diseases.

ACKNOWLEDGMENTS

This study was presented in part at the XI Scientific Meeting of the International Epidemiological Association, Helsinki, Finland, August 1987.

TABLE 2—Comparison of Risk Factors among Exposed and Comparison Individuals and Households

Individuals	Exposed Group (N: 171)	Comparison Group (N: 191)	Risk Ratio (95% CI)
Ever smoked	57 persons	64 persons	0.94 (.63, 1.57)
Ever smoked cigarettes	96% of smokers	94% of smokers	
Average # cigarettes smoked per day	21 cigarettes	18 cigarettes	
Average # of years smoked	19 years	22 years	
3+ alcoholic drinks per day at present	10 persons	12 persons	0.93 (.36, 2.37)
3+ alcoholic drinks per day in past	37 persons	32 persons	1.37 (.78, 2.40)
Drinking 5 oz per week at present	13 persons	25 persons	0.54 (.25, 1.15)
History of working in a chemical industry	20 persons	31 persons	0.7 (.36, 1.30)
Households			
Grow vegetables	19 households	23 households	
Cancer among first degree relatives	31 households	31 households	1.1 (.48, 2.44)

TABLE 3—Diseases and Adverse Birth Outcomes among Exposed and Comparison Groups

	Exposed Group N = 171	Comparison Group N = 191	Risk Ratio	95% CI
Low Birthweight	3	6	0.6	.11, 2.52
Skin diseases	7	13	0.6	.21, 1.62
Anemia	6	7	1.0	.27, 3.24
Spontaneous abortion	13	14	1.0	.44, 2.43
Liver diseases*	4	2	2.3	.35, 12.0
Birth defect**	9	5	2.1	.62, 7.25
Cancer***	11	10	1.2	.48, 3.26

*Including: jaundice and hepatomegaly.

**Born with birth defects at the present address including: cerebral palsy, congenital heart disease, inguinal hernia, hemangioma, foot deformities, other bone and joint deformities.

***Including: leukemia, bone cancer, lung cancer, larynx cancer, and nose cancer.

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Comparison of Antineoplastic Drug Handling Policies of Hospitals with OSHA Guidelines: A Pilot Study

BARBARA VALANIS, DRPH, KATHLEEN DRISCOLL, JD, AND VIRGINIA MCNEIL, MSN

Abstract: Hospital antineoplastic drug handling policies of 24 hospitals in eight Southwestern Ohio counties were compared with recommendations of the 1986 Occupational Safety and Health Administration (OSHA) guidelines. Although most study facilities where antineoplastics are handled have policies, content varies and is generally less complete than OSHA guidelines, particularly regarding storing, transporting, and disposing of drugs; managing equipment; informing personnel of risk; and surveillance. Recommendations for personal protection concur more closely with OSHA guidelines than do other content areas. (*Am J Public Health* 1990;80:480–481.)

Introduction

Evidence of probable risk associated with staff handling of antineoplastic drugs led the US Occupational Safety and Health Administration (OSHA) to issue comprehensive guidelines covering staff training and surveillance; use and care of protective equipment, apparel, and techniques; and storage, disposal, and transport of drugs and contaminated equipment.[1] Concerned about moral and legal issues, many hospitals have also developed institutional policies.

This study investigated the status of facility policies for handling antineoplastic drugs in hospitals and home health care agencies in eight southwestern Ohio counties and compared policy content to OSHA guidelines.

Methods

In July 1986, a questionnaire, modeled after Tortorici's[2] was mailed to the directors of the 32 hospitals and 25 community health agencies in the eight predominately rural counties comprising Region I of the Cancer Control Consortium of Ohio. Respondents were requested to enclose their facility's antineoplastic drug handling policy. Policy content was compared with that of the 1986 OSHA guidelines using an investigator-designed tool for systematically coding content relative to OSHA guidelines. For each item, the facility policy is rated 0 if the item is absent and 1 if the item is included. The instrument yields a maximum total score of 219 and five subscores: 1) General Guidelines (37); 2) Protective Equipment (14); 3) Facilities for Mixing and Storing Agents (15); 4) Protective Apparel (49); and 5) Protective Techniques (104). The content analysis was conducted by one investigator (KD).

Results

Twenty-four hospitals and 11 community health agencies responded, a response rate of 75 percent for hospitals, 44 percent for home health agencies. Administrators of 20 of the hospitals report antineoplastics are mixed and administered at their facility and one home health agency administers premixed antineoplastics; 18 of the 20 hospitals and the home health agency have implemented an antineoplastic drug handling policy. Eleven hospitals submitted their policy for analysis.

All 11 policies are less comprehensive than OSHA guidelines. Total policy scores of individual facilities ranged from 26 to 92 of a possible 219. The mean total score was 50.6 (median score 41). Areas least addressed under "General Guidelines" were maintaining files on specific antineoplastic agents and providing staff with information on known risks, medical policies, safe handling of investigational drugs as they are introduced, and follow-up evaluation of staff knowledge and competence regarding antineoplastic drug handling. Least addressed under "Protective Equipment" were procedures for installation, maintenance, and decontamination of hoods, while unaddressed content under "Facilities for Mixing and Storing Agents" related to reserving a hood only for mixing antineoplastics, posting warning signs, listing drugs covered by cytotoxic policies, and labeling stored antineoplastics. Under "Protective Apparel" recommendations for use of protective apparel during drug mixing conformed better to OSHA guidelines than recommendations for administration, while those for excreta handling and cleaning up spills were generally inadequate. Recommendations for glove and gown use were more often consistent with OSHA recommendations than for use of masks or eye protection. Under "Protective Techniques" least frequently

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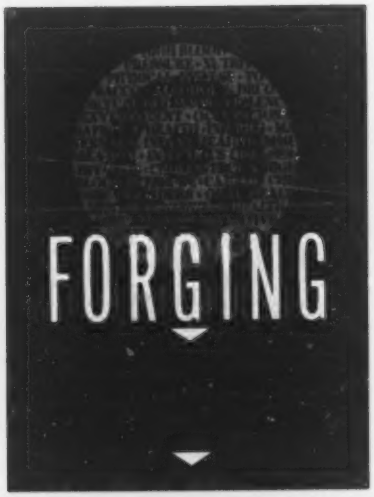

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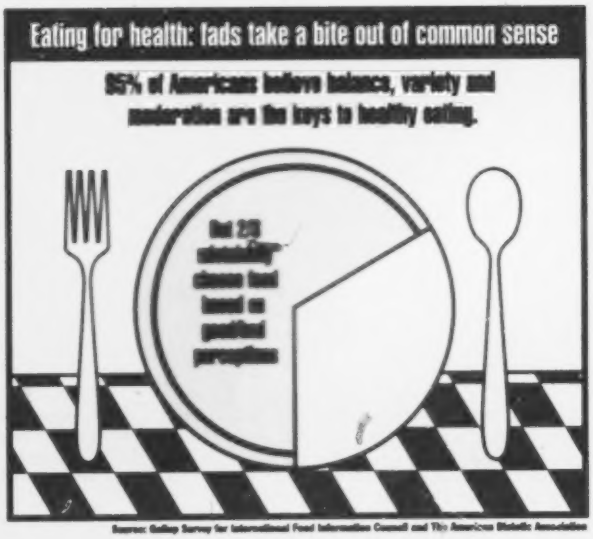
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Improving Influenza Vaccination Performance in an HMO Setting: The Use of Computer-Generated Reminders and Peer Comparison Feedback

MARY B. BARTON, BA, AND STEPHEN C. SCHOENBAUM, MD, MPH

Abstract: We evaluated a program for improving influenza immunization performance in a health maintenance organization (HMO). The HMO implemented several interventions successively from 1984–87: a postcard reminder to members at high risk for complications of influenza, a computer-generated reminder to the physician at the time of any primary care visit by high-risk patients, performance feedback to chiefs of service, and, finally, retrospective feedback to each physician comparing his/her performance with that of the other physicians. We examined immunization rates for a group of members older than age 65, a high-risk group under age 65, and a group of diabetic members who had not been subject to the reminders (vs a group who had been covered by the program).

Vaccination rates were increased in those diabetic members who received reminders. Nevertheless, among members younger and older than age 65 whose experience was observed over three flu seasons, a significant increase in vaccination rates was not achieved until physician feedback was added to the program. We conclude that each element of the reminder and feedback program has contributed to the overall increase in vaccination rates at the HMO and that effective ongoing influenza immunization programs can be implemented in practice settings with appropriate systems support. (*Am J Public Health* 1990; 80:534–536.)

Introduction

Influenza accounted for more than 10,000 excess deaths in the United States in 19 different epidemics between 1957 and 1986.[1] Yet, an influenza vaccine containing a strain similar to that causing epidemic disease has been found to be up to 80 percent effective at protecting the elderly, chronically ill population against hospitalization and death from acute respiratory disease during an influenza epidemic.[2] With the goal of reducing excess morbidity and mortality from influenza, the Immunization Practices Advisory Committee (ACIP) of the Centers for Disease Control recommends the annual vaccination of specific groups with chronic medical conditions who are at high risk for complications of influenza. In the early 1980s, the US Public Health Service's stated goal was vaccination of 80 percent of those at highest risk for morbidity and mortality from influenza infection. Unfortunately, estimates indicate that fewer than 25 percent of high-risk individuals were receiving an influenza immunization each year.[3]

Despite the good intentions of their physicians, Americans do not necessarily receive the benefits of available preventive health measures such as cancer screening and immunizations.[4] Several randomized controlled trials have shown that systems for physicians, such as reminders and feedback, can improve delivery of preventive measures.[5–7] In addition, postcard reminders to patients have been shown to increase influenza vaccination among elderly populations, including those in a health maintenance organization (HMO) setting.[8–10] Although these trials demonstrate the efficacy of practice support systems, there is little evidence that reminders and feedback actually have been adopted in practice settings or that they are effective in an ongoing practice-based program as distinct from a special study. The Health Centers Division of Harvard Community Health Plan (HCHP) is a large staff model HMO in the Boston area. Most of HCHP's health centers use a computerized medical record system for documenting ambulatory care.[10] This system has also been used as a tool in controlled trials of limited size to monitor clinician performance and provide feedback.[7,12]

In recent years HCHP has instituted a program to improve influenza vaccination of its high-risk members. The program includes educating members about the need for and availability of flu vaccine and consists of a series of systems for member reminders, physician reminders, and physician feedback on performance. The program has developed in increments. The HMO has not considered it to be research but rather part of its efforts to improve clinical operations and quality of care. This paper presents a description of the program as it has evolved, an evaluation of HCHP's influenza immunization performance and an assessment of the effectiveness of the reminder and feedback systems.

Methods

The membership of the Health Centers Division of HCHP grew from 178,000 in 1984–85 (Flu Season #1 in this evaluation) to 227,000 in 1986–87 (Flu Season #3). During that time, an average of approximately 100 internists practiced adult primary care at seven health centers in the metropolitan Boston area.

The first year's intervention involved postcard reminders to HMO members, educational materials (e.g., posters for waiting areas and examination rooms, pharmacy bag notices), and a reminder message to clinicians at the front of the summary of the computerized record which was prepared for each scheduled primary care visit for a high-risk patient under age 65. This was supplemented in the second year by chart reminders for persons over age 65 and feedback of performance to service chiefs. In the third year these actions were further supplemented by feedback of performance to individual physicians and by the periodic distribution to physicians of lists of patients not yet immunized.

In 1983–84, the year before the influenza vaccination program began, a survey of records of members age 65 and over revealed immunization rates of 24 percent in centers using the automated record and 22 percent in the one health center using a manual record. In order to detect change in vaccination rates over the three succeeding flu seasons, we selected a cohort of members representing the high-risk group under age 65 and a cohort of members age 65 and over.

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The latter were chosen from among those members who had belonged to HCHP for four or more years as of June 1987, had turned 65 by September 1984, and had designated a specific physician as their primary provider. Lists of members meeting these criteria were obtained for each health center and stratified by provider; a random sample was then selected from each center's list. By using computerized enrollment files it was possible to generate a similar list for the center not using the automated medical record.

The cohort of persons under age 65 at high risk for complications for influenza was identified from the computer-generated list used to send out reminder postcards in early autumn and to flag charts with a reminder message at the time of flu season visits. The computer algorithm used to create this list consists of a search for certain diagnostic codes corresponding to high-risk categories which were entered during a specific window of time in the prior year (roughly October 1-June 1 each year).

In order to assess the specific effect of a reminder, we focused on a single diagnostic group—persons with insulin-dependent diabetes: HCHP maintains a Plan-wide registry of insulin-dependent diabetics culled from diagnostic codes entered in the computerized medical records and also from all pharmacy slips recording prescriptions for insulin. In comparing this list with the flu vaccine reminder list for Flu Season #3, some members from centers with the automated record were identified who were not on the reminder list despite their having a high-risk diagnosis. Reasons for diabetic members not appearing on the flu vaccine reminder list include: joining HCHP after the flu vaccine reminder list had been made for the year, having no visit to HCHP during the window of time that computer records are searched for high-risk diagnoses, transferring care to another health center and thus a different member identification number, or having a very recent initial diagnosis of insulin-dependent diabetes mellitus (i.e., after the search window for creating the reminder lists).

Because the mean age of the non-reminder group (40.8 years) was significantly lower than that of the overall group of diabetics under age 65 on the vaccine reminder list (48.3 years), we used the subset of both groups between the ages of 40 and 65 for comparison of vaccination rates (respective mean ages ± SEM: 53.4 years ± .7 and 54.5 years ± .5).

Records of members at the computerized centers were searched by computer for the therapy code for influenza vaccine. Records for members at the non-automated center were reviewed by hand.

Data analysis was performed using SAS version 5.

Results

The overall vaccination rates at the computerized centers for the three flu seasons starting in 1984 are shown in Table 1. Vaccination rates increased substantially in both cohorts in Flu Season #3 (1986–87), which corresponded to the institution of the physician feedback mechanism. Immunization rates at the center without the automated record remained stable at 24–33 percent throughout the three flu seasons.

The focused study of diabetic members revealed that those on the reminder list were both more likely to have a visit during flu season and more likely to be vaccinated if they had a visit to their primary clinician (Table 2). The difference in visit rates probably reflects the effect of the postcard reminder on encouraging flu season visits. It is important to

TABLE 1—Influenza Immunization Rates in an HMO during Flu Seasons #1–3, 1984–1987

	Over Age 65 (n = 647)		High Risk, Under 65 (n = 198)	
Flu Season	% Immunized	Annual Change in Rate*	% Immunized	Annual Change in Rate*
#1	42		38	
		0 (−5, 5)		3 (−7,13)
#2	42		41	
		22 (17,27)		14 (4, 24)
#3	60		55	

*Difference between rates in successive seasons (95% confidence limits)

TABLE 2—Vaccination Status of Diabetic HMO Members, Ages 40–65

Vaccination Status	Reminder (%)	No Reminder (%)	Difference*
Vaccinated	55.9	27.0	28 (16, 40)
Patient refusal	0.0	1.8	−2 (−4, 0)
Not indicated	4.9	1.8	3 (−1, 7)
No flu season visit	23.8	33.3	−10 (−21, 1)
Not vaccinated despite a flu season visit	15.4	35.1	−20 (−33, −7)
Total in group	143 (100.0%)	111 (100.0%)	

*Difference in percentage between reminder and no reminder groups (95% confidence limits)

recall, however, that the reasons that prevented the "no reminder" group from being included on the computer's flu vaccine reminder list are directly related to lower visit rates in the previous year and these reasons may contribute to the continued lower visit rate in this group. In contrast, the difference in vaccination rates for those with visits—73 percent for those receiving the reminder versus 42 percent for those not getting the reminder—ought to reflect the effect of the computer-generated chart reminder on the physician.

Discussion

As noted at the outset, there have been several controlled studies of various interventions to improve performance of periodic health measures. All have shown efficacy of the specific intervention under study. None, however, has been a demonstration of sustained program effectiveness; nor has any of the studies attempted to determine the effect of additive interventions. The present report demonstrates that it is possible to achieve improvement in annual influenza vaccination performance in a practice setting using a combination of reminder and feedback techniques.

At least one other HMO has reported achieving performance similar to HCHP's but with a different combination of techniques. Group Health Cooperative of Puget Sound (GHC), which has a large elderly population, has reported vaccination rates for its members over age 65 of 48 and 53 percent in 1986–87 and 1987–88, respectively.[13] GHC sends reminder letters to its members over age 65, since they can be identified from automated enrollment files. The program runs mass vaccination clinics over a period of several weeks. Volunteer senior citizens help staff the clinics. The program, however, does not specifically target younger persons at high risk of complications of influenza.

HMOs can increase the cost-effectiveness of influenza vaccine delivery both by running mass immunization clinics and by appending the immunization process onto an encoun-

ter which was precipitated for another reason. In either case the cost per immunization delivered should be less than if a specific visit has to be set aside solely for vaccination.

The routine data on the flu program generated from HCHP's annual feedback show that in the centers with the computerized medical record approximately 70 percent of those members on the reminder list who visit their doctor during the flu season actually get the vaccine; 15 percent refuse; and for 15 percent there is no documentation of whether they were offered vaccine, got it, or refused. These are very positive results: many more HCHP members thought to be at greatest risk of complications from influenza, persons with chronic cardio-pulmonary disease under the active care of physicians, get vaccinated than national[3] or local[14] statistics indicate occurs elsewhere. Since physicians appear to be able to give the vaccine at HCHP and patients appear to be willing to accept it most of the time it is offered, neither physicians nor patients seem to be major barriers to more effective vaccination.

As a result of the evaluation, HCHP has continued the influenza immunization program, including: postcard and chart reminders for all members over age 65 and high-risk members under age 65, periodic distribution of lists of unimmunized members to clinicians during the vaccination season, and feedback of performance statistics to individual clinicians and chiefs of service. Since the basic computer programming of the reminders has been completed and the computer generates the lists and statistics for feedback, this influenza immunization program requires a minimum of effort to sustain.

ACKNOWLEDGMENTS

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ERRATUM

In: Marin G, Vanoss B, Perez-Stable EJ: Feasibility of a telephone survey to study a minority community: Hispanics in San Francisco. Am J Public Health 1990;80:323–326.

The name of the second listed author was incomplete as published. It should have read: Barbara Vanoss Marin. We regret that her last name was omitted on the table of contents cover and on the first page of the article.

Tobacco Excise and Declining Tobacco Consumption: The Case of Papua New Guinea

SIMON CHAPMAN, PHD, AND JEFF RICHARDSON, PHD

Abstract: Excise elasticities of demand for non-cigarette tobacco and cigarettes were calculated for Papua New Guinea for the 14 years 1973–86. Respectively, these were −0.50 and −0.71. This means that 10 percent increases in cigarette and non-cigarette tobacco excise are associated with an estimated 7.1 percent fall in demand for cigarettes and a 5 percent decrease in the demand for tobacco. As tax (excise) elasticities would normally be significantly smaller than price elasticities, these results imply that the consumption of cigarettes and tobacco in Papua New Guinea are more responsive to prices than in the United States and other Western countries. The level of excise is therefore an important and practical instrument for the control of consumption. These elasticities appear to be the first reported for a developing country. It is suggested that if they are indicative of consumer behavior in lower income countries then increasing tobacco and cigarette excise should be considered as an important strategy for the control of smoking in these countries which, because of their large populations, are huge markets for tobacco products and thus major targets for tobacco control measures. (*Am J Public Health* 1990; 80:537–540.)

Introduction

An expert working group convened by the American Heart Association recently recommended an increase in the rate of excise taxation on tobacco products and the resulting increase in tobacco prices as the leading priority for the United States among a range of smoking control strategies.[1] The success of the strategy would be directly proportional to the elasticity of demand which is defined as the percentage change in quantity demanded divided by the percentage change in price. Recent studies indicate that the price elasticity of demand for cigarettes by adults in the USA is approximately −0.4, meaning that a 10 percent increase in price should create a 4 percent decrease in demand.[2] A substantial portion of this decrease seems to result from a decrease in smoking prevalence through cessation and falling incidence, rather than through less smoking by continuing smokers.[2]

The elasticity of demand for cigarettes has been shown to be considerably greater in groups with lower levels of disposable income, namely teenagers (−1.4)[3] and people of low socioeconomic status[4]; such groups will stop smoking at a faster rate than those with higher disposable incomes if the price of cigarettes rises, and they may be influenced to not start smoking because of the additional financial burden. However, no studies appear to have been published on the effects of price changes on demand in developing countries, where disposable incomes are typically very low. Despite this, per capita consumption of cigarettes and smoking prevalence in some places is rising at alarming rates,[5] and the promotional activities of the tobacco industry are generally unchecked by any government regulations.[6]

Smoking in Papua New Guinea

Papua New Guinea (population 4 million) is located immediately to the north of eastern Australia. In 1987, it had a gross national product of per capita of $700(US), placing it 48th (equal with Swaziland) in the World Bank's ranking of poor countries.[7] Its population is predominantly Melanesian and it is governed from the coastal city of Port Moresby. Civil unrest and change in government have been common since independence. No national smoking prevalence surveys have been undertaken, although a 1981 survey conducted in two regions found rates as high as 85 percent among coastal Papuan men and 80 percent among highland women from Goroka.[8] The last published Household Expenditure Survey (1975/76) found that tobacco products accounted for 5.2 percent of average household expenditure, placing them after rent (10.5 percent), beer (7.2 percent), rice (5.6 percent), transport (5.3 percent) as the fifth most significant item in weekly expenditure.[9]

Smoking control policies and programs in Papua had been non-existent until the introduction of the Tobacco Products (Health Control) Act 1987 in November of that year.[10] The Act banned tobacco advertising; reportedly this ban has not been widely implemented. There are no health warnings on cigarette packs or advertisement, health education about smoking is negligible, and there has been neither public debate nor any regulation of smoking in public areas. Tobacco advertising and sports sponsorship both remain widespread, with the most remote bush trading posts typically being bedecked with posters advertising brands marketed by two Australian subsidiaries of transnational tobacco companies, i.e., W. D. and H. O. Wills and Rothmans. Cigarette advertising is almost certainly the most common form of outdoor advertising although no data are available on expenditure and consequently could not be included in our analysis.

Methods

Demand equations are usually estimated by regressing the quantity of the commodity demanded against the price of the commodity, the price of its close substitutes, and disposable income. Depending upon the commodity and the purpose of the analysis, other variables which influence demand may be included, such as advertising and health education expenditures.

In order to estimate the impact of taxes on consumption, a second equation must be estimated relating the level of taxation to the price level. This, in conjunction with the results of the demand equation may be used to infer the tax-consumption relationship. Regrettably, this conventional methodology could not be applied to the Papua New Guinea

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experience. Our inquiries about the contributions of wholesale and retail mark-ups to retail price were fruitless. Instead, the government statistical office in Port Moresby was able to provide information on the volume of cigarettes and non-cigarette tobacco consumed and the excise revenue obtained from these for the years 1973–86 (Table 1).

The use of excise rather than price data makes direct comparison of these findings with those obtained in other countries problematic although we believe our conclusions have important implications for policy since it is excise, and not usually retail price, that governments are able to manipulate directly. As excise tax is only a portion of retail price, excise elasticity is likely to be less than and so an *understatement* of the full price elasticity, thus suggesting that the elasticities shown below are quantitatively smaller than the relationship between retail price and demand. The two equations referred to above may be combined into a single equation which directly relates consumption to taxation. More formally, equations (1) and (2) below (which are usually estimated in demand studies) imply equation (3) which is the subject of the analysis here.

$$\text{Consumption} = f_1(\text{price}, X) \quad (1)$$

$$\text{Price} = f_2(\text{excise}, Y) \quad (2)$$

hence

$$\text{Consumption} = f_3(\text{excise}, X, Y) \quad (3)$$

where X and Y are sets of other relevant variables.
Equation (3) is a "reduced form" equation. Unlike the structural equations (1) and (2), it does not describe the direct, behavioral relationships between variables. More specifically, equation (1) embodies the fact that consumers react directly to market price and not to excise taxes. However, as retail price reflects taxes there is an indirect relationship which may be legitimately estimated. The resulting "excise elasticity" would not indicate the price elasticity of demand except under particular circumstances, i.e., when prices rise in direct proportion to taxes. If wholesale prices rose proportionately less than taxes (or remained unchanged or fell) then the percentage change in price would be less than the percentage change in tax and the excise elasticity would underestimate the magnitude of the price elasticity.

TABLE 1—Tobacco and Cigarette Consumption, Excise Tax Rate and Consumption Expenditure per Capita, Papua New Guinea 1973–86

Date	Quantity Tobacco kg/capita	Quantity cigarettes kg/capita	Excise Tax Rate Tobacco kina/kg	Excise Tax Rate Cigarettes kina/kg	Consumption Expenditure per Capita
1973	0.53	0.26	2.05	15.22	319.65
1974	0.39	0.22	2.45	20.03	263.56
1975	0.25	0.36	3.97	13.84	273.17
1976	0.25	0.33	4.85	16.51	281.66
1977	0.30	0.35	4.64	15.81	309.43
1978	0.29	0.38	4.46	15.31	326.11
1979	0.28	0.44	4.30	15.25	340.91
1980	0.26	0.44	4.05	13.45	355.07
1981	0.25	0.40	3.75	13.06	334.84
1982	0.27	0.34	3.53	15.78	313.69
1983	0.24	0.28	3.29	16.38	318.04
1984	0.26	0.30	3.07	16.04	317.48
1985	0.25	0.26	2.95	16.50	312.57
1986	0.27	0.26	2.80	16.37	—

The estimated "excise elasticity" would be biased if excise taxation correlated with one of the other variables and this variable was not included in the estimation procedure. The most important "other variables" in such analyses are the level of personal income per capita and the cost of production. As no information was available on personal income, data were obtained on a closely related measure, i.e., private consumption per capita. No data were available to act as a proxy measure for production costs. However, it appears most unlikely that these would correlate with annual changes in excise tax. Consequently, the regression coefficient should produce an unbiased estimate of the excise elasticity.

The data in Table 1 were used to estimate "demand" equations using ordinary least squares regression techniques and a multiplicative model shown below.

$$Q_i = e^{a+\beta 4T} E_i^{\beta 2} E_j^{\beta 2} C^{\beta 3}$$

where:
a = constant estimated by the regression
$\beta 1, \beta 2, \beta 3, \beta 4$ = coefficients estimated by the regression. Because of the multiplicative model, $\beta 1, \beta 2, \beta 3$ are the elasticity coefficients
Q_i = quantity of product i per capita (in kg)
E_i = excise per kg of product i (inflation adjusted kina)
E_j = excise per kg of the substitute product j
C = total consumption expenditure per capita on all goods and services
T = a trend variable (1 in 1973; 2 in 1974; 2 in 1975 etc)
a_o = constant
The multiplicative model is used as it has simple but acceptable theoretical properties and it may be readily estimated with a logarithmic transformation of the variables.

The resulting coefficients $\beta 1$-$\beta 3$ may be interpreted as "excise elasticities" and "cross excise" elasticities. Thus, for example, in the equation for cigarettes, a 10 percent change in excise E_1 will be associated with a $10\beta_1$ percent change in the consumption of cigarettes (Q_1); a 10 percent change in the excise on the substitute, tobacco, will result in a $10\beta_2$ percent change in consumption. As it is the level of excise tax which may be directly varied by government and not generally consumer prices, the model is relevant for the policy issue of controlling consumption.

Results

Results for the two demand equations are reported in Table 2. A very high percentage of the variation in the quantity demanded is explained by the variables in the model as would be expected in a time series analysis. The "income" elasticity in both cases is positive and has a plausible magnitude. Surprisingly, the income effect for cigarettes is smaller than that for non-cigarette tobacco. As expected in the case of non-cigarette tobacco, there is a negative value for the trend variable. The coefficient suggests that, after allowing for the effect of the other variables in the model, non-cigarette tobacco consumption has declined about 5

TABLE 2—Regression Equation Results for the Quantity of Tobacco and Cigarettes per Capita

	Q (tobacco)		Q (cigarettes)	
Dependent Variable	B	SE	B	SE
Tobacco excise	−0.50*	0.08	0.50	0.09
Cigarette excise	0.62	0.22	−0.71	0.26
Income	1.37*	0.30	0.86	0.34
Trend	−0.05*	0.006	−0.008	0.006
Constant	−9.84	2.13	−4.68	2.45
R^2	0.95		0.93	
Standard Error	0.06		0.07	
dW	2.30		2.19	
n	14		14	

Notes: The first column of each result gives the estimated regression coefficient ( ) and the second column, the Standard Error. the logarithm of all variables has been used.
T (Trend variable) = 1, 2, 3, . . . 14

percent per annum.* No trend is apparent for cigarettes. The most important results in Table 2 are for the excise variables. For tobacco and cigarettes the excise elasticities are −0.50 and −0.71, respectively.** In both cases, excise "explains" a very high proportion of the variation in the quantity demanded. The results indicate that there was a strong relation between declining per capita cigarette consumption and rising excise. All else being equal, a 10 percent increase in cigarette excise has been associated with a 7.1 percent fall in cigarette consumption; and a 10 percent increase in non-cigarette tobacco excise with a 5 percent in its consumption. The "cross excise elasticities" (which due to the limited data have been unconstrained) are 0.62 for non-cigarette tobacco and 0.50 for cigarettes. That is, there is the expected positive sign in both cases.

Discussion

On the basis of the evidence presented here it would appear that the elasticity of demand for cigarettes and tobacco in Papua New Guinea is much greater than has been estimated for the USA and other Western nations in recent years. As excise is only a fraction of the final retail price, a change in excise would normally be expected to cause a proportionately smaller change in price which implies that the price elasticity exceeds the excise elasticity of demand. Irrespective of the relative magnitude of these coefficients, the present results suggest that the consumption of tobacco products can be manipulated through variations in the excise rate. If these demand characteristics are typical of developing countries, this finding may have enormous implications for smoking control in the Third World. Such countries often have diverse linguistic subcultures which mitigate against mass communication in health education. Being oriented toward problems of infectious disease and malnutrition, their health services typically accord low priority to smoking control. With the exception of a small number of developing countries in which tobacco is a major industry—e.g., Brazil, Zimbabwe, Malawi, Thailand, India—most developing nations have a deficit balance of trade in tobacco, with imports requiring the expatriation of growing amounts of hard currency to the exporting transnationals or state monopolies.[6]

Tobacco's main attraction to such economies lies in the ease with which its consumption can be taxed. However, excise taxes represent only transfer payments within economics whereas money lost overseas in paying for tobacco imports represents a genuine drain on a nation's balance of payments. As such, countries such as Papua New Guinea with deficit tobacco trade should not balk at easily implemented measures that reduce tobacco consumption if the resultant economic and public health advantages could be effectively communicated to key officials and "extraneous" factors like corruption and bribery exposed or otherwise circumvented.

In the short to medium term, an excise-led smoking control policy can reduce smoking and simultaneously satisfy Treasury requirements for what is an easily collected and bountiful source of revenue. Our results suggest that a 10 percent increase in cigarette excise will decrease consumption by about 7.1 percent, therefore increasing total excise collections. A 10 percent increase in tobacco excise will similarly increase total collections as the percentage increase in excise exceeds the percentage decrease in the volume consumed. Eventually, a threshold would be reached where the decline in consumption caused by rising prices would produce diminishing total excise. However, this point will, in many cases, be well in the future and therefore of less concern to political decision-makers than the attractions of both a short-term gain to state finances and falling smoking prevalence.

By World Bank projections,[7] the population of developing countries by year 2000 will constitute 80 percent of world population. Further, the aggregate rate of increase between 1987–2000 of the populations of developing countries is estimated to be 28 percent compared to a forecast increase of only 8 percent in the population of industrial market economies. Cigarette consumption is decreasing in the industrialized world at about 1 percent per year, whereas in Asia and Latin America it is growing 7 percent faster than the population and, in Africa, 18 percent faster. If the results of the excise elasticity analysis we have provided are found to be typical of those in developing countries, then excise policy could be a powerful instrument in controlling smoking in these vast markets. Its use could reduce tobacco consumption rapidly, possibly faster than any other realistic instrument of public health policy and hence produce health benefits for many millions of people.

*The results are not the consequence of a correlation between income and trend. The coefficient of a correlation between these two variables is only 0.206. More generally, the correlation table does not suggest a problem of multicollinearity. (Data available on request from the authors.)

**This latter coefficient is increased to −1.08 if two further years (1971, 1972) are added to the data set. Excise data provided for these two years were outliers (33.98 and 34.49 kina/kg) and in the absence of any explanation for their large difference, we have omitted them from our main analysis, suspecting typographical error.

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WHO Fellowships for Travel/Study Abroad

The World Health Organization (WHO) will provide a limited number of short-term fellowships for use during 1991 for travel/study abroad related to the improvement of health status in the United States. This support is limited to US citizens employed in the health field.

Fellowship awards are offered to persons employed in educational or service aspects of the health field with government agencies, educational institutions, or non-profit organizations. Applications cannot be considered for basic laboratory bench research or attendance at international meetings or seminars. In addition, undergraduate or graduate students including medical interns and residents, self-employed health care providers, or individuals employed by profit-making organizations are not eligible.

The fellowship award provides per diem, transportation, and a small stipend to cover miscellaneous expenses. Employers of applicants are expected to endorse the application and agree to continue the applicant's salary throughout the fellowship period. Except in unusual circumstances, the fellowships will be limited to short-term programs of one or two months. The number of fellowship awards will be governed by the funds available.

The *deadline* for the submission of the completed application is September 30, 1990.

Further information and application form may be obtained from the Secretary, WHO Fellowships Selection Committee, Health Resources and Services Administration, 5600 Fishers Lane, Room 14-14, Rockville, MD 20857. Telephone: (301) 443-6152.

Smoking Before, During, and After Pregnancy

LOIS A. FINGERHUT, MA, JOEL C. KLEINMAN, PHD, AND JULIETTE S. KENDRICK, MD

Abstract: We report the first national data on smoking before, during, and after pregnancy. Estimates are based on the 1986 Linked Telephone Survey that reinterviewed 1,550 White women 20–44 years of age who were respondents to the 1985 National Health Interview Survey. An estimated 39 percent of White women who had smoked before pregnancy quit smoking while pregnant (27 percent when they found out they were pregnant and 12 percent later during pregnancy). Women with less than 12 years of education were five times as likely to smoke and one-fourth as likely to quit as those with 16 or more years of education. Women who smoked more than one pack of cigarettes per day before pregnancy were one-fifth as likely to quit as those smoking less. Of the women who quit, 70 percent resumed smoking within one year of delivery. Of those who relapsed, 67 percent resumed smoking within three months of delivery and 93 percent within six months. There is little evidence of educational differentials in relapse rates. The fact that relapse remains high suggests that while health of the fetus is a strong influence on women's smoking habits, women may be less aware of the effect of passive smoke on the infant. (*Am J Public Health* 1990; 80:541–544.)

Introduction

Increased risks of fetal and infant death and low birthweight have been linked to maternal smoking[1–3]; even if smoking is stopped late in pregnancy, there will be a positive effect on the infant's birth weight.[4,5] The effects of passive smoke on nonsmokers, and especially on young children, have also been shown to be harmful.[6–9]

For those women who smoke, pregnancy can be viewed as an ideal time for cessation: quitting can affect the health of their unborn child as well as their own health. It is also a time when contact with health professionals is very frequent. Thus there is substantial opportunity for health providers to have an impact on maternal and infant health through smoking cessation.

Among pregnant women, the proportion of married mothers 20 years of age and over who smoked before their pregnancy decreased between 1967 and 1980, while the proportion of those women who quit smoking after they found out they were pregnant increased.[10]

In this study, we report national data, the first ever available, regarding the frequency and timing of smoking relapse among women who quit smoking while pregnant.

Methods

Data Sources and Limitations

The National Health Interview Survey (NHIS) is a multistage stratified probability sample of the household population of the United States. In 1986, a linked telephone survey was conducted that reinterviewed respondents to the 1985 NHIS. The primary purpose of the telephone survey was to test methodology[11]; it included a battery of questions on smoking before, during, and after pregnancy. The target sample was selected from women responding to the NHIS during January–June 1985: nearly 2,000 women 18–44 years of age who either were pregnant at the time of the 1985 NHIS or whose most recent birth occurred during the five years preceding the NHIS. The women were interviewed using a computer-assisted telephone interview system during March–May 1986.

Definitions

- *Women who smoked prior to pregnancy* are those women who said that they smoked at least one cigarette per day during the 12 months before they found out they were pregnant. Women who said that they did not smoke regularly were not included as smokers prior to pregnancy.
- *Women who quit smoking while pregnant* are those who smoked prior to pregnancy and "quit early" (quit when they found out they were pregnant) or "quit later" (quit at a later time during their pregnancy). Those who quit early were not asked whether they resumed smoking prior to delivery. However, data from a large clinical trial of smoking during pregnancy show that very few women who quit early in pregnancy resume smoking before delivery.[12] Those who quit later continued to smoke cigarettes after they found out they were pregnant, but said that they quit for at least a week during their pregnancy and did not start smoking again before delivery; they were not questioned about when quitting occurred.
- *Women who relapse* are those who quit while pregnant and then resumed smoking within one year of the delivery.
- *Postpartum smokers* include women who relapsed within one year of delivery; women who continued smoking during the entire pregnancy and were still smoking within one year of the delivery; and women who smoked during the year after delivery even though they had not smoked in the 12 months prior to delivery.

The number of cigarettes smoked before pregnancy was dichotomized into less than 20 vs 20 or more cigarettes per day. Age of mother, education, and marital status were determined at the time of the 1985 NHIS.

Response Rates

The response rate to the first six months (January–June) of the 1985 NHIS was 96 percent.[13] Of these respondents, 86 percent completed the telephone followback survey yielding a sample of 1,918 women. Non-respondents included primarily refusals and those women with changed telephone numbers who could not be tracked.[11] In addition to excluding nonrespondents, 28 sample women who did not respond to

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the entire battery of smoking questions were omitted from the analysis.

As there were relatively few Black women in the sample (n = 278), this analysis is limited to White women. Further, there were only 35 White teenagers in the original sample and because teenagers are known to have different smoking habits than adults,[14] the analysis is limited to White women 20–44 years of age. The overall response rate for these women was 88 percent, yielding a final sample of 1,550 women.

For White women 20–44 years of age, response rates were higher for married than for unmarried women (90 vs 83 percent), and for those with at least a high school education than for those with less education (91 vs 78 percent). All percentages are weighted to reflect national estimates of women in the civilian noninstitutionalized population of the United States.

Analysis

The proportions and standard errors in this analysis were computed using the SESUDAAN program that takes into account the complex sample survey design of the NHIS.[15] Multiple logistic regression analysis was performed using the Proc Logist procedure within SAS.[16] Because the design effects were small (less than 1.2, based on the ratio of standard errors from SESUDAAN to simple random sample standard errors), standard errors were approximated using weighted estimates in the logistic regression procedure normalized to reflect the sample size (NORMWT option in SAS). The results of the logistic regression are presented as adjusted odds ratios with 95 percent confidence intervals calculated by taking the anti-logarithms of the regression coefficients and corresponding confidence intervals.

Results

One-third of White women 20–44 years of age said that they smoked cigarettes prior to pregnancy. Older women were less likely to smoke than younger women; and married women were much less likely than unmarried women to have been smokers. The prevalence of smoking decreased sharply with increasing education (Table 1).

Using multiple logistic regression to control for age and marital status, White women with less than 12 years education were five times as likely as college graduates (16 or more years education) to have smoked prior to pregnancy. Similarly, White unmarried women (controlling for age and education) were twice as likely as married women to have smoked. Women in their 20s were about 40 percent more likely to smoke than women in their 30s and early 40s (Table 1).

Of the White women who said they smoked, 39 percent indicated that they quit while pregnant (27 percent early and 12 percent later during pregnancy) (Table 2). The Health Promotion and Disease Prevention (HPDP) supplement to the 1985 NHIS estimated that 21.2 percent of all women 18–44 years quit smoking when they learned they were pregnant.[17] Further analysis of the HPDP data show that among White women 20–44 years of age, 24.2 percent quit smoking early in pregnancy. This estimate is not significantly different from our estimate of 27.4 percent.

Sixty percent of college graduates quit compared with 28 percent of those with less than 12 years education. Women who were lighter smokers prior to pregnancy were much more likely to quit than were heavier smokers.

Based on the logistic regression results, the relation between quitting and education was nearly as strong as between smoking and education. The amount smoked prior to pregnancy was also strongly associated with quitting. Age and marital status had little effect on quitting (Table 2).

In all, 70 percent of White women who quit smoking while pregnant resumed smoking within one year. This may be a slight underestimate since there were eight women who quit smoking while pregnant but who had less than a year of exposure time—from delivery to interview—in which to relapse and therefore were not considered relapsers. Unlike "smoking prior to pregnancy" and "quitting," there is little association between education and relapsing (Table 3). However, one year relapse rates are higher for those who quit later during pregnancy as opposed to those who quit when they found out they were pregnant (81 vs 66 percent).

Based on the regression results, younger women, women who were heavy smokers prior to pregnancy, and women who quit smoking later during pregnancy were more likely to relapse. However, the relatively small numbers of women who quit smoking (n = 191) result in rather wide

TABLE 1—Prevalence of Smoking Prior to Pregnancy among White Women 20–44 Years of Age

	Sample Size*	Percent Smoking	Standard Error	Adjusted Odds Ratios** (95% CI)
All women	1550	32.0	1.3	—
Age (years)				
20–29	832	37.5	1.7	1.38 (1.09, 1.74)
30–44	718	25.1	1.7	ref
Marital Status				
Unmarried	127	52.5	5.1	2.01 (1.37, 2.94)
Married	1423	30.1	1.5	ref
Education (years)				
Less than 12	165	54.1	4.3	4.99 (3.26, 7.65)
12	679	37.4	1.9	2.71 (1.94, 3.77)
13–15	353	26.7	2.4	1.73 (1.19, 2.52)
16 or more	352	16.1	1.9	ref

*Sample size is number of White women 20–44 years of age. Education was missing for one woman.
**Based on multiple logistic regression.

TABLE 2—Smoking Cessation during Pregnancy among White Women 20–44 Years of Age

	Sample Size*	Percent Quitting	Standard Error	Adjusted Odds Ratios** (95% CI)
All women	486	39.3	2.3	—
Age (years)				
20–29	308	38.3	2.8	1.05 (0.68, 1.61)
30–44	178	41.1	4.0	ref
Marital Status				
Unmarried	64	36.6	7.2	0.88 (0.49, 1.60)
Married	422	39.7	2.4	ref
Education (years)				
Less than 12	86	27.5	4.9	0.26 (0.12, 0.56)
12	249	37.0	3.0	0.39 (0.21, 0.75)
13–15	93	44.4	5.5	0.51 (0.25, 1.07)
16 or more	57	60.1	7.0	ref
Amount smoked before pregnancy				
<1 pack/day	204	59.0	3.6	ref
1+ packs/day	281	24.6	2.7	0.22 (0.15, 0.33)

*Sample size is number of women who smoked prior to pregnancy. Education and amount smoked were missing for one woman.
**Based on multiple logistic regression.

TABLE 3—One-year Relapse among White Women 20–44 Years of Age Who Quit Smoking while Pregnant

	Sample Size*	Percent Relapsing	Standard Error	Adjusted Odds Ratios** (95% CI)
All women	191	70.5	3.6	—
Age (years)				
20–29	121	74.0	4.0	1.94 (0.95, 3.94)
30–44	70	64.4	7.0	ref
Marital Status				
Unmarried	24	64.3	9.1	0.72 (0.27, 1.90)
Married	167	71.4	3.8	ref
Education (years)				
Less than 12	25	58.2	10.1	0.36 (0.10, 1.22)
12	92	75.2	5.7	1.05 (0.42, 2.66)
13–15	41	68.9	7.1	0.73 (0.25, 2.10)
16 or more	33	68.5	7.9	ref
Amount smoked before pregnancy				
<1 pack/day	121	66.6	4.5	ref
1+ packs/day	69	76.8	5.3	1.58 (0.77, 3.25)
Quit Time				
Early	131	65.9	4.6	ref
Later during pregnancy	60	81.0	5.4	2.15 (0.96, 4.82)

*Sample size is number of women who quit smoking while pregnant. Amount smoked was missing for one woman.
**Based on multiple logistic regression.

confidence intervals for all of these estimates (Table 3). Age of mother and time of quitting (early vs later) were the only factors for which the confidence intervals came close to excluding 1.0.

Of all White women who quit smoking while pregnant, 27 percent relapsed within a month of the delivery, and 52 percent relapsed within 4 months (Figure 1). Those women who quit later during pregnancy resumed smoking much sooner than women who quit smoking earlier in their pregnancy; 69 percent of the former group relapsed within three months of the delivery compared with 37 percent of the latter group of women.

The net effect of quitting and relapsing is the prevalence of smoking one year postpartum. The estimate includes women who relapsed within a year, those who did not quit during their pregnancy, and those who smoked postpartum but said they were not smoking before or during their pregnancy. (This latter group is small—only 2 percent of the total sample.) The great majority, 87 percent, of the women

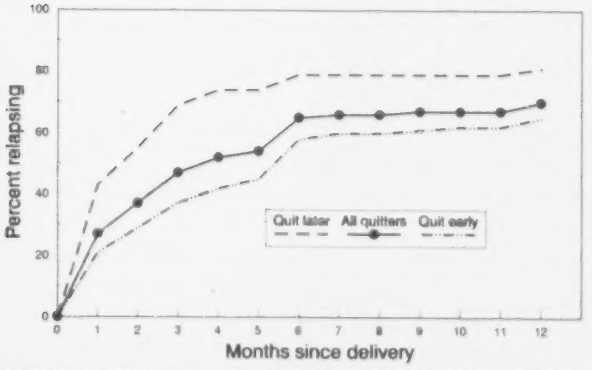

FIGURE 1—Relapse Rates among White Women Ages 20–44 Years Who Quit Smoking While Pregnant (data are from the 1986 Linked Telephone Survey)

who smoked prior to pregnancy were also smokers one year after delivery. Hence, the prevalence of smoking one year after the delivery was only slightly lower than before pregnancy; 30.3 vs 32.0 percent. Postpartum smoking has similar associations with age, marital status and education as shown in Table 1 for smoking prior to pregnancy.

Prenatal Care and Smoking

Nearly all (99 percent) of the women said that they received prenatal care during their pregnancy. Those who smoked prior to pregnancy were less likely to begin prenatal care in the first trimester than those who did not smoke during this period (90 percent vs 95 percent). Those smokers who quit while pregnant were more likely to have begun care in the first trimester than those who did not quit, 94 vs 88 percent.

Regardless of whether a woman went to a clinic or to a private physician for prenatal care, about three-fourths of the smokers prior to pregnancy said that a doctor had advised them to quit or cut down on smoking during their pregnancy (57 percent who quit smoking and 83 percent who did not quit).

In response to a question about different methods used to quit or help cut down on smoking, about 95 percent of the women who quit smoking stated that they quit entirely on their own, without a formal program or counseling. While some of the women also said that they stopped with the help of friends and that they switched to low tar and nicotine cigarettes to help themselves quit, clearly the vast majority, regardless of whether they quit early or later during pregnancy, attributed their behavior to their own initiatives.

Discussion

The potential effect of recall bias on the results of this study should be considered. Because of the survey design, a woman may have been asked to recall her smoking habits that occurred five or six years earlier. About 22 percent of the women in the sample delivered in 1980–81, 56 percent from 1982–84, and 22 percent in 1985–86. There was a decrease in the proportion of White women smokers between 1980 and 1985–86, similar to the decline observed in the NHIS data collected during those years.[18] Moreover, smoking history recall over a 10-year period was found to be quite reliable.[19] Thus, recall bias in the current study should not have a major effect on the conclusions.

Nearly all women in this sample received prenatal care and most smokers reported that they had been advised by a doctor to quit smoking. Those who did not quit were, in fact, more likely than quitters to report such advice (83 vs 57 percent); this apparent paradox probably reflects their longer opportunity to be given this advice on repeated prenatal visits. We cannot distinguish the role that counseling during prenatal care might have played in encouraging women to quit; it is possible that women who quit early did so in response to counseling that occurred at their first prenatal visit when they were told they were pregnant. Several previous studies have documented that the advice of a doctor or nurse can be very important in encouraging smokers to quit.[20] A variety of materials are available to health providers who want to provide smoking cessation help to their smoking patients.[21]

We found strong educational differentials in smoking prevalence and in quitting. This reinforces the conclusions of a recent study of smoking prevalence in the general population.[22] Between 1974 and 1985 smoking declined five

times faster among college graduates compared with those with less than a high school education.

Pregnant women appear to quit smoking at a greater rate than the general population. Based on data from the 1986 Adult Use of Tobacco Survey, an estimated 28 percent of persons 17 years of age and over who had smoked in the preceding 12 months quit smoking for at least one week during the year.[1] The proportion of White women who quit for at least a week while pregnant is higher—39 percent.

Relapse rates among women who quit smoking during pregnancy appear to be somewhat less than among quitters in the total population. In general, about 80 percent of smokers who quit resume smoking within a year. This is similar to the relapse rates for other drug dependencies.[23] In our study, 81 percent of White women who quit late in pregnancy also resumed within a year, but only 66 percent of those who quit early resumed smoking again within a year. Thus for some women, pregnancy appears to be an event that could be more likely to result in longer-term abstinence than smoking cessation for other reasons.

It is also noteworthy that women who quit smoking while pregnant resume smoking after delivery at a similar pace to quitters in the general population. In both instances, there is a precipitous rise in the proportion who resume smoking by three months followed by a relatively smaller increase by six months. The likelihood of resuming after six months is less than during the preceding months.[23] The underlying difference between these two groups, however, is that these pregnant women were abstinent during their pregnancy before they resumed smoking again.

However, the fact that relapse rates are high suggests that while the fetus's health is a strong influence on women's smoking habits, women may be less aware of the effect of passive smoke on the infant. Children of parents who smoked are more likely to have a variety of health problems such as respiratory illnesses than children of parents who do not smoke.[6–9] Education about the deleterious effects of infant and childhood exposure to passive smoke should be an essential part of comprehensive prenatal and postpartum care. Intervention programs for pregnant smokers should focus on assisting them to quit permanently.

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Screening for *Chlamydia Trachomatis* in Adolescent Males: A Cost-Based Decision Analysis

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Abstract: To evaluate the cost and benefits of screening tests for *Chlamydia trachomatis* in adolescent males, we developed a decision analysis model and compared the leukocyte esterase urine dipstick test with culture, with direct-smear fluorescent antibody (DFA), and with the option of no screening (no treatment). The leukocyte esterase test has the lowest average cost-per-cure ($51) compared with direct-smear fluorescent antibody ($192) and culture ($414). Compared with the DFA, we estimate that the leukocyte esterase test saves over $9,727 per cohort of 1,000 sexually active adolescent males screened. Sensitivity analyses show the leukocyte esterase test results in a lower cost-per-cure and lower overall costs (per cohort) than culture and direct-smear fluorescent antibody at any prevalence of *C. trachomatis* infection, and lower overall costs (per cohort) than no screening at prevalences above 21 percent. (*Am J Public Health* 1990; 80:545–550.)

Introduction

Infections caused by *Chlamydia trachomatis* are among the most prevalent and costly in the United States. Over four million chlamydial infections occur each year at a cost exceeding $1.5 billion dollars.[1,2] Adolescents have the highest rates of chlamydial infection and associated complications of any age group.[3–5] Considerable attention is now being focused on young women at risk for contracting *C. trachomatis* because of the dire reproductive consequences of a chlamydial infection. These include mucopurulent cervicitis, acute pelvic inflammatory disease, ectopic pregnancy, infertility, and maternal and infant infections during pregnancy and following delivery.[6–8]

Young men are equally at risk for a chlamydial infection. *C. trachomatis* causes approximately 50 percent of the reported cases of nongonococcal urethritis among males, and is also responsible for over 50 percent of cases of acute epididymitis in young men[8]; epididymitis is a painful, serious complication of chlamydial infection that could result in sterility. Moreover, infected adolescent males, particularly the 30 percent with asymptomatic chlamydial urethritis, represent the major source of *C. trachomatis* transmission to teenage females.[4,9]

Nevertheless, limited resources and limited availability of diagnostic tests have dictated the need to focus on screening females first. With newer, inexpensive and reliable tests now available, however, routine screening of males should be considered and evaluated. In this article, we evaluate the cost and benefits of the three commercially available screening methods for *C. trachomatis* in a population of adolescent males at risk for infection. We compare the leukocyte esterase urine dipstick test to urethral culture, to antigen detection* of a urethral smear, and with the option of not screening or treating.

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Editor's Note: See also related editorial p 531 this issue.

*Direct-smear fluorescent antibody (DFA) examination. Commercially available as Microtrak (tradename), manufactured by Syva Diagnostics.

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Methods

Decision Analysis Model

We used a decision tree (Figure 1) to evaluate the possible outcomes associated with three screening tests for *C. trachomatis* in a population of adolescent males: 1) leukocyte esterase (LE) urine dipstick with a 1+ or 2+ reading on first catch urine; 2) urethral culture; and 3) antigen detection* (DFA) or a urethral smear. These were compared with the option of no screening (no treatment). Baseline probabilities used in our model were obtained from published data (Table 1). Our denominator for calculating baseline outcomes and costs is a cohort of 1,000 sexually active adolescent males.

Data and Assumptions

Chlamydia Prevalence (Node 1): We used 15 percent as the baseline prevalence to be conservative in estimating outcomes, but we also performed sensitivity analyses** to test the effect of using different prevalences on our conclusions.[4,9–14]

Sensitivity (Node 2) and Specificity (Node 3) of Screening Test: We used a conservative sensitivity estimate of 75 percent.[10,11] Since many false positive dipstick tests will be due to the presence of *N. gonorrhoeae*, for which at least 85 percent of strains are susceptible to tetracycline,[15] and has sequelae for uncured cases similar to that of *C. trachomatis*, we assumed a specificity of 85 percent based on results of screening only asymptomatic males.[11] Test sensitivity and test specificity for culture of *C. trachomatis* were assumed to be 90 percent and 99 percent, respectively.[16] For the DFA test we estimated the sensitivity to be 80 percent and the specificity to be 97 percent.[17,18] We performed a sensitivity analysis of the LE test through the range of probabilities in Table 1.

Follow Up (Node 4): When there is a delay between taking a test and the availability of results, some patients will be lost-to-follow-up. We used a loss-to-follow-up rate of 3 percent (range 3 to 10 percent) for the culture and direct-smear (DFA) test, because in most clinical settings results are not available for one to three days after obtaining a specimen. This follow-up rate is based on a high yield, cost-effective, field follow-up method of tracking males with positive chlamydial tests.[19] Results from the LE urine dipstick are

**Sensitivity analysis is the process of varying assumptions in a decision analysis.

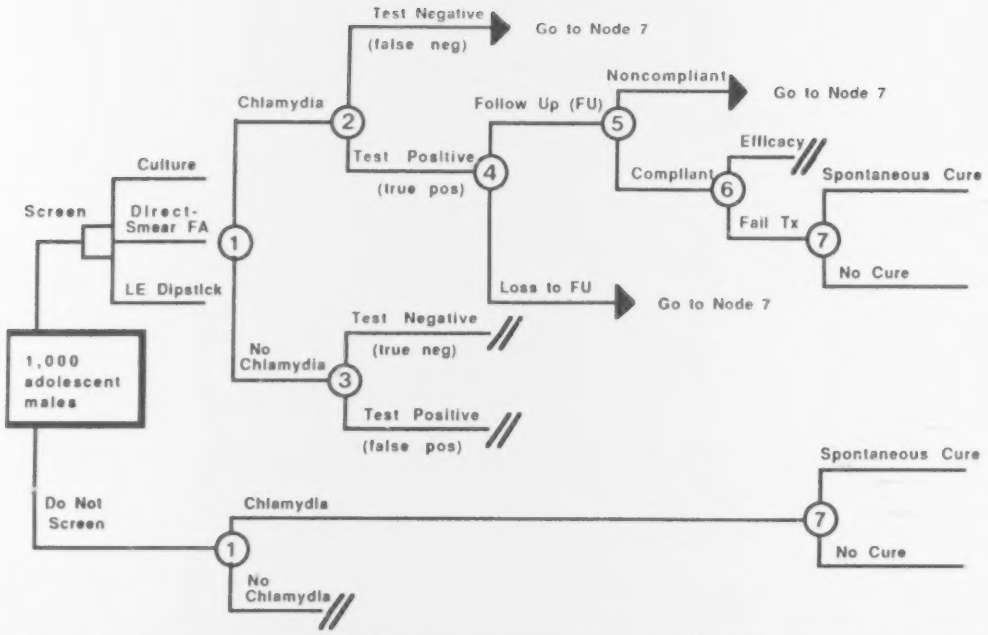

FIGURE 1—Decision Tree Depicting Outcomes for Screening (with three different tests) or not Screening for *Chlamydia trachomatis* in Adolescent Males. Squares indicate decision nodes; circles, chance nodes; and asterisk, sexually active adolescent males.

TABLE 1—Probabilities Used in Decision Analysis

	Probability		
	Baseline %	Range %	Reference
Prevalence of chlamydia	15	5–35	4,9–14
Sensitivity of leukocyte esterase	75	65–85	10,11
Specificity of leukocyte esterase	85	75–95	10,11
Sensitivity of culture	90		16
Specificity of culture	99		16
Sensitivity of direct-smear fluorescent antibody	80		17,18
Specificity of direct-smear fluorescent antibody	97		17,18
Loss-to-follow-up rate	3	3–10	19
Compliance with treatment	65	65–95	20–22
Effectiveness of treatment	95		15,23
Spontaneous cure rate	5		8
Pelvic inflammatory disease rate (infected partners)	20	10–30	3,26,29
Probability of infecting a female	30	20–40	19

generally available immediately, therefore we assumed no loss-to-follow-up for this screening test.

Compliance (Node 5): We conservatively estimated the compliance to be 65 percent (range 65 to 95 percent) in this population based on studies that show: a compliance rate of 66 to 79 percent of men treated with a five-day course of doxycycline for *N. gonorrhoeae*,[20] and an overall compliance rate for pediatric populations of approximately 50 percent, with a range of 20 to 80 percent.[21,22] We assumed that noncompliers had failed treatment, and that only those with spontaneous cure (5 percent) were free of disease.

Efficacy of Treatment (Node 6): Cure rates for the tetracyclines in treatment of chlamydial urethritis in males range from 91 percent to 99 percent.[15,23] We used a cure rate of 95 percent for our base-line analysis, the mean rate from published reports. Theoretically, other drug regimens such as trimethaprim-sulfamethoxasole[15] could also be used with the cost estimates adjusted accordingly.

Spontaneous Cure Rate (Node 7): We assumed a spontaneous cure rate of 5 percent for infected males who did not receive treatment.[8]

Cost Estimates

This decision model used medical care costs as utilities (Table 2). Only direct costs were considered with charges used to approximate costs.

Screening Test: We surveyed several laboratories in the San Francisco Bay metropolitan area to derive an average cost of $0.50 for LE dipstick (including the cost of the urine container), $30 for culture, and $10 for DFA. The cost of collecting the tests, performing the LE urine dipstick in the office, and of packaging the DFA and culture and sending them to the laboratory was assessed as $10.

Follow-up and Loss-to-Follow-up: The cost of tracking down clients with positive cultures or positive DFA tests and arranging an office visit has been shown to be $10.67.[19] Since a fair amount of time can be spent attempting to track down

TABLE 2—Cost Assumptions Used in Decision Analysis

Leukocyte esterase urine dipstick	$ 0.50
Culture	30.00
Direct-smear fluorescent antibody	10.00
Test collection and processing	10.00
Follow-up	10.67
Loss-to-follow-up	10.67
Treatment (doxycycline hyclate)	10.00
Complications in untreated males	34.34
Infecting a female partner	366.72

persons lost to follow-up, a cost of $10.67 was included for all clients with positive cultures or DFAs lost to follow-up.

Treatment: The $10 cost of treatment includes the cost of medication and the pharmacy charge. The current bulk cost for a seven-day treatment course of doxycycline hyclate 100 mg PO bid is less than $2.[24] We added in an additional $8 charge for dispensing, storing, and ordering the medication.

Complications in Sex Partners: The average cost of infecting a female partner, including transmission to neonates, was $366.72 using the baseline PID rate of 20 percent (Appendix Table A). This estimate is based on the probabilities shown in Table 1, and costs per incident of medical care in Table 2, and published data on the probability of chlamydial complications in women and neonates and associated costs.[1,2,25,26] The Appendix provides details of these calculations. This cost was decreased to $195.38 per female by decreasing the pelvic inflammatory disease (PID) rate to 10 percent, and increased to $538.05 per female by increasing the PID rate to 30 percent. We assumed that the probability of a male infecting a female was 0.30.[19] However, this variable was also ranged from 0.20 to 0.40.

Complications in Males: The cost of return visits for symptomatic infections in untreated males was assessed to be $45 ($35 office visit plus $10 treatment) with a probability of occurrence of 75 percent. This means that only about 56 percent (1×.75×.75) of infected men will return, because 25 percent will be asymptomatic. The cost of outpatient treatment for epididymitis was assessed to be $50 with a probability of occurrence of 3.6 percent in untreated males.[2,8] The cost of inpatient treatment for epididymitis was calculated to be $1,836 with a probability of occurrence in 0.4 percent of untreated males.[2,8] This results in a total cost for complications per untreated male of $34.34.

Results

Cost Savings at Baseline Assumptions

When total medical costs and cost-per-cure are considered, screening with the LE test results in the greatest cost savings (Table 3). Screening with the LE test costs 12 percent as much as culture and 27 percent as much as the DFA. The average cost-per-cure of a chlamydial infection is $51 when the LE test is used compared with $414 per cure with culture and $192 per cure with the DFA. This cost-per-cure is unchanged by decreasing the cohort of adolescent males screened to 100. Overall, screening with the LE test will save over $28,459 per 1,000 adolescent males at risk for *C. trachomatis* infection compared with culture and $9,727 compared with the DFA.

TABLE 3—Overall Cost and Outcomes for Different Screening Strategies*

Screening Strategy	Cost		Health Outcome	
	Total	Per Cure**	Cure (%)	Unnecessary Rx
Leukocyte esterase	$23,944	$ 51	73 (49%)	219
Culture	52,403	414	84 (56%)	15
Direct-smear fluorescent antibody	33,671	192	76 (51%)	44
No screening	20,571	—	8 (5%)	0

*Per cohort of 1,000 sexually active adolescent males screened for *C. trachomatis* infection.

**Cost-per-cure = (total costs screening method minus total costs no screening)/(total cures screening strategy minus total cures no screening).

TABLE 4—Sensitivity Analysis on Seven Variables

Variables	Percent	Cost/Cure LE	Cost/Cure DFA
Prevalence			
	35	$ (−)53	$ 19
	5	$ 415	$ 795
LE Sensitivity/Specificity			
	85/95	$ 19	$ 192
	65/75	$ 93	$ 192
PID rate in infected partners			
	30	$ 0	$ 140
	10	$ 103	$ 243
Probability of infecting a female			
	40	$ 14	$ 155
	20	$ 88	$ 229
Compliance			
	95	$ (−)11	$ 86
Loss-to-Followup DFA			
	10	$ 51	$ 216
Best Case*		$(−)195	$(−)137
Worst Case**		$ 619	$ 866

* = Prevalance = 35%; LE Sensitivity 85%/Specificity 95%; PID Rate 30%; Infected Partner Rate 40%; Compliance 95%

** = Prevalence = 5%; LE Sensitivity 65%/Specificity 75%; PID Rate 10%; Infected Partner Rate 20%; Compliance 65%.

Health Outcomes at Baseline Assumptions

Screening with culture results in the highest percent cure rate (56 percent) per 1,000 adolescent males (Table 3). Screening with the DFA results in the second highest percent cure rate (51 percent), while the LE results in the lowest percent cure rate of the three tests (49 percent). The strategy of not screening results in a 44 to 51 percent lower cure rate than any of the three screening options. Fewer patients will be treated unnecessarily with culture and direct-smear (DFA) screening compared with the LE test (see Table 3).

Sensitivity Analysis and Break-Even Prevalence

Sensitivity analyses were performed on seven variables (Table 4). Because the LE test and the DFA appear to be the more practical options for screening, culture was not included in the sensitivity analysis.

At base-line assumptions, the disease prevalence which leads the LE test to break even when compared with the cost of not screening is 21 percent. Above this prevalence, the LE test always costs less than not screening. The break even prevalence for the DFA is 41 percent. Using base-line assumptions, the LE test will be less costly than the DFA at any prevalence. As the prevalence is lowered to 5 percent, the cost/cure for the LE rises to $415 and for the DFA to $795.

Increasing the sensitivity/specificity of the LE test will, as expected, make the LE test even more cost-effective (lower cost-per-cure, and/or lower total costs and higher or equivalent cure rate) compared with the DFA. Even at the lowest sensitivity/specificity for the LE, the cost-per-cure is still only 48 percent of the cost of the DFA.

Increasing the PID rate in infected partners to 30 percent results in the LE breaking even compared to not screening. The DFA would still cost $140 per cure. Decreasing the PID rate to 10 percent results in the LE costing $103 per cure with the DFA costing $243 per cure. Increasing the probability of infecting a female to 40 percent results in the LE test costing $14 per cure and the DFA costing $155 per cure. Decreasing the probability of infecting a female partner to 20 percent

results in the LE costing $88 per cure and the DFA costing $229 per cure.

If the loss-to-follow-up for culture and the DFA is raised to 10 percent, the percent cure rate for culture is still the highest (53 percent), with the LE next (49 percent) and the DFA the least effective (47 percent cure). The cost per cure for the DFA rises to $216 compared to $51 for the LE. If the loss-to-follow-up rate is 7 percent, the percent cure rate for the LE is equal to the DFA.

If the compliance rate were increased to 95 percent with all other variables at base-line, the LE would save $11 per cure (cost-per-cure = $51 at 65 percent compliance) and the cost-per-cure for the DFA would drop to $86 (from $192 at 65 percent compliance).

If the "best case" scenario for use of screening tests is applied (high sensitivity/specificity of tests, high prevalence of disease, high PID rate in infected partners, high probability of infecting a partner) the LE saves $195 per cure, the DFA saves $137 per cure, and culture saves $6 per cure. If the "worst case" scenario for use of screening tests is applied (low sensitivity/specificity of tests, low prevalence of disease, low PID rate in infected partners, low probability of infecting a partner) then the LE costs $619 per cure and the DFA costs $866 per cure.

Discussion

Our analysis indicates that the leukocyte esterase (LE) urine dipstick test, when used to screen for *Chlamydia trachomatis* in a population of sexually active adolescent males results in greater cost savings than screening with either DFA or culture. This finding is true throughout the range of sensitivity analyses for seven separate variables. Moreover, screening with the LE urine dipstick test in this population can result in an equivalent number of cures if the loss-to-follow-up rate for the DFA test is greater than 6 percent. These findings clearly underscore the effectiveness and substantial benefits of the LE test and support its use in this at risk population that is currently not routinely screened.

In populations where contacting patients with test results and treatment recommendations is not problematic and the prevalence of *C. trachomatis* is very high, the cost-per-cure for the DFA is substantially reduced. The higher costs associated with use of the DFA over the LE must be balanced against the high incidence of unnecessary treatment resulting from the lower specificity of the LE test. Since the LE false positive rate was established using cultures which probably have <90 percent sensitivity,[17] the LE may be detecting infections which would be missed by DFA or culture. We also recognize that some false positive LE dipstick tests in adolescent males are due to the presence of *N. gonorrhoeae* so that treatment unnecessary for the eradication of chlamydia would ultimately be of benefit. We did not attempt to place a value (or utility) on the outcome of unnecessary treatment. While the morbidity associated with tetracycline therapy in males is certainly minimal, the psychosocial consequences of inappropriate diagnosis and unnecessary treatment could conceivably impose a heavy burden on these young males and their sex partners.

Recognizing that the specificity of the LE test is much lower than that estimated for the DFA, and that precise identification of an infecting organism has potential therapeutic and health education value, have led practitioners to advocate the use of the LE as a screening test leading to further testing with culture or DFA.[10,11] Although use of this strategy would decrease the number of unnecessary treatments, the number of cured chlamydial infections would be lowered due to the combined false negative rates of both tests. Overall costs and cost-per-cure would be increased when compared with use of the LE alone. In populations where the prevalence of chlamydial infection is high and few patients are lost to follow-up, the cost-per-cure for the DFA may be acceptable enough for use as a screening test. In these populations, the higher expense for the DFA compared with the LE may be offset by the decreased number of unnecessary treated patients. Use of the LE as a predictor for further testing may be most beneficial in populations with a low prevalence of chlamydial infection and a high rate of follow-up, since the low specificity of the LE has the greatest relative impact in terms of unnecessary treatments at low disease prevalences.

Compliance with treatment is an important variable that significantly impacts the total costs and cost-per-cure for each screening strategy. If compliance with treatment could be increased to 95 percent, screening with the LE would save money over no screening. Similarly, increasing the compliance with treatment significantly reduces the cost-per-cure for the DFA and, if the follow-up rate was also high, some practitioners may find the cost-per-cure for the DFA to be in an acceptable range even at moderately low prevalences.

Our analysis focused on screening only for *C. trachomatis* with the LE test when the studies we cite[10,11] which determined the effectiveness of the LE test actually addressed both *C. trachomatis* and *N. gonorrhoeae* in asymptomatic adolescent males. The sensitivities and 100 percent specificities found in these studies were based on confirmed infection with either *C. trachomatis* or *N. gonorrhoeae*, or both. Since the sensitivity was 100 percent in one study, and the specificity was 100 percent in the other, our conservative sensitivity and specificity assumptions of 75 percent and 85 percent for the LE should absorb the effect of *N. gonorrhoeae*.

As with the LE, the sensitivity and specificity of the DFA in this population of all sexually active adolescent males are unknown. The DFA test was evaluated in men with symptoms or signs of urethritis and has not been evaluated in low-risk, asymptomatic men.[17] The sensitivity and specificity may be lower in adolescent male asymptomatic carriers. The LE test is aimed at detecting the pool of asymptomatic adolescent males who may be the main transmitters of infection to adolescent females. Sensitivity and specificity of the DFA test are also highly dependent on the skill and expertise of the microscopist interpreting the slide.[17] In laboratories with inexperienced technicians, the number of false-positive and false-negative results will bring the sensitivity and specificity toward the lower end of the spectrum.

We did not include in our analysis the strategy of empiric treatment of all sexually active adolescent males attending a clinic or physician's office. One study of patients at a sexually transmitted disease (STD) clinic[27] found this strategy to be the most cost-effective when compared with the strategies of culturing all patients and only treating those with positive results; empirically treating patients at high risk and culturing those at low risk; and empirically treating patients at high risk and neither culturing nor treating those at low risk. We initially evaluated this strategy and found it resulted in cost-per-cure of $(-)14, with 95 cures, 55 no cures, and 850 unnecessary treatments per 1,000 adolescent males. However, we elected not to present the empiric treatment data in

our comparative decision analyses because we agree with other investigators that this is not a feasible health policy option.[27] Not only would there be no basis for determining frequency of treatment, except perhaps level of sexual activity, but deciding about treatment of sex partners would also prove difficult. Furthermore, indiscriminate use of tetracycline could adversely affect antimicrobial sensitivity patterns, leading to resistant *N. gonorrhoeae* and possibly *C. trachomatis* as well.

The major costs associated with adolescent male chlamydial infection result from the costs associated with infecting a female partner. Although we have data to support the incidence of sequelae from chlamydial infection in females, and the associated costs resulting from the medical management of these sequelae, there are no firm data on the transmission rate from males to their female sexual partners. Recognizing that the transmission might have been from female partner to the adolescent male, that some adolescent females might already be infected with the same serovar of *C. trachomatis* from another partner and cannot be reinfected, and that the transmission rate is probably lower than 100 percent, we conservatively assumed that an infected male has a 0.30 change of infecting a female. Studies have shown that 30 percent of reported female partners of males with urethritis will be infected with *C. trachomatis*.[19] Since many adolescents have multiple sexual partners and may not report all of these partners, our sensitivity analysis which ranges the probability of infecting a female from 0.20 to 0.40 should cover any inaccuracy surrounding this variable.

These considerations notwithstanding, our analysis provides reasonable estimates of the risks, benefits, and costs of screening for *C. trachomatis* with the LE urine dipstick test compared with culture, DFA and not screening. Where imprecision exists, we believe it is in the direction of underestimating benefits and cost savings. For example, we did not include indirect costs, which are about equal to direct costs[2,26]; adding them would further increase the cost saving and enhance the cost-effectiveness of the LE test. Neither did we include the noneconomic benefits. These include the prevention of adverse psychosocial effects in males at risk for epididymitis and in their female partners who might later suffer from PID, ectopic pregnancy (with its resultant fetal loss and risk of maternal mortality), or infertility.

One other benefit not included in our analysis is the treatment of coexisting *N. gonorrhoeae* in adolescents diagnosed with *C. trachomatis* by the LE test. While data are not available on this coinfection rate, the rate of coexisting *C. trachomatis* in patients diagnosed with *N. gonorrhoeae* ranges from 15 to 30 percent in males and 25 to 50 percent in females.[8,13,28] Assuming the rate of coexisting *N. gonorrhoeae* in patients diagnosed with *C. trachomatis* is similar, then a significant number of gonococcal infections will be treated along with the chlamydial infection, because a seven-day course of doxycycline hyclate (100 mg PO bid) will eradicate at least 85 percent of infections due to *N. gonorrhoeae* since the highest reported rate of tetracycline resistent *N. gonorrhoeae* is 15 percent.[15,20] Importantly, treatment of these asymptomatic gonococcal infections would prevent transmission to susceptible females and subsequent adverse reproductive consequences. This would lead to even greater cost savings with the LE test compared to other screening methods and to not screening than what was calculated in our study.

The added benefits of the LE in detecting *N. gonorrhoeae* leads to combined *C. trachomatis*/*N. gonorrhoeae* treatment recommendations.[29,30] Not only does there appear to be an increasing treatment failure rate among patients with *N. gonorrhoeae* taking tetracycline exclusively,[31] but also the expected compliance with a seven-day treatment course is only 65 percent. Compliance with *N. gonorrhoeae* treatment would be raised to 100 percent if LE dipstick-positive adolescent males were given 250 mg ceftriaxone intramuscularly once or a one time oral dose of 3.5 g ampicillin/1 g probenicid before leaving the physician's office, and then given their seven-day course of doxycycline hyclate 100 mg PO bid to eradicate 95 percent of *C. trachomatis* in those who are compliant with treatment. Tracking of penicillin-resistant *N. gonorrhoeae* could be accomplished by culturing all males with a discharge, dysuria, history of exposure to a sexually transmitted infection, and/or a positive LE urine dipstick test for *N. gonorrhoeae*.

Although there are several studies that compare the costs and benefits of screening programs for *C. trachomatis* in females,[27,32,33] this is the first study that directly compares the costs and benefits of screening in adolescent males. This population represents a key link in the chain of transmission of chlamydial disease, yet routine screening of this group is not widely practiced. Since the cost-per-cure is unaffected by the size of the cohort, our analysis provides a model that practitioners can apply once they outline the characteristics of their cohort. To prevent further transmission and minimize the risk that an infected individual will have adverse consequences, clinicians should assess the prevalence and loss-to-follow-up rate in their populations, and consider using the LE urine dipstick test or the DFA, alone or in combination, for screening sexually active adolescent males to detect *Chlamydia trachomatis* infection.

APPENDIX

Cost-effectiveness Calculations

The cost of infecting a single female partner and the costs of potential neonatal transmission and its probability are calculated as shown in Appendix Table A. Appendix Table B shows the cost per *C. trachomatis* infected male assuming that 30 percent (range 20 to 40 percent) of female partners exposed by the male would become infected and have the costs and risks in Table A. We did not include potential male-to-male transmission. We assumed that 20 percent of female partners would present for treatment.[19] The cost of treating infected female partners included two outpatient visits, one DFA test, a seven-day course of doxycycline and treatment of the vaginitis expected in 10 percent of treated partners from the antibiotic therapy. Given the 20 percent false negative rate for the DFA,[17,18] we assumed that 16 percent of infected female partners would be treated with complete eradication of *C. trachomatis* and prevention of any sequelae. For the 84 percent of untreated female partners, we expected a 20 percent PID rate (range 10 to 30 percent) with the costs of treating the PID and it's sequelae listed in Table A. We predicted a 5 percent birth rate and estimated a 20 percent chlamydial conjunctivitis rate and a 10 percent chlamydial pneumonia rate in neonates born to *C. trachomatis* infected mothers. The cost of complications in males infected with chlamydia is combined with the cost of infecting female partners and the costs of potential neonatal transmission to result in an average cost per *C. trachomatis* infected male of $144.36 (Table B). The number of infected males was calculated by multiplying probability estimates down each pathway of the model and considering all those ending with "no cure" as infected and all those ending with efficacious treatment or spontaneous cures as "cured." Total costs were calculated by multiplying all "no cure" totals with the cost of infected males ($144.36) and adding this product to the following: 1) the cost of each test times the cohort; 2) the cost of follow-up and loss-to-follow-up times their probabilities of occurrence; plus 3) the cost of treatment and its probability of occurrence. This total cost calculation was divided by the number "cured" to obtain the cost-per-cure. The number unnecessarily treated equals the number of false positive tests. We used a microcomputer spreadsheet program to model the number of cures and costs.

APPENDIX TABLE A—Cost of Infecting Female Partners with *C. trachomatis* and Transmission to Neonates

	Cost	Probability	Reference
Infected female partners			
Rx fluorescent antibody		.20	
Direct-smear	$ 8	1.00	
Two outpatient visits	70	1.00	25
Vaginitis (complication Rx)	30	.10	26,27,29
7-Day course doxycycline	10	.80	
Infected female partners not Rx		.84	
PID Rate		.20	3,26,29
PID-outpatient treatment	180	1.00	25,26,29
PID-inpatient treatment	4,259	.21	25,26,29
Surgery	1,500	.12	25,26,29
Ectopic pregnancy	5,759	.04	25,26,29
Tubal infertility	2,500	.21	25,26,29
Chronic pelvic pain	200	.15	25,26,29
Birth Rate		.05	
Neonatal pneumonia	1,375	.10	7,8,25,29
Neonatal conjunctivitis	55	.20	7,8,25,29
Average cost/case	365.12		

APPENDIX TABLE B—Cost per *C. trachomatis* Infected Male

	Cost	Probability	Reference
Infected female partner & neonatal transmission	$ 365.12	0.3	Table A
Return visit symptoms	45.00	0.56	27
Epididymitis outpatient treatment	50.00	0.036	2,8
Epididymitis inpatient treatment	1,836.00	0.004	2,8
Average Cost/Case	144.36		

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Information on Tar and Nicotine Yields on Cigarette Packages

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Abstract: We examined information on tar and nicotine yields on the packages of 160 cigarette brands, 58 percent of the 275 brands for which tar and nicotine yields were listed in a recent Federal Trade Commission report. The tar yield was indicated on 14 percent, the nicotine yield was indicated on 11 percent. As tar yield increased among brands, the yield was progressively less likely to be shown on the package and was not disclosed on the package of any cigarette yielding 11 mg or more of tar. (*Am J Public Health* 1990; 80:551—553.)

Introduction

Since 1971, all major cigarette manufacturers have voluntarily disclosed the tar and nicotine yields of cigarette brands in advertisements.[1] The cigarette industry agreed to "voluntary" disclosure after the US Federal Trade Commission (FTC) had proposed a regulation that would have required such disclosure.[2] This agreement does not apply to cigarette packages.

The tar and nicotine yields listed in cigarette ads have been obtained by machine smoking of cigarettes under standardized laboratory conditions using an FTC method developed in the 1960s.[3] Through early 1987, the FTC conducted tar and nicotine measurements in its in-house laboratory; currently, the Tobacco Institute Testing Laboratory performs tar and nicotine measurements using the FTC's standard method. The Institute provides these test results to the FTC which, in turn, makes the information available in a public report.[4]

In 1981, the Secretary of Health and Human Services recommended that:

> " . . . manufacturers should list yields of 'tar', nicotine and other hazardous components on their packages and in their advertising with appropriate explanatory information on the health significance of these measurements. This would be a minimum first step in giving cigarette consumers full and adequate information about the products they are buying."[5]

Seven years after that recommendation was made, we conducted a study to examine the types of information related to tar and nicotine yields that are provided on cigarette packages.

Methods

In May of 1988, we visited eight retail stores that sell cigarettes in Montgomery County, Maryland to find as many different cigarette brands as possible. We found 160 (58 percent) of the 275 brands listed in a 1988 FTC report on tar, nicotine, and carbon monoxide yields of domestic cigarettes.[4] (The 275 brands include 272 in the report's primary listing, plus three varieties of Barclay cigarettes for which tar and nicotine yields were provided in the text.) These 160 brands accounted for approximately 78 percent of the domestic cigarette market in the second quarter of 1988.[6]

Many of the 160 brands were "extensions" of the same primary brand name but differed in cigarette length, the type of package (soft or hard pack), the number of cigarettes per package (20 or 25), the presence or absence of a filter, or the presence or absence of metholation. All of these variables (including type of package) may be associated with differences in tar and nicotine yield.[4,7]

For each brand, we examined the package for any information relating to tar and nicotine yields, including quantitative measurements (mg per cigarette) and qualitative descriptions (e.g., "light," "low-tar," "99% tar free"). Because tar and nicotine yields in cigarette brands are highly correlated (high-tar brands tend to have high nicotine yield, and low-tar brands tend to have low nicotine yield),[4] we analyzed package labeling patterns only with respect to tar yields.

For six brands, the tar yield indicated on the package was 1–2 mg higher or lower than the yield listed in the FTC report. In these cases, we used the yield indicated on the package in our analysis of package labeling patterns.

Results

Package Disclosure of Tar and Nicotine Yields

The tar yield was indicated on the package of 23 brands (14 percent). For 17 (74 percent) of the 23 brands, the nicotine yield was also indicated. In no case was the nicotine yield shown without the tar yield. The 23 brands fell within six brand "families" produced by five different manufacturers: seven Carlton varieties (American Brands); seven Merit varieties (Philip Morris Company); three Now varieties (RJR/Nabisco); three Barclay varieties (Brown & Williamson Corp.); two Triumph varieties (Loews Corp.); and one Benson & Hedges variety (Philip Morris). The 23 brands accounted for approximately 5 percent of the domestic cigarette market in the second quarter of 1988,[6] or about 6 percent of the combined market share of the 160 brands in our sample (78 percent).

Table 1 shows the proportion of brands whose packages disclosed tar yield within the six tar-yield categories used by the FTC in analyzing cigarette sales and advertising expenditures.[8] As tar yield increased among brands, the package was progressively less likely to show the yield. The tar yield was disclosed for all 11 brands yielding 3 mg or less of tar but not for any of the brands yielding 11 mg or more. When the six tar-yield categories shown in Table 1 are collapsed into four categories to allow use of the chi-square test for trend, the trend is statistically significant ($p < 0.001$, two-tailed).

Package Label Messages

Table 2 shows the frequency of use of words and phrases on cigarette packages that indicate or suggest a low-tar/

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TABLE 1—Proportion of Cigarette Brands That Disclose Tar Yield on Packages, among a Sample of 160 Brands (stratified by tar yield), United States, May 1988

Tar Yield* (mg/cigarette)	Total Brands	Brands with Tar Yield on Package	
		N	%
≤ 3	11	11	100
4–6	19	6	32
7–9	29	5	17
10–12	46	1	2
13–15	23	0	0
≥ 16	32	0	0
Total	160	23	14

*Source of information on tar yields of cigarette brands is the Federal Trade Commission.[4]

low-nicotine yield, stratified by tar yield. Five brands claimed to have the lowest yield. Two varieties of Carlton cigarettes, each having a tar yield of 1 mg, claimed that "Carlton is lowest." Three varieties of Now cigarettes, having tar yields of 1 mg, 2 mg, and 3 mg, claimed that "Now is lowest." Three varieties of Barclay cigarettes, ranging from 3 to 5 mg of tar, were said to be "99% tar free."

The phrases "ultra low tar" and "ultra lights" were used almost exclusively for brands yielding 7 mg or less of tar. The terms "low tar" and "lights" were generally used for brands yielding 15 mg or less of tar, conforming with the FTC's usual definition of low-tar cigarettes.[9] However, three brands with tar yields of 16–18 mg were called "lights" (Doral Lights king-size soft-pack, Richland Lights 100's soft-pack, and Richland Lights Menthol king-size soft-pack). One "ultra low tar" brand (Merit Menthol 100's soft pack) had a tar yield of 10 mg, which is higher than that of some brands labeled as merely "low tar." One brand (Lucky Strike Unfiltered Lights) had the phrase "only 9 mg tar" on the package.

TABLE 2—Frequency of Use of Words and Phrases on Cigarette Packages That Indicate or Suggest a Low-Tar/Low-Nicotine Yield, among a Sample of 160 Brands (stratified by tar yield), United States, May 1988

Tar Yield* (mg/cigarette)	Wording	Brands	
		N**	%
≤ 6	Ultra low tar	16	53
(N = 30)	Ultra lights	13	43
	Lowest	5	17
	99% tar free	3	10
	Ultra low	2	7
	Smooth	2	7
7–12	Light/Lights	49	65
(N = 75)	Low tar	29	39
	Lowered tar & nicotine	5	7
	Ultra low tar	3	4
	Mild/Milds	3	4
	Low nicotine	2	3
	Smooth	2	3
	Ultra lights	1	1
	Recessed filter	1	1
	Famous Micronite II filter	1	2
≥ 13	Light/Lights	8	15
(N = 55)	Smooth	6	11
	Low tar	5	9
	Mellow	2	4
	Activated charcoal filter	2	4
	Famous Micronite II filter	1	2
	Mild	1	2

*Source of information on tar yields of cigarette brands is the Federal Trade Commission.[4]

**Within a particular tar-yield category, the sum may exceed the total because some package labels used more than one descriptive term.

Several words or phrases that may suggest a low-tar yield are used on packages, including "smooth," "mild," "mellow," and references to special filters (Table 2). One filter was described in detail on the package: "Lark Lights filter has two outer 'tar' and nicotine filters, plus an inner chamber of tiny charcoal granules specially treated to smooth the taste in a way no other filter can." The term "full flavor," often used by cigarette companies as a euphemism for high tar, appeared on the package of three brands yielding 14–17 mg of tar (Cambridge and Doral varieties).

Discussion

Although several studies have shown that those who smoke lower tar cigarettes have a reduced risk of lung cancer,[5,10,11] many concerns have been expressed about low-yield cigarettes.[12,13] First, because the cigarettes are less harsh, they may facilitate experimentation with and initiation of smoking among children and adolescents. Second, smokers who switch to lower yield brands often change their smoking behavior to compensate for reduced nicotine delivery (e.g., by increasing daily cigarette consumption; by increasing the depth, duration, or frequency of puffs; or by smoking cigarettes to a shorter butt length).[14–17] These compensatory changes may, in some smokers, increase the total intake of tobacco smoke constituents. Third, the use of flavoring additives is thought to be more common in low-tar brands to compensate for reduced "taste." These additives and their products of combustion may pose additional health risks to smokers. Fourth, those who smoke lower yield cigarettes may consider them to be safe or less hazardous, and may therefore be less motivated to quit.[18] Fifth, there is little or no evidence that low-yield cigarettes reduce the risk of cancers other than lung, of cardiovascular disease, of chronic obstructive pulmonary disease, or of fetal damage when smoked during pregnancy.[5,13,19]

Consumers have received mixed messages about the advisability of switching to low-tar cigarettes. For example, widespread publicity in the media has followed the publication of articles reporting "benefits" of low-yield cigarettes.[20] These reports have been buttressed by cigarette advertising claims implying that low-tar cigarettes are less hazardous or safe.[18] On the other hand, articles reporting no "benefits" have also received substantial publicity.[19]

Consumers' ability to sort out and act on these disparate messages is complicated by the fact that package information on tar and nicotine is provided selectively and is ambiguous or inconsistent at times. Among the 160 brands included in our study, the tar yield was not disclosed for any brand yielding 11 mg or more of tar. (The sales-weighted average tar yield was 13 mg in 1987.[21]) Five different brands claimed to be "lowest," even though the tar yield of one (3 mg) was three times the tar yield of another (1 mg). One "ultra low tar" brand had a tar yield higher than that of several "low tar" brands. Three brands with tar yields ranging from 3 to 5 mg claimed to be "99% tar free," an ambiguous term which suggests the brands are virtually free of tar.

Requiring disclosure of tar and nicotine yields on all cigarette packages would eliminate the selective manner by which cigarette companies now provide this information. Uniform disclosure on packages would also make these figures more accessible than those currently provided on

cigarette advertisements. A standard coding system should be developed to define tar-yield categories using easily understood terms (e.g., low tar, medium tar, high tar). The appropriate designation could be required on each package and advertisement. Such a coding system is used in the United Kingdom for cigarette advertisements.[22] In the United States, no official government coding system exists for tar-yield terminology, and no industry standard apparently exists (Judith P. Wilkenfeld, Federal Trade Commission, oral communication, July 6, 1989).

Chapman, *et al*,[22] have suggested that retailers be required to display at the point of sale a table showing the yields of all brands (from lowest to highest yields). According to these authors, the state of South Australia has adopted a law implementing this recommendation.

Above all, mechanisms should be developed to warn consumers that any health benefits of switching to lower tar brands are minimal when compared with the benefits of quitting and are offset by smoking more cigarettes per day, puffing more frequently, etc. Warnings could be required, for instance, on package inserts, in advertisements, and at the point of sale. According to the 1986 Adult Use of Tobacco Survey, 21 percent of smokers (approximately 10 million Americans) believe that the kind of cigarettes they smoke are less hazardous than other cigarettes.[21] Many of these smokers might quit if they understood that any benefits of switching to low-yield cigarettes pale in comparison to the benefits of quitting.

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Comparative Evaluation of American Cancer Society and American Lung Association Smoking Cessation Clinics

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Abstract: We compared the effectiveness of American Cancer Society, *FreshStart*, American Lung Association, *Freedom from Smoking*, and laboratory clinic methods in subjects (N = 1041) from three communities. Three-month follow-up results favored the laboratory method over the two public service approaches on both a prevalence and a sustained abstinence measure. At one-year follow-up, treatment effects for smoking prevalence were no longer significant. However, sustained abstinence results at one-year remained highly significant and favored the more intensive laboratory and *Freedom from Smoking* clinics over the *FreshStart* method. *FreshStart* fared less well than the other interventions both in producing initial quit attempts and in sustaining abstinence among initial quitters. It should be noted, however, that *FreshStart* requires considerably less facilitator contact than do the other approaches. Unexpected outcome effects occurred for treatment location. Future clinic programs should include a specific target date for quitting and should place more emphasis upon recycling participants who fail to sustain abstinence. (*Am J Public Health* 1990; 80:554–559.)

Introduction

Public service programs have been developed to assist chronic smokers to quit. Characteristics common to most of these programs include not only the use of behavioral principles, but also a focus on multiple program components, increasing emphasis on maintenance and relapse prevention, and a heightened appreciation of variables such as cognitive processes and social support.[1] As smoking cessation programs have become relatively more sophisticated, there has also been an increased awareness of the need for more cost-effective delivery methods.[2] Among the most widely offered public service programs are smoking cessation clinics developed by the American Cancer Society and the American Lung Association. Despite their widespread use, no evaluations of the effectiveness of these clinics have appeared to date in the literature.

The present study undertook a comparative evaluation of the American Cancer Society (ACS) *FreshStart* and American Lung Association (ALA) *Freedom from Smoking* clinic methods. These methods were assessed in a context that also included a laboratory-derived clinic approach.[3,4] The laboratory method was included to allow not only assessment of the effectiveness of the two public service clinics themselves, but of their effectiveness relative to that of a method systematically derived from a paradigmatic program of research[5] which has been systematically disseminated in community settings as part of a program of technology transfer. Costs have been kept to a minimum through the use of donated facilities and volunteer group leaders who receive only nominal fees.[6]

Methods

Subjects

Subjects were 1,041 smokers (57.2 percent women) who responded to newspaper advertisements announcing the availability of free smoking cessation clinics. The clinics were described as being part of a federally funded project. Subjects were recruited from three Iowa locations: Des Moines (N = 743), Iowa City (N = 173), and Waterloo-Cedar Falls (N = 125). Three study locations were used to ensure adequate subject enrollment and to allow a test of the major study hypotheses. The investigator in charge of administering treatment in Waterloo-Cedar Falls left after the first year of the study and recruitment efforts in the area were discontinued at that point.

Facilitators

Facilitators were 10 women and five men specifically recruited and trained to administer intervention for purposes of the current study. Although specific information on facilitator characteristics is not available, group leaders in Des Moines were older, ranging in age between 35 and 50, and most had previous experience in conducting group programs. In contrast, facilitators in Waterloo and Iowa City consisted primarily of undergraduate and graduate students less than 30 years old. Efforts were made to see that each facilitator conducted all three interventions. This was not always possible, however. It should be emphasized that we have no evidence of a facilitator effect upon treatment outcome.

All facilitators were formally trained in all three methods: the American Cancer Society *FreshStart* program, the American Lung Association *Freedom from Smoking* clinics, and the Lando laboratory derived approach. *FreshStart* training consisted of a four-hour program provided by the American Cancer Society. The smoking intervention coordinator for the American Lung Association of Iowa provided training in the *Freedom from Smoking* method. Project staff provided training in the Lando method. Formal instruction in the American Lung Association and Lando clinic procedures required approximately six to eight hours. Facilitators were provided copies of detailed treatment manuals for all three clinic approaches.

Facilitators were informed that the study was intended to assess the relative effectiveness of different methods, that it was unclear whether any method would prove superior to the others, and that a major purpose of the study was to determine which methods might be more effective for different people. Emphasis was placed upon the point that a critical overriding study goal was to assist as many people as possible to quit across all conditions. Every effort was made to minimize bias on the part of group leaders. Facilitators were recruited from each of the communities and individuals were employed who had no prior identification with any of

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the programs. Strong emphasis was placed upon the need to conduct procedures exactly as described in the respective protocols.

Experimental Design

Subjects were randomly assigned to one of the three methods as a function of orientation sessions attended. Subjects were also informed that the study was comparing different methods and that while each of the methods was effective, the investigators believed that a particular method might be more effective for certain types of smokers. Treatment was offered free of charge.

Assigning all individuals attending a given orientation session to a single method minimized problems of self-selection to treatment. This was deemed important enough to outweigh any limitations arising from having different units of randomization and analysis. Only very general information that could apply to any of the three methods (e.g., small group format and a varying number of sessions) was released prior to the orientation. Friends and relatives who came together to orientation sessions were ensured the same treatment condition. A total of 70 orientation sessions were held and 97 treatment groups were eventually formed in the three intervention sites.

Preintervention Assessment

Subjects completed a brief demographic and smoking history questionnaire as well as an informed consent statement at the initial orientation session and provided the names and telephone numbers of three informants who could assist in locating them for purposes of follow-up data collection.

All subjects were informed that the programs would teach them skills to actively confront urges to smoke and enable them not only to stop smoking, but to remain nonsmokers. They were asked to verbalize a commitment to quitting that was intrinsic rather than a product of coercion from others. Emphasis was placed upon the premise that smoking is a learned habit and that it can be unlearned as well.

Attendance records allowed an indication of attrition *before* the fact (e.g., differential dropout subsequent to the orientation sessions as a function of anticipated treatment procedures). Details of the different interventions are contained in the Appendix.

Follow-up Data

A brief smoking status questionnaire was mailed to subjects at three-month, six-month, and 12-month follow-up intervals. Subjects who failed to respond to mailed questionnaires were contacted by telephone. Point prevalence was defined as complete abstinence from tobacco products on the day in question. Sustained abstinence was defined as the absence of relapse. Permitted slips were distinguished from relapses following guidelines recommended by the National Heart, Lung, and Blood Institute National Working Conference on Smoking Relapse[7] (a relapse was defined as seven consecutive days of smoking at least one puff per day). We specified an additional definition of relapse as seven consecutive slips within three months. Data were collected on whether subjects had made a quit attempt during the clinic they attended. A quit attempt was defined as at least 24 hours of continuous tobacco abstinence.[7]

Eighty subjects who claimed sustained abstinence through one-year follow-up (43.0 percent of the total claiming sustained abstinence) were asked to submit saliva samples about one month after their one-year follow-up had been completed. Saliva samples were collected in subjects' homes. All samples were analyzed for thiocyanate. Samples with borderline values (80–100 ng/ml) or higher (which appeared to contradict self-reported abstinence) were subsequently analyzed for cotinine (smoking was defined as a value of greater than 15 ng/ml). In the case of subjects who refused to submit to saliva testing, informants were telephoned and queried concerning the subjects' smoking status.

Results

Subject Characteristics

The average age of subjects was 42.4 years. Mean baseline cigarette consumption was 28.8 per day and the average number of years smoking was 23.6. Subjects had initiated a mean of 3.8 prior quit attempts with the longest abstinence period averaging 7.7 months. In an open-ended question asking about reasons for attempting to quit, 86 percent of subjects listed health concerns as one reason for quitting (Table 1).

Multivariate analysis of variance, performed to identify location differences, indicated that the Iowa City sample was on average 6.9 years younger and smoked 4.4 cigarettes less than their counterparts in the other two communities. Clinic size (i.e., the number of participants per clinic) was also considerably smaller in Iowa City.

Response Rates at Follow-Up

There was a 93.8 percent response rate at one-year follow-up. Response rates at three-month and six-month follow-ups were 98.2 percent and 93.0 percent, respectively. Forty-two people were not successfully contacted, 15 were lost to follow-up, five refused the follow-up interview, and three died. Those not reached, lost to follow-up, and refusers

TABLE 1—Mean Values on Baseline Variables for Total Sample and by Each Location Separately

Measure	Total	Des Moines	Iowa City	Waterloo
Sample Size	1041	743	173	125
Age	42.4 (±12.7)	43.7 (±13.1)	36.3 (±10.4)	43.1 (±10.4)
% Females	57.3	58.5	57.8	48.8
Cigarette consumption	28.8 (±12.2)	29.4 (±12.4)	25.3 (±10.9)	30.4 (±12.3)
Cigarette nicotine level	0.75 (±.30)	0.76 (±.31)	0.72 (±.26)	0.72 (±.30)
Years smoking	23.6 (±12.0)	24.7 (±12.5)	18.2 (±9.6)	25.2 (±10.4)
Previous quit attempts*	2	2	2	3
Longest quit attempt (months)*	1.1	1.1	1.3	1.2
% giving health reason to quit	86.1	86.7	81.7	88.4
Self-efficacy (0–100)	53.4 (±19.2)	52.4 (±19.1)	54.5 (±19.3)	57.4 (±19.2)
Clinic Size	10.7 (±4.0)	11.8 (±3.6)	7.2 (±2.2)	12.5 (±5.0)

*The median is reported for this variable because of the substantial skewness of responses.

were counted as smokers for purposes of data analysis. There were no differences between the treatment groups or locations in attrition rates at one-year.

Return to Treatment

For 62 of 70 informational meetings, data were available on the number attending the meeting as well as the number who returned to participate in treatment. A weighted least squares ANOVA indicated that the proportion returning was not a significant function of the treatment being offered.

Group size did not differ significantly as a function of the intervention method. Mean group sizes were 10.0, 11.7, and 10.5 for the ACS, ALA, and Lando methods, respectively.

Abstinence

The effects of method and location (and their interaction) on abstinence outcome were evaluated using logit analysis.[8] Lack of independence of outcome within treatment groups was expected due to individuals receiving treatment together in groups and having correlated outcomes, and related individuals (e.g., spouses) entering the same treatment group. A group effect was observed on one-year abstinence prevalence after method and location effects were taken into account ($\chi^2(92) = 123.32$, $p = .016$). Thus, an estimate of the design effect[9] due to clustering was computed (= 1.469), and the original chi-square test statistics were divided by the design effect to correct for this within group dependence.[10] Corrected chi-square statistics and confidence intervals are reported below.

Location effects did not interact with those due to treatment classification in any of the analyses reported. Also there were neither facilitator nor sex of subject effects on abstinence outcome.

Abstinence outcomes are illustrated in Figure 1; mean levels are presented in Table 2. While point prevalence differences are no longer statistically significant after the three-month follow-up interval, sustained abstinence differences are significant at all follow-up points. The ACS *FreshStart* program fared most poorly, and the Lando program achieved better outcome than the ALA *Freedom from Smoking* method.

Ninety-five percent confidence intervals comparing the ACS *FreshStart* treatment with the average of the two more intensive programs, and the Lando with the ALA *Freedom from Smoking* program on various follow-up measures are presented in Table 3. The *FreshStart* program fared less well than the other two methods on all measures.

Point prevalence abstinence rates at three months were highest in Waterloo (41.6 percent), followed by Des Moines (29.7 percent) and Iowa City (23.7 percent). These differences no longer held at six months and one year. However, at one-year follow-up, sustained abstinence rates were 27.2 percent, 17.0 percent, and 15.0 percent in Waterloo, Des Moines, and Iowa City, respectively ($\chi^2(2) = 6.05$, $p = .048$).

Quit Attempts

There were significant differences in the proportion of subjects in the different treatment groups making quit attempts (Table 4). Subjects in the ACS *FreshStart* program were again less likely to make a quit attempt than those in the other two programs.

Considering only those who made a quit attempt, there was no significant difference in one-year point prevalence

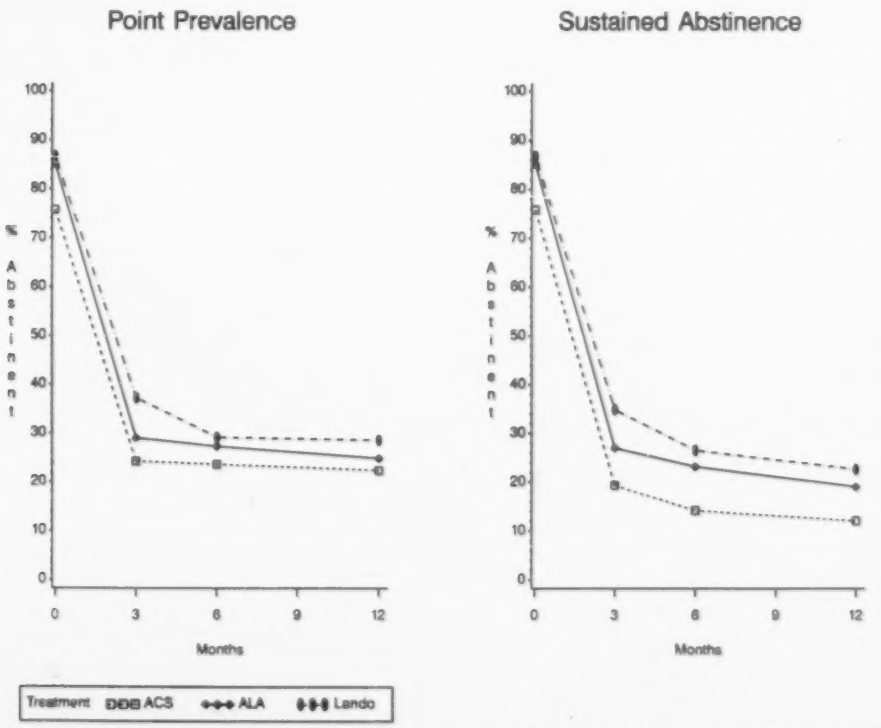

FIGURE 1—Abstinence Outcome at Three Follow-up Times Using Two Abstinence Criteria (Initial abstinence rates (at months = 0) represent initial quitting rates, i.e., those who remained abstinent for 24 hours after quitting.)

TABLE 2—Outcome Rates (expressed in percent) at Three Follow-up Intervals

Outcome Measure	ACS	ALA	Lando	Total	P*
3M Point Prevalence	24.17	28.93	37.18	30.16	.005
6M Point Prevalence	23.56	27.27	29.11	26.71	.38
1 Yr Point Prevalence	22.36	24.79	28.53	25.26	.28
3M Sustained Abstinence	19.34	27.00	34.87	27.19	<.001
6M Sustained Abstinence	14.20	23.14	26.51	21.42	.004
1 Yr Sustained Abstinence	12.08	19.01	22.19	17.87	.014
Total N	331	363	347	1041	

*P value is derived from χ^2 statistic with 2 degrees of freedom corrected for DEFF = 1.469.

TABLE 3—Percentage Differences and 95% Confidence Intervals for Percentage Differences in Outcome Measures among the Treatment Conditions

	ACS vs Others		Lando vs ALA	
Outcome Measure	Difference*	95% CI	Difference	95% CI
3M Point Prevalence	−8.76	(−16.02, −1.51)	10.37	(1.61, 19.14)
6M Point Prevalence	−4.45	(−11.41, 2.51)	2.73	(−5.51, 10.98)
1 Yr Point Prevalence	−4.20	(−11.00, 2.60)	4.33	(−3.76, 12.42)
3M Sustained Abstinence	−11.68	(−18.56, −4.80)	9.70	(1.07, 18.33)
6M Sustained Abstinence	−10.90	(−17.25, −4.56)	4.82	(−3.36, 13.00)
1 Yr Sustained Abstinence	−8.85	(−14.92, −2.79)	4.94	(−2.90, 12.78)

*Estimates of differences and confidence intervals are adjusted for location effects.

TABLE 4—Quit Attempts and One-Year Outcome among Those Making a Quit Attempt

Outcome Measure	ACS	ALA	Lando	Total	P*
Making quit attempt	75.83	85.12	86.74	82.90	.004
1 Yr Point Prevalence	27.09	28.48	31.56	29.15	.59
1 Yr Sustained Abstinence	15.94	22.33	25.58	21.60	.068

*P value is derived from χ^2 statistic with 2 degrees of freedom corrected for DEFF = 1.469.

outcome between the methods but persons in the ACS *FreshStart* program were less likely to sustain abstinence than were those in the other two treatment groups [difference = −8.02%; 95% confidence interval = (−15.37, −0.67)].

Clinic location did not affect the number making quit attempts but did influence one-year sustained abstinence outcome among those attempting cessation (data available on request to author).

Biochemical Validation

Results of biochemical validations performed at one-year tended to be consistent with self-reported abstinence. Of the 80 persons selected for biochemical validation, 73 provided saliva samples. No samples exceeded the cutoff for disconfirmation. Six of the remaining seven persons, while claiming abstinence, refused biochemical validation while one person had returned to smoking between the one-year follow-up and testing. One of those who refused was identified as a smoker by an informant and three additional refusers either did not list or we were unable to contact their informants. If all four of these subjects are counted as smokers (which appears to be an appropriate, albeit conservative, practice) the lie rate would be 5.1 percent (= 4/79). Adjusting the reported outcome data by this factor would not markedly affect absolute abstinence levels and would have little impact upon interpretation of results.

Discussion

The present study represented the first comparative evaluation of the American Cancer Society *FreshStart* and the American Lung Association *Freedom from Smoking* clinic methods. Results indicated relatively modest outcomes for both clinics. The disappointing outcome for the *FreshStart* method on the sustained abstinence measure (12.1 percent sustained abstinence at 12 months) appears noteworthy. This figure would be even lower (10.9 percent) if more rigorous continuous abstinence guidelines had been used (i.e., absolutely no use of tobacco in any form whatsoever).

The reduced number of initial quit attempts with the *FreshStart* method would appear to be largely a function of the absence of a set quit date. However, the present study design does not permit unequivocally eliminating other possible explanations including the lesser total clinic contact. Even so, it should be noted that intensity of clinic contact prior to quit attempts was fairly comparable for the *FreshStart* and *Freedom from Smoking* methods. The major difference in scheduling between the two public service approaches occurred during the maintenance phase rather than in preparation for quitting. Although the results for *FreshStart* are disappointing, it should be remembered that the *FreshStart* method also requires considerably less facilitator contact.

The fact that these programs were offered totally free to participants may have been partially responsible for overall modest abstinence rates. The absence of a fee may also have reduced participants' commitment.

Facilitator bias cannot be dismissed as a possible factor mediating differences between methods. However, every effort had been made to eliminate this bias. Facilitators had no a priori commitment to a given approach. They had been specifically instructed to work within the confines of each method to produce the best possible outcomes. Furthermore, the larger differences occurred between the two public service methods. Differences between *Freedom from Smoking* and the laboratory program (in which the investigators would have been expected to have a vested interest) were less pronounced and were no longer significant at the one-year follow-up.

Differential enrollment in treatment conditions was not a factor in mediating outcome. There were no differences in enrollment in the three clinic programs following the initial informational meeting. It is possible that a limitation inherent in the interpretation of these results may arise from the fact that the unit of randomization and the unit of analysis in this study are different. However, the large sample size provides a measure of confidence in the results.

The location effects were unanticipated. Although no obvious explanation is apparent for the superior outcome in Waterloo, clinics were relatively new to this community. Numerous cessation programs had been offered in both Des Moines and Iowa City. Perhaps smokers in Waterloo responded to the novelty of the clinic programs. Des Moines and Iowa City subjects would have had many previous opportunities to enroll in cessation clinics and these subjects may have been more resistant to quitting. It should be noted that at no point did location effects interact with intervention. The order of outcomes was comparable across all three study communities despite the main effects for location. The fact

that the study results were replicated in three separate communities with a relatively large total subject pool lends increased confidence in their generalizability. Ironically, the Iowa City sample which fared most poorly was younger and apparently less habituated (as indicated by lower daily cigarette consumption) than were the other subject samples.

Although cost-effectiveness analyses were not a formal part of the present study, cost considerations are obviously important.[11–12] Costs of programs can be sharply reduced by use of lay volunteers and donated facilities.[6] Training requirements are minimal. Each of the three methods can be learned in a few hours of formal instruction. Costs that are incurred can be largely defrayed by relatively modest fees to participants (the maximum fee per participant in public service applications of the laboratory-derived clinic method is $75.00). Costs should be lower for the ACS method given the requirement of only four hours of participant contact. These lower costs must be weighed against the reduced effectiveness of this method.

It has proven possible to achieve low-cost technology transfer of the laboratory method. Collaboration between laboratory researchers and a voluntary agency (the American Lung Association of Iowa) has proven critical in effecting this transfer. Especially encouraging is the apparent lack of decay in outcome in the transfer between the laboratory and field settings. There have been virtually no net costs to field sites (often community hospitals). Participant fees have defrayed costs of facilitators and promotion (often promotion has occurred through public service messages).

The American Lung Association of Iowa in the 1988–89 fiscal year reported total expenses of $32,438.70 in overseeing the statewide implementation of 111 laboratory-derived public service clinics reaching approximately 1,000 smokers. Expenses included research and evaluation, travel, facilitator training and updates, printing, postage, computer and software, telephone, personnel, advertising and audiovisuals. These expenses were largely offset by income ($18,235.00), fees charged for facilitator training and updates ($2,982.08), and a grant from an insurance company ($3,187.34). Net expenses were $8,034.28 or approximately $8.03 for each smoker who went through the program. Cost per abstinent smoker at 12 months was $26.77. Considerable public health impact might be achieved through widespread application of this type of collaborative model involving voluntary agency dissemination of validated cessation clinic programs.

The disappointing overall success rates suggest the need for further intervention among non-abstinent subjects. Prevalence outcomes at one year considerably exceeded sustained abstinence figures. Of those subjects who reported abstinence at one year, 41 percent had relapsed during the follow-up period. Schachter[13] has noted that cumulative success rates in self-initiated quitting substantially exceed commonly reported clinical findings for single quit attempts.

Perhaps the spontaneous initiation of renewed quit attempts observed in the present study can be facilitated through systematic intervention. Little research has been devoted to recycling individuals who fail to sustain abstinence. Too much emphasis may have been devoted to one-shot (albeit relatively intensive) treatment approaches. The need for recycling is underscored by the fact that outcomes in our more recent studies have been generally less successful than was true for studies that we undertook five to 10 years ago. This is true despite the fact that smoking history and demographic characteristics of our clinic enrollees have remained remarkably similar over time. More emphasis should be placed on viewing cessation as a process rather than as a discrete event.[14] Given that most quit attempts end in failure, the importance of eliciting multiple attempts over extended periods of time becomes obvious.

APPENDIX

American Lung Association Clinics

The basic American Lung Association clinic format consists of an orientation session and seven additional 90 minute to two-hour sessions over a seven-week period. The first session provided an in-depth discussion of the general health effects of smoking. Emphasis was placed upon the fact that subjects can quit and that help and encouragement to do so would be available in the program. The second session introduced coping strategies for confronting urges to smoke. By the third session, subjects were expected to make a personal commitment to quitting and to state this commitment publicly at the session. Quit Day occurred at this third session. At a fourth session held two days later benefits of quitting were reiterated and possible withdrawal symptoms were discussed.

The remaining sessions were focused upon maintenance and included a consideration of healthier, more enjoyable nonsmoking life-styles. Discussion included relaxation techniques, exercise or physical fitness programs, avoiding weight gain, and coping with tension. Session 7 was a celebration in which subjects elected options such as a wine and cheese party or dinner at a restaurant. This event was intended to emphasize enjoyment of their new life-style (and also to provide practice in confronting what for many might be a difficult situation). At this time awards were given to everyone who completed the program. Although the American Lung Association clinic has approximately one-half the sessions of the Lando clinic, total clinic contact hours for the two methods are approximately equal. Twelve of 31 American Lung Association clinics followed a modified format adopted by the American Lung Association as an alternative for worksite settings. This modified format entailed 10 one-hour sessions. No significant differences in outcome were found between the traditional and modified formats.

American Cancer Society Clinics

Treatment consisted of an orientation session plus four, one-hour group sessions over a two-week period. Emphasis was placed upon individualization of program content by the facilitator and eliciting the active involvement of group members. The focus of treatment was again upon coping strategies and a positive smoke-free life-style. However, instructions to clinic leaders placed relatively more weight upon individual situations as opposed to group process. Subjects were informed that smokers who take two weeks to quit smoking are as likely to be successful as are smokers who take two months to quit. Quitting is again described as a two-part process: 1) stopping, and 2) staying stopped. Unlike the other clinic methods there is no set target date for abstinence, although participants are expected to quit during the latter half of the program.

Laboratory Clinics

Treatment consisted of 16 sessions (approximately 45 minutes to one hour in length) over a nine-week period. The first three weeks were devoted to preparation for quitting and the final six weeks to maintenance. A specific preparation technique involved a nicotine fading procedure adapted from that originally described by Foxx and Brown.[15] Subjects switched brands on a 30–60–90 percent weekly reduction schedule. They were free to choose any cigarette that provided the required nicotine dosage. Subjects also underwent a smokeholding technique similar to that described in 1979 by Kopel, Suckerman, and Baksht* during two clinic sessions held the week prior to the quit date. Smokeholding consisted of two sets of 10 trials separated by a five-minute break.

Subjects attended eight maintenance sessions over a six-week period following the quit date. Considerable emphasis was placed upon relatively unstructured group discussion. In addition, subjects signed contracts calling for specific rewards for abstinence. Group discussion tended to emphasize problem-solving. Subjects were encouraged both to discuss their own problems and to suggest possible strategies for other group members. The laboratory clinics are described in greater detail elsewhere.[6,16]

*Kopel S, Suckerman K, Baksht A: Smoke holding: an evaluation of physiological effects and treatment efficacy of a new nonhazardous aversive smoking procedure. Paper presented at a meeting of the Association for Advancement of Behavior Therapy, November 1979

ACKNOWLEDGMENTS

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National Nursing Shortage Commission Announced

A 15-member Commission on the National Nursing Shortage has been established, as announced in the February 23 *Federal Register*. The Commission will advise federal officials—including the DHHS Secretary, the Assistant Secretary for Health, and the HRSA Administrator—on projects implementing the recommendations of the Secretary's Commission on Nursing. The projects should attempt to make optimal use of available resources from federal, state, and local government and private organizations. The recommended projects will focus on five areas:

- Recruitment and the educational pathway;
- Retention and career development;
- Restructuring nursing services and effective utilization of nursing personnel;
- Data collection and analysis requirements; and
- Information systems and related technology in nursing.

The Commission will consist of four ex-officio members and 11 members appointed by the Secretary. The ex-officio members will be:

- A Health Care Financing Administration representative;
- The Chief Nurse Officer of the Public Health Service;
- The Director of the Division of Nursing, HRSA; and
- The Director of the National Center for Nursing Research, National Institutes of Health.

The appointed members will be selected as follows:

- Four from the nursing community;
- Four from health care providers and other nurse employers;
- One from third party payers;
- One representing economics and data policy fields; and
- One from the general public.

Caroline Bagley Burnett, MSN, ScD, a registered nurse and health researcher, was selected Executive Director of the new Commission on the National Nursing Shortage. Formerly she was a research consultant and clinical researcher at the National Cancer Institute and, earlier, assistant professor and oncology program director at the Catholic University of America's School of Nursing.

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Benzene, Benzo(a)Pyrene, and Lead in Smoke from Tobacco Products other than Cigarettes

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Abstract: Benzene, benzo(a)pyrene (BaP), and lead in mainstream smoke from cigars, roll-your-own (RYO) cigarette and pipe tobaccos were sampled to evaluate their potential health significance. Results with reference cigarettes were consistent with published values, providing support for the methodology employed. The emissions of benzene and BaP, expressed as mass emitted per gram of tobacco consumed, were similar for all products evaluated; for benzene, the mean values for cigars, RYO cigarette and pipe tobaccos were 156 ± 52, 68 ± 11, and 242 ± 126 μg/g, respectively. Mean values for BaP were 42 ± 7 and 48 ± 4 ng/g for cigars and RYO cigarette tobacco, respectively. Lead values were below the limit of reliable quantitation in all cases. The mean benzene concentrations in a puff ranged from 1 to 2 × 10^5 μg/m^3 for cigars, RYO cigarette and pipe tobaccos. For BaP, the puff concentration averaged about 60 μg/m^3 for cigars and RYO cigarette tobacco. The results suggest that smoking cigars, pipes or RYO cigarettes leads to potential exposures which exceed the No Significant Risk levels of benzene and BaP set pursuant to California's Proposition 65. These tobacco products are now required to bear a health hazard warning when sold in California. We recommend that this be adopted as national policy. (*Am J Public Health* 1990; 80:560–564.)

Introduction

Cigarettes sold in the US are required by federal law to bear warnings of their health hazard. Other tobacco products, viz., cigars, pipe, and roll-your-own (RYO) cigarette tobaccos are not covered by federal law. A previous publication discussed California's Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65), including the requirement for warnings on commercial products containing or forming chemicals "known to the State" to cause cancer or other reproductive toxic effects.[1] Accordingly, the levels of three tobacco smoke constituents subject to Proposition 65 regulation, benzo(a)pyrene, (BaP), benzene, and lead, were determined. For benzo(a)pyrene, there is limited evidence of carcinogenicity in humans, but sufficient evidence of carcinogenicity in experimental animals.[2] Benzene has been shown to increase the incidence of leukemia in exposed workers, and to produce cancer in animals at various sites by several routes of exposure.[3] The reproductive toxicity of lead has been well documented in both human and animal investigations.[4]

Tobacco smoke includes both particle-phase and gaseous combustion products.[5,6] The particle-phase material represent about 8 percent (by weight) of the total. The gas-phase constituents include air (58 percent by weight), excess N_2 (15 percent by weight) and vapor constituents (19 percent by weight), the latter being hydrocarbons, oxygenated organic compounds and other gases (e.g., carbon monoxide). Polyaromatic hydrocarbons (PAHs) are found in both phases, the low molecular weight materials (e.g., naphthalene) being in gas phase, together with single ring aromatic hydrocarbons (e.g., benzene), and the five-ring PAH, benzo(a)pyrene (BaP) being almost exclusively in particle phase under usual conditions. Lead in cigarette tobacco smoke exists primarily in the particle phase. Commercial cigarettes from five countries showed a range for lead in mainstream tobacco smoke of 0.4 to 1.0 μg/cigarette in the particle phase, compared with 0.1 to 0.2 μg/cigarette in the gas phase.[7]

Mainstream cigarette tobacco smoke samples are routinely generated with laboratory "smoking machines" using Federal Trade Commission (FTC) standard smoking conditions: a two-second puff of 35 ± 2 ml volume, once/min, and a butt length of 23 mm.[8] FTC guidelines are lacking for RYO cigarette and pipe tobaccos, as well as for cigars. The Tobacco Research Council of London (TRC) specified for cigars the same parameters as used with cigarettes, with a butt length of 25 mm.[9] A cigar smoking protocol using a 1.5 sec puff, 20 ml puff volume, 40 sec puff interval and 33 mm butt length has also been recommended.[10] No standard protocol is available for testing pipe tobacco or RYO cigarettes, to our knowledge.

The present study measured and compared cigars, pipe tobacco and RYO cigarettes, for the levels of benzene, BaP, and lead in their mainstream smoke; and compared the mainstream smoke results to the daily exposure levels of benzene, BaP and lead judged in California Proposition 65 to pose no significant risk. Because of time limitations, pipe tobacco smoke was assessed for benzene levels only.

Methods

Tobacco Products

The tobacco products evaluated are described in the Appendix. One cigar was smoked for each lead or BaP plus benzene sample. The RYO cigarettes were prepared with a rolling machine intended for private use, with a rice paper wrapper ("Abadie" brand), length 70 mm, to which was added a pre-weighed amount of tobacco. Three such cigarettes were smoked per sample. Pipe tobacco was evaluated when smoked in a pipe, as well as in RYO cigarettes, prepared as described above, weighing 1.50 ± 0.02 g, smoking one cigarette per sample.

For pipe smoking, 1 to 2 g of tobacco were smoked in a pre-conditioned wooden pipe (bowl dimensions 20 mm I.D., depth at center 39 mm, stem length 60 mm from center of bowl) requiring 3 to 4 g of tobacco for filling as follows: the pipe was filled loosely with tobacco to the top of the bowl, the tobacco compressed in half, the bowl refilled and the tobacco

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compressed to about three-fourths of a full bowl. The tobacco consumed was approximated from the weight difference for the pipe plus tobacco before and after smoking.

Sample Collection and Smoking Parameters

The smoking parameters adopted here for cigars, pipe and RYO cigarette tobaccos retained the puff duration and volume of the FTC guidelines and varied the puff frequency as necessary to keep the tobacco burning and (for cigars) to provide a smoking time <1 hour. Pipe tobaccos were evaluated as RYO cigarettes as well as when smoked in a pipe, to assess whether smoking conditions played a major role in determining emissions of the pollutants measured.

A smoking system (Figure 1) drew air through the cigar, cigarette or pipe for brief periods to simulate a smoker's puff. The system was adjusted to a 35 ± 2 ml puff volume per 2.00 ± 0.05 sec puff using a bubble flowmeter attached upstream of the system. The RYO cigarettes prepared from cigarette tobacco followed FTC smoking parameters (i.e., two sec puff, 35 ml puff volume, once/min, butt length 23 mm). The RYO cigarettes prepared from pipe tobacco employed the same parameters except for puff frequency, which was increased to two/min. Cigar testing followed TRC-recommended parameters (two sec duration, puff volume 35 ml, butt length 25 mm),[9] with puff frequency altered to two/min. With pipe tobacco smoked in a pipe, a puff frequency of 12/min was used. Before smoking, tobacco products were equilibrated at room temperature for 24 to 48 hours in a chamber at about 60 percent relative humidity.

The sampling scheme employed a Gelman A/E glass fiber filter to collect BaP, or an 0.8 μm pore size (Millipore AA) cellulose ester membrane filter to collect particle-phase lead. An 8 × 110 mm glass tube filled with granular, activated charcoal (400 and 200 mg charcoal in the front and rear sections, respectively), downstream of the glass fiber filter, collected benzene with a measured front section collection efficiency >99.99%. The 47-mm diameter filters were contained in a Teflon filter holder. To minimize filter plugging with smoke constituents from cigars, filters were replaced when half the cigar had been consumed. By this strategy, the decrease in flow rate during filter loading was ≤20 percent. The two filters were extracted together to recover the BaP or lead. Single filter collection with ≤20 percent decrease in flow rate was achieved with the other tobacco products. The decrease in flow rate caused an estimated ≤10 percent positive error in calculating the total air volume sampled, and a corresponding negative error in calculated concentrations of benzene and BaP in a puff.

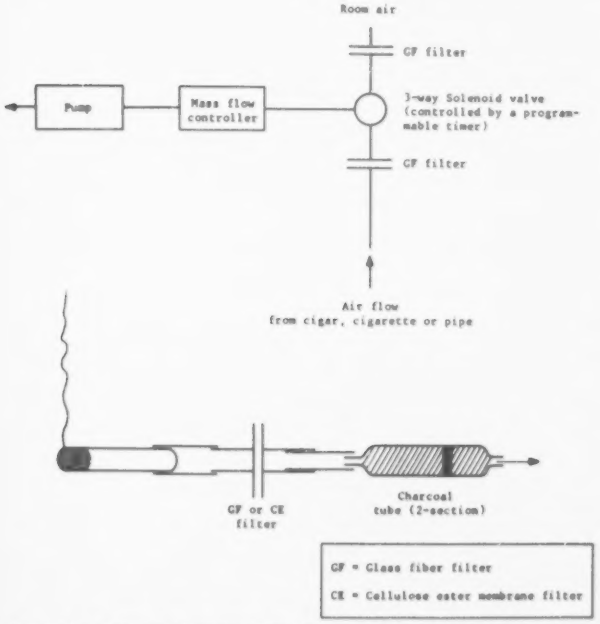

FIGURE 1—Schematic Drawing of the Collection of Tobacco Smoke Particulate and Vapor Phase Constituents

Chemical Analysis

BaP was extracted by one-hour ultrasonic agitation of the filter(s) in 70:20:10 v/v/v dichloromethane-methanol-benzene. Following removal of acidic and alkaline constituents by liquid-liquid extraction, and treatment with silica gel, samples were analyzed for BaP by reverse phase, high performance liquid chromatography, with fluorescence detection. Using NBS Standard Reference Material 1649 (urban dust), the recovery of BaP from the sample cleanup and analysis averaged 77 ± 4 percent. Benzene was extracted from the charcoal-filled collection tubes with CS_2 and directly analyzed by gas chromatography with photoionization detection.[11] Independent confirmation of the identity of the GC peak as benzene, as well as an interlaboratory comparison of benzene quantitation, was provided by gas chromatography-mass spectrometry (GC-MS) analysis on three CS_2 sample extracts. Benzene results from the two laboratories for the three extracts agreed, on average, within 10 percent. Lead was extracted from filter samples in concentrated HNO_3, and analyzed by flameless atomic absorption spectroscopy, using a heated graphite analyzer.[12] Blank trials with unlit tobacco showed no significant levels of BaP, benzene or lead.

Results are reported per gram of tobacco consumed (i.e., the total weight of tobacco consumed during as well as between puffs) as well as per cigar, or cigarette. Tobacco products were also compared based on average concentrations in the air stream sampled (i.e., puff concentrations). Comparison of results among the tobacco products is complicated by differences in puff frequencies. For example, at two puffs/min, a greater proportion of the tobacco will be consumed in generating mainstream smoke, compared to one puff/min. It is inappropriate to normalize results to a common puff frequency (e.g., by dividing results obtained at two puffs/min by two), since increased puff frequencies cause higher burning temperatures, enhance pyrolysis and decrease the amount of combustion or pyrolysis products retained on unburned tobacco in the butt.

Results

Quality Assurance

A Kentucky reference cigarette, type 2R1 (University of Kentucky), was evaluated for benzene and BaP production in the mainstream smoke. The procedures followed were the same as those for RYO cigarettes with cigarette tobacco; three cigarettes (average cigarette weight 1.18 g) were used to load a single charcoal tube. The benzene results for two such charcoal tube samples were 56 ± 2 and 67 ± 6 μg/cigarette (67 ± 2 and 81 ± 6 μg/g of tobacco burned), respectively. These results compare to 75 μg/cigarette reported for a similar Kentucky reference cigarette 1R1,[13] using a commercial smoking machine with the same smoking parameters. Smoke

constituent deliveries for the two reference cigarettes are expected to be equal within about 30 percent.* Our benzene results for reference cigarette 2R1 averaged 18 percent lower than the published results for reference cigarette 1R1. Mainstream smoke BaP results for two trials, corrected for 77 percent recovery, were both 32 ng/cigarette (39 ng/g of tobacco burned). This compares to the 28.4 ± 3.4 ng/cigarette previously reported for this cigarette, using the same (FTC) smoking parameters.[14]

To assess the accuracy and precision of the analysis for lead at the relatively low levels expected, four blank cellulose ester membrane filters were each spiked with 0.1 μg lead from an aqueous solution of lead nitrate, and then subjected to extraction and analysis. The recovery averaged 83 ± 19 percent (n = 4).

Benzene

Mean tobacco smoke results, by brand, are shown for cigars and RYO cigarette tobacco in Table 1. Expressed by amount per cigar or cigarette, the benzene values for cigars and cigarettes showed a wide range of values. Brand F cigars were the only filter-tipped tobacco product evaluated in the present study. Benzene results for these filter-tipped cigars were similar to those for the others. The range in benzene results is reduced when expressed per gram of tobacco burned.

Benzene levels from RYO cigarettes were similar to those for the Kentucky reference cigarettes, smoked following the same parameters, and, expressed per gram of tobacco burned, averaged about a factor of two below those for cigars. This difference reflects, at least in part, the factor of two difference in puff frequency. Table 1 also shows average concentrations calculated for benzene in a puff, as well as the average number of puffs required with each product. The concentrations ranged from 0.7×10^5 to 3.3×10^5 μg/m^3, about 10^4 times those in urban ambient air.[15]

Table 2 summarizes pipe tobacco smoke benzene results for single cigarettes rolled from pipe tobacco, and for the same tobacco smoked in a pipe, expressed per gram of tobacco burned as well as by average puff concentrations. Again, ranges are wide. On average, results for pipe smoking were 2.5 times those in the RYO cigarette configuration. The difference probably reflects the much higher puff frequency used in pipe smoking, 12 vs two puffs/min, and the correspondingly greater proportion of the tobacco consumed in generating mainstream smoke. The mean result in the RYO cigarette configuration, 139 ± 45 μg/g, compares to 156 ± 52 μg/g for cigars, also smoked with two puffs/min. Thus, when tested using the same smoking parameters, pipe tobacco and cigars were not significantly different regarding mainstream smoke benzene levels. The puff concentrations from pipe tobacco smoked in both configurations are similar to those found for cigars and RYO cigarettes from cigarette tobacco.

BaP

The coefficient of variation (C.V.) for BaP measurement, with each brand of cigar or RYO cigarette tobacco evaluated in two or more trials, varied from 3 to 24 percent (Table 1). The BaP levels ranged from 96 to 292 ng per cigar, and were about 40 ng/cigarette for both RYO cigarette tobaccos tested. BaP data for Brand F filter-tipped cigars were not obtained. Expressed per g of tobacco burned, the range in BaP results is sharply compressed, and the results are similar for cigars and cigarettes. Both cigar and RYO cigarette BaP results are also similar to those obtained with the Kentucky reference cigarettes, 39 ng/g. Calculated puff concentrations of BaP showed a correspondingly small range for all tobacco products evaluated, and no difference between cigars and RYO cigarette tobacco, with an overall mean of 61 ± 14 μg/m^3 (C.V. 23 percent). It is unclear why differences in puff frequency between cigars and RYO cigarettes appear to influence benzene levels and not those for BaP. Benzo(a)pyrene concentrations are about 10^5 higher than those usually found in urban ambient air.[16]

Lead

Mainstream tobacco smoke particulate lead results for all cigars and RYO cigarette tobaccos tested were below the limit of reliable quantitation, 0.2 μg/cigar or cigarette.

Discussion

The yields of benzene and BaP, per gram of tobacco burned, in the non-cigarette tobacco products tested here are similar to those previously reported for a reference cigarette. However, the lead values obtained are lower than those in previous work (e.g., 0.4 to 1.0 μg Pb/cigarette cited in reference 7). This is consistent with a decrease in atmospheric lead observed in recent years, concomitant with the

*Jenkins RA: Oak Ridge National Laboratory, personal communication, Sept. 5, 1988.

TABLE 1—Benzene and BaP in Mainstream Tobacco Smoke from Cigars and Roll-Your-Own Cigarettes[a]

Sample Type	Brand	N[b]	Benzene μg/cig[c]	Benzene μg/g	BaP ng/cig[c]	BaP ng/g	Puff Conc. (μg/m^3) Benzene (×10^{-6})	Puff Conc. (μg/m^3) BaP	Mean No. Puffs
Cigar	A	7	1025 ± 57	151 ± 9	248 ± 44	36 ± 5	2.9 ± 0.3	63 ± 12	107
	B	1	977	165	206	35	1.9	41	137
	C	2	316 ± 17	92 ± 4	135 ± 1	39 ± 1	1.7	71	53
	D	2	482 ± 65	246 ± 31	96 ± 23	49 ± 12	3.3	84	43
	E	1	732	122	292	49	1.4	54	157
	F	2	228 ± 68	157 ± 59	ND[d]	ND[d]	2.3 ± 0.7	N.D.	26
RYO Cigarette[e]	A	2	61 ± 13	75 ± 16	37 ± 3	45 ± 4	0.9 ± 0.1	53 ± 1	65
	B	2	50 ± 10	60 ± 13	43 ± 4	51 ± 3	0.7 ± 0.1	63 ± 4	58
Mean for Cigars				156 ± 52		42 ± 7	2.3 ± 0.7	63 ± 16	
Mean for Cigarettes				68 ± 11		48 ± 4	0.8 ± 0.1	58 ± 7	

a) Results are means and standard deviations.
b) N = number of trials.
c) "Cig" refers to both cigarettes and cigars.
d) ND = not determined.
e) Roll-your-own cigarettes prepared from cigarette tobacco.

TABLE 2—Benzene in Mainstream Smoke from Pipe Tobacco[a]

Brand	Benzene (μg/g)		Benzene Puff Conc. (μg/m³) × 10^{-5}		Mean No. Puffs[c]
	Cig. Config.[b]	Pipe Config.	Cig. Config.[b]	Pipe Config.	
A	85 ± 5	253 ± 25	0.54 ± 0.01	1.3 ± 0.2	45, 72
B	114 ± 23	277 ± 20	0.9 ± 0.3	1.50 ± 0.04	37, 84
C	144 ± 48	354 ± 34	1.1 ± 0.5	1.9 ± 0.1	38, 90
D	145 ± 11	363 ± 2	1.1 ± 0.1	1.51 ± 0.01	40, 72
E	207 ± 12	473 ± 22	1.5 ± 0.1	2.6 ± 0.6	40, 78
Mean value	139 ± 45	344 ± 86	1.0 ± 0.4	1.8 ± 0.5	
Overall mean	242 ± 126		1.4 ± 0.6		

a) Results for each brand are the mean and standard deviation for two trials.
b) "Cig." indicates "RYO cigarette."
c) The two values refer to RYO cigarette and pipe configurations, respectively.

decreased use of leaded gasoline. For example,[17] over the period 1975 to 1984, the highest calendar quarter mean lead concentration decreased at a downtown Los Angeles site from 4.9 to 0.7 μg/m³. Similar decreases have been observed in North Carolina, an important tobacco-growing State.**

Under regulations implementing California's Proposition 65,[18] carcinogens are deemed to pose no significant risk (NSR) if daily exposures do not exceed those expected to produce a lifetime excess cancer risk of 1 in 100,000. The daily NSR level set for benzene is 20 μg, and for BaP, it is 0.06 μg.

The significance of the levels of these chemicals measured in mainstream tobacco smoke can be judged by comparison to the NSR levels. For example, the amounts of benzene in mainstream smoke from burning one 1.2-g RYO cigarette, a 7-g, 16-cm ("Churchill size") cigar (e.g., cigar brand E), or a bowlful (e.g., 3.5 g) of pipe tobacco were approximately 3, 36 and 60 times the NSR level, respectively. The corresponding amounts of BaP from a RYO cigarette and the Churchill-size cigar were approximately 0.9 and five times the NSR level, respectively. Thus, benzene and BaP in mainstream tobacco smoke from less than one Churchill-size cigar, two RYO cigarettes or a bowl of pipe tobacco exceeded daily NSR levels under California's Proposition 65, using simulated smoking protocols. With smokers, actual exposures to these chemicals will vary by the degree to which the smoke is inhaled. Similarly, the puff concentrations reported are not intended to reflect concentrations potentially inhaled by smokers, since the puff is subject to substantial dilution by air not drawn in through the cigarette.

California's interest in limiting toxic exposures, as expressed through Proposition 65, may portend an increased national focus on the adverse health consequences of smoking. Recently, manufacturers of cigars, RYO cigarette and pipe tobaccos have added warning labels for these products sold in California. We recommend that warning labels be made a standard requirement for all such products sold in the US.

**Aerometric Information Retrieval System, Air Quality Subsystem Quick Look Report, US Environmental Protection Agency, Unpublished Report, 1989.

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Tobacco samples were obtained from retail stores by the Food and Drug Branch, California Department of Health Services. We wish to acknowledge the helpful comments of Dr. Roger Jenkins, Oak Ridge National Laboratory, regarding methodology for generating and sampling tobacco smoke. Dr. Jenkins also supplied the Kentucky reference cigarettes. We also acknowledge the helpful comments of Dr. H.C. Pillsbury, Federal Trade Commission. GC-MS determinations of benzene were provided by Dr. J. Hsu.

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APPENDIX
Tobacco Products Evaluated and Smoking Parameters Used[a]

Product Type	Brand	Weight Range, g	Length, cm	Puff/min
Cigar	A	7.1–8.6	13.8	2
Cigar	B	7.1–7.3	16	2
Cigar	C	4.4–4.8	11.5	2
Cigar	D	2.5–2.8	10.4	2
Cigar	E	6.4–7.1	15.9	2
Filter-Tipped Cigar	F	1.7–2.0	10.0	2
RYO[b] cigarette tobacco	A	1.2 ± 0.05	7.0	1
RYO cigarette tobacco	B	1.2 ± 0.05	7.0	1
Pipe tobacco	A	—[c]	—[c]	2,12[d]
Pipe tobacco	B	—	—	2,12
Pipe tobacco	C	—	—	2,12
Pipe tobacco	D	—	—	2,12
Pipe tobacco	E	—	—	2,12

a) Puff volumes were 35 ± 2 ml, and puff duration were 2.05 ± 0.05 sec in all cases. Butt lengths for cigars and RYO cigarettes were 25 and 23 mm, respectively. All tobacco products were preconditioned at ca. 60% R.H. for 24 to 48 hr before smoking. All uncertainty values in this table reflect ranges, not standard deviations.
b) Roll-your-own.
c) Evaluated as RYO cigarettes, 1.50 ± 0.05 g and 7.0 cm length each, and when smoked a pipe, consuming 1 to 2 g of the 3 to 4 g of tobacco used in filling the bowl.
d) Two-min in the RYO cigarette configuration, 12-min in the pipe.

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Rockefeller Foundation Grants: Global Environmental Program Announced

The Rockefeller Foundation has announced its new Global Environmental Program, a grant-making initiative to "promote the skills, attitudes, relationships and institutions necessary for environmentally sound international development." Whether humans can live in balance with the natural environment and whether all the countries and cultures of the world can achieve a reasonable and just standard of living are two emerging aspects of the same issue which will be addressed by the new program.

The new Global Environmental Program is part of the Foundation's International Program to Support Science-Based Development built on the premise that the well-being of people in developing nations can be advanced in important ways through the application of science and technology. The environmental initiative takes into account, however, that the application of science and technology to development in the industrialized nations has not always been kind to the environment. The challenge before the world community is to promote a more equitable sharing of the world's resources and yet avoid repetition of environmentally unsound development practices.

The Global Environmental Program will begin with four broad areas of interest for grant and fellowship activity:

- supporting young environmental scientists, analysts, policymakers, and community leaders who can be architects of future policy in development and the environment;
- advancing the development of economic analysis which can guide and illuminate the process of environmentally sound development;
- laying the groundwork for multilateral bargains and accords that will be necessary to address selected environmental issues; and
- Seeking innovative ways to help the United States public prepare for new environmental realities.

No special form is required for grant applications. However, they should include:

- a description of the project, with clearly stated plans and objectives;
- a comprehensive plan for the total funding of the project during and, where applicable, after the proposed grant period; and
- a listing of the applicant's qualifications and accomplishments and, if applicable, a description of the institutional setting.

Address grant applications and requests for further information to:

Global Environmental Program
The Rockefeller Foundation
1133 Avenue of the Americas
New York, NY 10036, USA

Long-Term Effectiveness of Mass Media Led Antismoking Campaigns in Australia

JOHN P. PIERCE, PHD, PETRA MACASKILL, MAPPSTAT, AND DAVID HILL, PHD

Abstract: A community antismoking campaign began in Sydney, Australia in 1983, and in Melbourne in 1984. These campaigns purchased prime-time television advertising spots to set the community agenda. An intense effort was made to ensure that antismoking activities were maximized at the school, organizational, and community level. Smoking prevalences in both cities from 1981 were fitted with a statistical model to identify any underlying trend, to assess any immediate impact, and to assess the longer term effect of continuing to conduct such campaigns, i.e. to identify any change in the underlying trend. During the years before the antismoking campaigns, there was no observable trend in smoking prevalence in either city. At the beginning of the campaigns, there was an immediate drop of more than two percentage points in male and female smoking prevalence in both cities. Thereafter, a decline of about 1.5 percentage points per year was observed among males. No post campaign trend was observed in smoking prevalence for women in either city. These data support conducting coordinated community campaigns to reduce current smoking prevalence. (*Am J Public Health* 1990; 80:565–569.)

Introduction

Smoking prevalence in Australia did not decline much from 1974 to 1983 according to age standardized comparisons,[1,2] in contrast to the pattern seen in other Western developed countries.[3] For this reason, a number of individual states in Australia introduced a coordinated campaign approach to promoting change in health behavior, as pioneered in cardiovascular disease by the Stanford Three Community Project[4] and the North Karelia Project.[5]

In 1983 in Sydney and in 1984 in Melbourne, the State Health Departments established anti-smoking steering committees composed of personnel from the health departments, the major voluntary organizations, health professional societies, and university public health departments. Television commercials were developed to motivate smokers to quit smoking and to set the agenda for professionals whom the campaign team hoped to involve in promoting nonsmoking, given the evidence of the effectiveness of medical advice.[6–8] The effectiveness of school education programs[9] also made establishing such programs a high priority within the community. Although the campaign committees in both cities shared campaign materials and ideas, the actual activities undertaken within each city were dictated by the perceived local needs and opportunities.

A detailed evaluation was undertaken of the first year of the campaign in Sydney using Melbourne, which did not have a campaign that year, as a reference community.[10,11] More than 80 percent of the population of Sydney remembered the television commercials. After these commercials were aired, use of services such as a telephone quit line and enrollment in cessation classes increased. The campaign was also associated with higher levels of intention to quit in Sydney, a variable that had previously been shown to be associated with actual quitting.[12] Changes in smoking prevalence during the first campaign year were assessed by self-reported smoking behavior (after validation with salivary cotinine[13]) from both cross-sectional and longitudinal surveys. No decrease was seen in the reference city, Melbourne. The pooled estimate of the difference in smoking prevalence attributable to the campaign was 2.8 percent (95% confidence interval = 0.5, 5.1).[11]

Since 1984, the campaigns have continued in both Melbourne and Sydney, spearheaded each year by commercials shown on prime time television for six to eight weeks during the winter months. In this paper, we assess the long-term effect of this approach to reducing the prevalence of smoking. We used data from both cities to determine whether these key intermediate goals were achieved:

- High public awareness of the campaign during each campaign year;
- A strengthening of the stand of health professionals, particularly physicians, against smoking;
- An increased proportion of the smoking population who both believed that smoking was harming their own health and also were being influenced by their social network to quit smoking.

The effectiveness of the campaign was assessed by using self-reported smoking data from 1981 to 1987 for each city.

Methods

General Design

This project has a before and after design in each of two intervention cities with a temporal lag of 12 months between the onset of the intervention in the second city. Prevalence data were obtained for several years before the onset of the intervention, as well as for the duration of the campaign. In 1983 and 1986, more detailed questions about health beliefs, intentions, and social influences were asked in each city. Small random surveys were undertaken approximately two months after the completion of each component of the television campaign to assess recall of commercials.

Mass Media Used in Campaigns

Television—In both Sydney and Melbourne, television advertising made up about two-thirds of the media budget for the campaigns. Typically, commercial spots were purchased during prime or fringe time for approximately four weeks out of eight at the start of the campaign year. Approximately 40 spots per week were shown during a schedule designed to ensure at least 50 percent market penetration in the Sydney

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and Melbourne metropolitan areas during each year of the campaign.

Production of commercials followed research on messages that were likely to be effective among the target audience. These commercials were designed to use strong visual images of the health consequences of smoking. Further, they sought an emotive response among the audience to ensure attention and to maximize the likelihood that viewers would reassess the hazards of smoking and then possibly quit in the near future (for description, see Appendix).[12,15] All of the commercials ended with a "Quit line" telephone number that people could call 24 hours a day to hear a recorded informative and encouraging message about quitting smoking.

Other Mass Media—A concerted effort was made to coordinate all mass media advertising around a central theme that was dictated by the message in the major television commercial being run at the time. For billboard advertising, a simple message from the television commercial was displayed at numerous sites, especially those near mass transit locations. Advertising in newspapers included the normal large advertisement and, in Sydney, a section was purchased for journalistic coverage of smoking-related events and issues. Antismoking skits by major personalities complemented the normal quit smoking commercials on the radio. In addition, the campaign generated many news releases and events that resulted in substantial news coverage in all the mass media.

Physician's Offices

In both cities, there was active collaboration with the Australian Medical Association to develop and distribute printed resources that physicians could use to encourage and help their patients quit smoking. In Melbourne, a special training videotape about smoking was produced and shown widely to family physicians. In Sydney, a special training program was available to family physicians to enable them to more effectively counsel their patients to stop smoking.[16]

Schools

In Sydney, two school programs were developed: a comprehensive kindergarten through 12th grade curriculum and a grade-specific peer or teacher led curriculum. By 1986, 80 percent of all schools in Sydney had at least one person who was trained by the campaign team in conducting the program of choice for that school. Sixty-four percent (88 percent of the 73 percent who responded) of these schools reported using these materials during the 1986 academic year. Further, the campaign team sponsored theatre performances, rock concerts, and sports activities with antismoking themes.

In Melbourne, kindergarten through 12th grade curriculum resources on smoking were made available to all schools, and project workers encouraged and helped teachers to use these resources. By 1986, in sample surveys of school children in both Sydney and Melbourne, more than 70 percent of 12–15-year-olds reported receiving at least minimal health education about smoking during the year.[17]

Other Community Activities

Each year, the campaigns either organized smoking cessation clinics or informed the population in each city about the availability of clinics. Training programs were available for health educators who might be appointed to local government or other affiliated positions. In addition, quit smoking display stands were regularly erected and staffed in shopping malls and at public fairs.

Assessing Smoking Prevalence

Between 1981 and 1987, 68,136 males and 70,634 females aged 16 years and over from the suburbs of metropolitan Sydney and Melbourne were interviewed in their homes as part of an ongoing weekly survey. Sampling procedures, reported in detail elsewhere,[11] involved selecting a household starting point at random from the electoral roll (in Australia, it is compulsory for citizens over 17 years of age to be registered on the electoral roll). Field interviewers proceeded to move from house to house in a clockwise direction until they obtained a cluster of 10 interviews from separate households. Within the household, the youngest person aged 16 years and over of a selected sex was interviewed. The resulting sample has been shown to be representative of the population on major sociodemographic variables.[11]

A smoker was defined as anyone who responded positively to questions on whether he or she smoked factory-made cigarettes, roll-your-own cigarettes, cigars, or a pipe. In two separate subsamples, self-reported smoking was validated with biochemical analysis of saliva for the presence of cotinine.[13]

Measuring Campaign Exposure

For each year of the campaign, recall of the television commercials was assessed on separate random samples of the population of at least 1,000 persons in the three months following the completion of the television phase of the campaign. Shortly after the completion of each annual television campaign, a subsample of persons in Sydney were shown a set of six still photographs taken from the commercials. They were first asked whether they had seen the commercial and, if they responded yes, what the message was (care was taken that no "giveaway" words or cigarettes appeared in the photographs). In Melbourne, the respondents were asked whether they had seen any television commercials against smoking, recently. The interviewer then probed all positive responses and recorded details of the message recalled.

Assessing Predictors of Quitting

In 1983 and 1986, additional random samples of the population of Sydney and Melbourne were surveyed. The Sydney survey included 271 smokers in 1983 and 557 smokers in 1986. The Melbourne sample included 217 smokers in 1983 and 550 smokers in 1986. Smoking respondents were asked to agree or disagree with a set of statements previously shown to be scales representing health beliefs and social influences.[12] The items in the health belief scale were "Smoking cigarettes doesn't greatly affect my health," "The problems with smoking only affect heavy smokers," and "I'd rather enjoy life as a smoker than live a little longer without cigarettes."

The items in the social influence scale were "I find that my smoking annoys people around me who don't smoke," "What I dislike about smoking is the smell that it leaves on clothes", "All the physicians that I know are strongly against smoking," "My close friends would prefer that I didn't smoke," and "I find that people are talking a lot more about smoking and the problems of giving up." Internal consistency reliability (Cronbach's alpha) on these scales have been

previously reported at 0.57 on the health benefit scale and 0.56 on the social influence scale.[12]

Statistical Analysis of Prevalence

For the purpose of analysis, data were grouped into intervals of six months for each city and were adjusted for age and sex to the estimated population of Sydney in 1986 using direct standardization. Salonen, *et al*,[18] have suggested using multiple linear regression models to evaluate the effectiveness of community-based intervention programs by testing for interaction effects between the type of community (intervention or reference) and the timing of the onset of the intervention effect. In this study, which lacked a nonintervention community, the following simplified form of the suggested model was fitted to each of the data sets.

$$P = \beta_0 + \beta_1 C + \beta_2 T + \beta_3 CT + \beta_4 S + \text{error}$$

where P represents the prevalence of smoking,

C is a binary indicator variable representing whether the data are from the precampaign (C = 0) or the postcampaign (C = 1) period,
T is the trend variable for time,
S is a binary indicator of season with a value of 1 for the first six months and −1 for the second six months of each year.

Using this model, β_0 is an estimate of the expected prevalence at the start of the campaign (T = 0) given the underlying trend in the precampaign period. β_1 is an estimate of the immediate effect of the intervention (as such an effect was demonstrated in a previous paper[15]). β_2 estimates the underlying trend in prevalence over the whole time period, and β_3 estimates the change in this underlying trend associated with the commencement of the campaigns. The coefficient β_4 provides an estimate of the effect of seasonal variation on the data. For each analysis, a model which included all the explanatory variables described above (full model) was fitted. A parsimonious model (adjusted for seasonal effect) was then developed by using backward elimination to remove variables, other than S, with the chosen level of significance set at 0.05. This reduced model identifies the pattern of change in prevalence in relation to the timing of the campaigns. A series of curvilinear (polynomial) models were also fitted to the data. Quadratic and cubic models were compared with the linear parsimonious models described above to assess whether the more complex models fit the data better.

Results

Population Knowledge of the Campaign

We assessed awareness about the antismoking campaign by asking people in each city what they recalled about the most public aspect of the campaign, the television commercials (Table 1). For all the major commercials in these campaigns, there was little difference between the recall of the commercial and identification of an appropriate antismoking message. In both cities, more than two-thirds of the community knew that an antismoking campaign existed in the years that such campaigns were conducted.

Action by Physicians

During the campaign, physicians' roles were assessed by asking respondents how strongly the physicians were against smoking; these data were then divided by age and sex for each city in 1983 and 1986 (Figure 1). Before the campaign, only 15 percent of males and females smokers over age 40 in Sydney thought their physicians were strongly against smoking. By 1986, this had increased dramatically to 47 percent for males and 50 percent for females. In those under the age of 40 in Sydney, this proportion increased from 36 percent to 48 percent in males and from 28 percent to 46 percent in females. In Melbourne males, this proportion increased from 35 percent to 41 percent in those under 40 years and from 28 percent to 35 percent in those over 40. In Melbourne females, this proportion increased from 27 percent to 47 percent in those under 40 years and from 33 percent to 37 percent in those over 40 years.

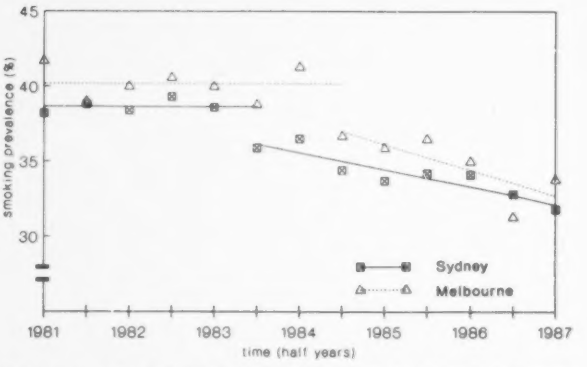

FIGURE 1—Age-Standardized Smoking Prevalence for Males in Sydney and Melbourne, 1981 to 1987

The Health Belief/Social Influence Variable

A previous study[12] had demonstrated that only a combination of personal beliefs about smoking and the environmental influences to quit was associated with an increased likelihood to quit. This proportion reporting both social influence and health beliefs was low in those over the age of 40 years in Sydney in 1983 (males, 10 percent; females, 12 percent). By 1986, this proportion had increased to 29 percent in males and 27 percent in females. In those under 40 in Sydney, males increased from 34 percent to 53 percent and females increased from 30 percent to 44 percent. In those over 40 years in Melbourne, males increased from 21 percent to 29 percent and females increased from 22 percent to 32 percent. In those under 40 years in Melbourne, males increased from 30 percent to 43 percent and females increased from 31 percent to 48 percent.

Smoking Prevalence

We considered both the full and parsimonious statistical models fitted to the age-standardized data for men and

TABLE 1—Percentage of Males and Females Who Recalled Television Advertising in Sydney and Melbourne

Year	% Males	% Females	N
Sydney (aided by picture prompt)			
1983	84	84	2,970
1984	72	80	1,046
1985*	79	76	996
1986	92	94	457
Melbourne (unaided)			
1984	70	67	886
1985	73	68	1,818
1986	68	67	1,830

*Only 14-to-19 year-olds.

women separately for each city. For men, these models fit the data very well, explaining 96 percent of the variation in Sydney and 91 percent in Melbourne. For females, the best models did not fit quite so well, explaining 72 percent of the variation in the Sydney sample and 79 percent in the Melbourne sample. However, the most parsimonious model for the Melbourne women sample was not consistent with the other models. A consistent model fitted almost as well and explained 68 percent of the variation.

Figures 1 and 2 are graphical representations of the trends identified in the parsimonious models in conjunction with the observed data. For this presentation, the consistent model was chosen for Melbourne women.

Thus, the best estimate of smoking prevalence among Sydney men (Table 2) indicated that prevalence did not change in the three years before the campaign (June 1983) and was 38.7 percent ($\hat{\beta}_0$ in the parsimonious model). From 1981 to 1987, there was no significant underlying trend in smoking prevalence other than that associated with the campaign. The introduction of the campaign coincided with an estimated immediate drop in smoking prevalence of 2.5 percentage points in that six-month period. Continuation of the campaign was associated with a further drop of 1.12 percentage points per year ($2 \cdot \hat{\beta}_3$). These estimates are consistent with those provided by the full model.

For women, smoking prevalence had again been constant during the years before the campaign and was 31.6 percent in Sydney and 29.8 percent in Melbourne. In Sydney, there was an immediate drop of 2.6 percentage points associated with the introduction of the campaign but smoking prevalence did not significantly decline in the later years of the campaign. Again, this estimate is consistent with that from the saturated model.

Men in Melbourne responded similarly to those in Sydney. As Table 3 shows, the expected prevalence of smoking at the beginning of the campaign (June 1984) was 40.1 percent ($\hat{\beta}_0$). Again, there was no underlying trend. With the start of the campaign, there was an immediate drop of 2.9 percentage points. A further decline of 1.9 percentage points per year occurred in conjunction with the continued campaign. Once again these estimates are consistent with those from the full model.

In Melbourne, two models fitted the data and, as indicated earlier, model 2 is the preferred model. Smoking prevalence prior to the campaign was 30.9 percent. The immediate effect of the campaign was a 2.5 percentage point drop in smoking prevalence, again with no subsequent decline associated with the continued campaign. However, in this instance, the estimate from the full model is very different, highlighting the uncertainty that exists as to the true underlying model.

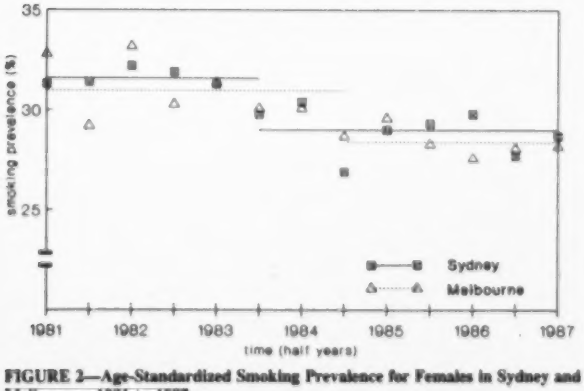

FIGURE 2—Age-Standardized Smoking Prevalence for Females in Sydney and Melbourne, 1981 to 1987

TABLE 2—Statistical Models for Smoking Among Males and Females in Sydney

		Full Model Estimate (SE)	Parsimonious Model Estimate (SE)
Sydney Males	$\hat{\beta}_0$	39.06 (0.67)	38.67 (0.28)
	$\hat{\beta}_1$	−2.91 (0.79)	−2.52 (0.49)
	$\hat{\beta}_2$	0.13 (0.20)	
	$\hat{\beta}_3$	−0.69 (0.22)	−0.56 (0.10)
	$\hat{\beta}_4$	−0.04 (0.18)	−0.04 (0.17)
		(R^2 = 0.96, 8df)	(R^2 = 0.96, 9df)
Females	$\hat{\beta}_0$	031.69 (1.02)	31.56 (0.42)
	$\hat{\beta}_1$	−2.11 (1.20)	−2.61 (0.54)
	$\hat{\beta}_2$	0.05 (0.31)	
	$\hat{\beta}_3$	−0.23 (0.34)	
	$\hat{\beta}_4$	0.38 (0.27)	0.32 (0.26)
		(R^2 = 0.77, 8df)	(R^2 = 0.72, 10df)

Comparison with other Models

The goodness of fit of these parsimonious linear models was compared with quadratic and cubic models. In each case, the best-fit linear model was a superior fit to either of the alternative models, except for Melbourne women, again emphasizing the problems in interpreting this set of data.

Discussion

This study suggests that smoking prevalence in the two major Australian capital cities did not decline prior to the campaign, as previously observed for the period 1974 to 1980 for Australia as a whole.[1–3] The mass media led antismoking campaigns coincided with a marked drop in smoking prevalence during the six-month period immediately following the start of the campaign for all persons in Sydney and for men in Melbourne (the effect on Melbourne women is less clear). The estimated size of this immediate effect was 2.6 percentage points in Sydney. A larger 2.9 percent effect of the antismoking campaign was seen among Melbourne men 12 months later. If we accept the model that suggests that women in Sydney and Melbourne reacted similarly to the antismoking campaign, the campaign immediately reduced smoking prevalence in Melbourne by 2.5 percent, an amount

TABLE 3—Statistical Models for Smoking Among Males and Females in Melbourne

		Full Model Estimate (SE)	Parsimonious Model Estimate (SE)	
Melbourne Males	$\hat{\beta}_0$	39.89 (1.00)		40.12 (0.42)
	$\hat{\beta}_1$	−2.64 (1.33)		−2.87 (0.93)
	$\hat{\beta}_2$	−0.06 (0.22)		
	$\hat{\beta}_3$	−0.89 (0.36)		−0.95 (0.27)
	$\hat{\beta}_4$	0.58 (0.34)		0.57 (0.32)
		(R^2 = 0.91, 8df)		(R^2 = 0.91, 9df)
			Model 1	Model 2
Females	$\hat{\beta}_0$	29.77 (0.83)	29.40 (0.26)	30.93 (0.41)
	$\hat{\beta}_1$	−0.53 (1.10)		−2.52 (0.61)
	$\hat{\beta}_2$	−0.29 (0.19)	−0.37 (0.07)	
	$\hat{\beta}_3$	−0.04 (0.30)		
	$\hat{\beta}_4$	0.64 (0.28)	0.65 (0.25)	0.56 (0.30)
		(R^2 = 0.80, 8df)	(R^2 = 0.79, 10df)	(R^2 = 0.68)

similar to the estimates of the effect obtained in both men and women for Sydney one year earlier. In the alternative model, women in Melbourne were the only group who recorded a decreasing prevalence of smoking during the precampaign period. Given that there was no downward trend in smoking prevalence throughout the 1970s,[1-3] this model must be considered less plausible.

For men in both cities, continued antismoking campaigns were associated with an average continued decrease in smoking prevalence of approximately 1.5 percent per year. The situation for women is less clear, but suggests that the continued campaigns in Sydney and Melbourne were not associated with any further decrease in smoking prevalence among women.

This analysis of changes in community smoking prevalence does not consider the effect that such campaigns may have on uptake of smoking. In any given year, the influence of new smokers between the ages of 16 and 20 years (the teen years included in this data set) on the overall prevalence will be small. Before we can conclude how effective these campaigns really are, changes in the uptake of smoking among teenagers need to be explored. However, even with the effect on uptake of smoking being unresolved, this study supports conducting coordinated mass media antismoking campaigns (using purchased television time) as an effective way to reduce smoking prevalence in the community. Whether conducting campaigns for up to four consecutive years is cost-effective will be the topic of a subsequent paper.

APPENDIX

TV Commercials by City and Year

	Year			
Commercial	1983	1984	1985	1986
"Sponge"[a]	S	S, M	M	
"I've had enough"[b]	S	S, M	M	
"Mrs. Holden"[c]		S		
"Yul Brynner"[d]				S, M
"Heartbeat"[e]			M	M
"Stairs"[f]				M
"Mirrors"[g]			S	
"Tough Guy"[h]			S	
"Mates"[i]			S	

S = Sydney, M = Melbourne.
[a] an analogy between a sponge and a lung soaking up tar.
[b] smoking histories of three people outlining reason for quitting included ashtray on a girl's face and modeling of calling the Quitline.
[c] a laryngectomy patient (with subtitles) outlining her smoking related problems with communications.
[d] a posthumous exhortation "Whatever you do, don't smoke."
[e] cardiograph used to outline smoking effects on circulation."
[f] mirror distortion to reduce fears of weight gain and stress with quitting.
[g] linked smoking to shortening a young girl's life.
[h] teens, visiting cancer patient, review smoking-related disease.
[i] outlined social problems teens might face if they smoke.

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A Twin Study of the Effects of the Vietnam Conflict on Alcohol Drinking Patterns

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Abstract: This study examines the association between military service in Southeast Asia and alcohol drinking patterns in 2,169 male-male monozygotic twin pairs who both served on active military duty during the Vietnam era (1965–75). Data on alcohol drinking were collected in 1987 by mail and telephone interview. The alcohol drinking measures include three indicators of abstention (lifetime abstainer, lifetime non-regular drinker, and current abstainer) and two indicators of consumption (average daily ethanol consumption and high consumption). In unadjusted and co-twin adjusted analyses, neither service in Southeast Asia nor combat exposure was significantly associated with any measure of abstention. In the co-twin adjusted analysis, there was no association of Southeast Asia service and combat exposure with average daily ethanol consumption. After adjustment for co-twin effects, 4.0 percent of non-Southeast Asia veterans were high consumers compared to 6.7 percent of Southeast Asia veterans who served in high combat. We conclude that prior military service in a war zone has a relatively modest long-term effect on the alcohol drinking patterns of male veterans. (*Am J Public Health* 1990; 80:570–574.)

Introduction

The relationship between service in the military and alcohol drinking patterns is a subject of considerable debate. Numerous clinical studies have concluded that veterans have high rates of alcoholism.[1–3] The evidence for increased alcohol consumption among veterans in non-clinical population samples is equivocal; some studies conclude that increased consumption among veterans is a function of demographic differences between veterans and nonveterans,[4,5] while other more recent research indicates that veterans consume more alcohol than nonveterans regardless of demographic differences.[6]

A number of investigations have examined the association between military service in Southeast Asia and alcohol consumption. Some studies have found that service in Southeast Asia and exposure to combat are associated with increased rates of alcoholism and problem drinking,[7,8] while other research has failed to demonstrate any association with alcohol consumption patterns.[9–12]

The potential for selection bias is a fundamental problem in all studies of alcohol drinking among veterans. Observed associations between military service experience and alcohol drinking patterns may simply reflect an underlying association of predispositional characteristics with both military service experience and alcohol drinking. The present study addresses this problem by examining alcohol drinking in monozygotic twin pairs contained in the Vietnam Era Twin Registry.

Methods

Data Sources and Study Eligibility

The Vietnam Era Twin Registry contains 7,375 male-male twins who both served in the military during the official Vietnam era (May 1965-August 1975). A complete description of the development of the registry can be found elsewhere,[13] and only a brief overview is presented here.

A survey of all twins on the registry was conducted in early 1987, with the goals of collecting information on a wide range of physical and psychological indicators of health. The overall case-wise response rate was 74 percent and the pair-wise response rate was 65 percent or 4,774 pairs. Twins were eligible for the present study if: both members of the pair responded to the survey, the pair was originally ascertained from the US Department of Defense computer files, and complete information was available for all alcohol drinking measures. After applying the exclusion criteria, the final number of eligible twin pairs was 4,048.

Zygosity was evaluated by using a series of questions on twin similarity and limited blood group typing obtained from the military records.[14] Of the 4,048 pairs meeting the eligibility requirements for the present study, 2,169 (53.6 percent) were monozygotic, 1,773 (43.8 percent) were dizygotic and 106 (2.6 percent) were of indeterminant zygosity. The present study focuses exclusively on the monozygotic twin pairs.

Service in Southeast Asia and Combat Exposure

Military service in Southeast Asia was determined by the response to the following question: "When you were in the military, were you stationed in Vietnam, Laos, or Cambodia; in the waters in or around these countries; or fly in missions over these areas?" Those men who responded in the affirmative were then asked a set of questions about 18 specific combat roles or experiences (see Appendix A). For each item the veterans indicated whether they had that role or experience while serving in Southeast Asia. An index of combat exposure was constructed by taking the summation of all positive responses for an individual. For analytical purposes the combat index was grouped into five levels: (non-Southeast Asia service; Southeast Asia service, no combat; low, combat index = 1–2; medium, combat index = 3–6; high, combat index > 6). Elsewhere,[15] we have demonstrated that in a subset of twins who completed the health survey twice more than a year apart, concordance was high (weighted kappa = 0.84) for the combat exposure index. Likewise, the combat index was found to be significantly correlated with objective indicators of threat to life and limb as measured by combat medals and decorations.

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Measures of Alcohol Drinking

A negative response to having had more than 20 alcoholic drinks in their entire life defines a lifetime abstainer. Veterans who were not lifetime abstainers, were asked at what age they started drinking alcoholic beverages regularly; as part of the response option to this question the veteran could indicate that he never drank regularly. This is referred to as lifetime non-regular drinker. The next question asked if the veteran drinks alcoholic beverages now. This provides a third measure of alcohol abstention which separates current drinkers from noncurrent drinkers. It should be noted that the lifetime abstainer measure is subsumed by the lifetime non-regular drinker measure, which is subsumed by the current abstainer measure.

For men who are current drinkers quantity was obtained as the average number of drinks consumed on days when the veteran had at least one drink; frequency was obtained in terms of the average number of days per week that the veteran had at least one drink. If the ethanol content of an unspecified drink is assumed to be 0.5 ounces, then average daily ethanol consumption can be calculated as: (frequency × quantity × 0.5)/7. Following the guidelines of the National Institute of Alcohol Abuse and Alcoholism,[16] average daily ethanol consumption can be categorized into four drinking levels: abstainers, average intake of 0.0 ounces ethanol daily; light drinkers, 0.01–0.21 ounces ethanol daily; moderate drinkers, 0.22–0.99 ounces ethanol daily; heavy drinkers, 1.00 or more ounces ethanol daily. A second measure of alcohol consumption is constructed based on the number of ounces of ethanol corresponding to the upper fifth percentile of the average daily ethanol consumption distribution. This measure, referred to as high consumption, conformed to a usual intake of five drinks per day (2.5 ounces of ethanol) in the present study.

Reliability of Alcohol Drinking Measures — In 1985 a pilot study was conducted of 150 veteran twin pairs born in the State of Connecticut. This pilot study was designed as a field test of the methodology to be used in the main study. The measures of alcohol drinking in the field test and the subsequent survey were identical. Because these 150 twin pairs were included in the main study using the Vietnam Era Twin Registry it is possible to evaluate the reliability of the five measures of alcohol drinking.

Complete alcohol drinking data were available from 191 individuals from both studies. Lifetime abstention had a kappa of 0.43, a Yule's Y[17] of 0.77, and a 97 percent overall agreement. Considerably less reliable was the lifetime non-regular drinker measure, which had a kappa of 0.10, Y of 0.37, and a 82 percent overall agreement. The current abstainer measure had a kappa of 0.44, a Y of 0.48, and an overall agreement of 78 percent.

Both of the alcohol consumption measures demonstrated similar levels of reliability. Average daily ethanol consumption had a weighted kappa of 0.55; overall agreement was 81 percent. The measure of high consumption had a kappa of 0.57, a Y of 0.72 and an overall agreement of 92 percent.

Statistical Analysis

The analysis begins by randomly selecting one member of each monozygotic twin pair as the index twin and calculating the association between Southeast Asia service and combat exposure with each of the five alcohol drinking measures. This yields the association of Southeast Asia service and combat exposure with alcohol drinking, unadjusted for co-twin effects. Hypothesis testing for these contingency tables uses standard Chi-square methods and the Mantel extension to test for a linear trend in proportions when appropriate.[18]

The potential confounding effect of co-twin alcohol drinking on the association between index twin alcohol drinking and military service in Southeast Asia is addressed using a logistic regression model. In this model, index twin alcohol drinking is the dependent variable while co-twin alcohol drinking and index twin military service in Southeast Asia are the independent variables. The logistic regression model, adapted for twin data, provides an estimate of the probability of an index twin having an event (e.g., being a lifetime abstainer) based on the co-twin's event history and the index twin's service in Southeast Asia or combat exposure.[19] Hypothesis testing is conducted by dividing the logistic regression coefficient for Southeast Asia service or combat exposure (entered as a five level ordinal variable) by its respective standard error; when squared this follows a Chi-square distribution with one degree of freedom.

The potential confounding effects of demographic and military service factors were examined in a logistic regression model that already included co-twin alcohol drinking. Confounding factors include: years of education at enlistment, branch of service, length of active duty military service, age at enlistment, calendar year of discharge, and score on a general aptitude test taken at enlistment. These factors were examined both singly and in combination, and did not substantially alter the results from the co-twin adjusted analysis.

Results

Table 1 displays the nearly identical distribution of the three measures of alcohol abstention and the two measures of alcohol consumption for index twins and co-twins separately.

Table 2 displays the joint frequency distribution of the alcohol drinking measures with service in Southeast Asia and combat exposure for index twins. There is no evidence of a trend in the prevalence of lifetime abstention, lifetime non-regular drinking, and current abstaining with increasing levels of combat exposure. Conversely, veterans who served in Southeast Asia tend to consume more alcohol than

TABLE 1—Distribution of Alcohol Drinking Measures for Monozygotic Twin Pairs in the Vietnam Era Twin Registry

Alcohol Drinking Measures	Index Twin (n = 2,169) %	Co-Twin (n = 2,169) %
A. Lifetime Abstainer		
Yes	6.2	6.5
No	93.8	93.5
B. Lifetime Non-Regular Drinker		
Yes	24.2	24.9
No	75.8	75.1
C. Current Abstainer		
Yes	37.3	37.2
No	62.7	62.8
D. Average Daily Ethanol Consumption		
Abstainer	37.3	37.2
Low	19.8	20.8
Moderate	24.2	23.9
Heavy	18.7	18.2
E. High Consumption		
Yes	5.5	5.4
No	94.5	94.6

TABLE 2—Distribution of Alcohol Drinking Measures by Southeast Asia (SEA) Service and Combat Exposure in Monozygotic Index Twins

	Service in Southeast Asia		Combat Exposure				
	No	Yes	Non SEA	SEA No Combat	SEA Low Combat	SEA Med Combat	SEA High Combat
	(n = 1,340)	(n = 829)	(n = 1,340)	(n = 196)	(n = 222)	(n = 245)	(n = 166)
Alcohol Drinking Measures	%	%	%	%	%	%	%
A. Lifetime Abstainer							
Yes	6.6	5.6	6.6	7.6	3.2	5.3	6.6
No	93.4	94.4	93.4	92.4	96.8	94.7	93.4
	(X^2 = 0.916, p = 0.339)			(X^2_{trend} = 0.835 with 1 d.f., p = 0.361)			
B. Lifetime Non-Regular Drinker							
Yes	24.6	23.4	24.6	28.6	22.1	20.0	24.1
No	75.4	76.6	75.4	71.4	77.9	80.0	75.9
	(X^2 = 0.419, p = 0.517)			(X^2_{trend} = 1.352 with 1 d.f., p = 0.245)			
C. Non-Current Drinker							
Yes	38.4	35.5	38.4	39.8	33.3	33.1	36.8
No	61.6	64.5	61.6	60.2	66.7	66.9	63.2
	(X^2 = 1.929, p = 0.165)			(X^2_{trend} = 2.395 with 1 d.f., p = 0.122)			
D. Average Daily Ethanol Consumption							
Abstainer	38.4	35.5	38.4	39.8	33.3	33.1	36.8
Light	19.6	20.1	19.6	21.9	22.5	16.7	19.9
Moderate	24.9	23.0	24.9	23.0	23.4	25.7	18.7
Heavy	17.0	21.4	17.0	15.3	20.7	24.5	24.7
	(X^2 = 3.738, p = 0.053)			(X^2_{trend} = 6.625 with 1 d.f., p = 0.010)			
E. High Consumption							
Yes	4.6	7.0	4.6	5.6	5.0	8.6	9.0
No	95.4	93.0	95.4	94.4	95.0	91.4	91.0
	(X^2 = 5.898, p = 0.015)			(X^2_{trend} = 9.320 with 1 d.f., p = 0.002)			

veterans who did not serve in Southeast Asia (p = 0.053). The prevalence of light and moderate consumption is similar regardless of where a veteran served, but there are 4.4 percent more Southeast Asia veterans than non-Southeast Asia veterans who are heavy alcohol consumers. The extent of combat exposure is directly associated with increased average daily ethanol consumption. Non-Southeast Asia veterans and Southeast Asia veterans who experienced no combat consume about the same amount of alcohol, but veterans who have been exposed to either medium or high combat are found to consume more alcohol. When high consumption is examined, a 2.4 percent excess of high consumers is found in Southeast Asia veterans compared to non-Southeast Asia veterans. Further, a nearly monotonic gradient is observed in the prevalence of high alcohol consumption with combat exposure.

The association of the five alcohol drinking measures with Southeast Asia service and combat exposure, after adjustment for co-twin effects, is displayed in Table 3. None of the three abstention measures as well as average daily ethanol consumption are associated with Southeast Asia service or combat exposure. However, of all index twins who served in Southeast Asia, 5.6 percent are classified as high consumers, compared to 3.9 percent of index twins who did not serve in Southeast Asia (p = 0.048). Similarly, the ordinal index of combat exposure remains positively correlated with the co-twin adjusted prevalence of index twins who are classified as high consumers (p = 0.034).

Discussion

Our results demonstrate a complex pattern of association between military service experience and alcohol drinking patterns. The abstention measures are not associated with military service experience in either the unadjusted or adjusted analysis. In the unadjusted analysis there is a strong relationship of both measures of alcohol consumption with Southeast Asia service and combat exposure. In the cotwin adjusted analysis, average daily ethanol consumption is no longer significantly related to service in Southeast Asia and combat exposure; the association of high consumption with Southeast Asia service and combat is substantially diminished.

What is the likelihood that our findings are due to bias? The principal biases that are likely to be operating in this study relate to uncontrolled confounding, non-response, and flaws in measurement due to the self-reporting of military service experience and alcohol drinking. The principal uncontrolled confounding factor in this study is the pattern of alcohol drinking prior to entering military service. We chose not to collect measures of pre-military service alcohol drinking because of the potential of biased recall. It seems highly probable that the recollection of alcohol drinking patterns 15 to 20 years ago might be influenced by whether a veteran served in Southeast Asia.

Non-response bias might distort our results if veterans who responded are systematically different from veterans who did not respond *and* this difference is related to both alcohol drinking and military service in Southeast Asia. To gain further insight into the possibility of non-response bias we compared the association of Southeast Asia service with demographic and military service confounding factors (years of education at enlistment, branch of service, length of active duty military service, age at enlistment, calendar year of discharge, and score of the general aptitude test taken at enlistment), in responders and nonresponders. This analysis revealed that the potential for bias exists for education at enlistment and age at enlistment. In particular, education at enlistment was directly related to the probability of Southeast Asia service in responders and inversely related to the

TABLE 3—Distribution of Alcohol Drinking Measures by Southeast Asia (SEA) Service and Combat Exposure in Monozygotic Index Twins Adjusted for Co-Twin Alcohol Drinking Measures

	Service in Southeast Asia		Combat Exposure				
	No	Yes	Non SEA	SEA No Combat	SEA Low Combat	SEA Med Combat	SEA High Combat
Alcohol Drinking Measures	%	%	%	%	%	%	%
A. Lifetime Abstainer							
Yes	4.1	3.9	4.1	4.1	4.0	3.9	3.9
No	95.9	96.1	95.9	95.9	96.0	96.1	96.1
	(X^2 = 0.07, p = 0.789)			(X^2 = 0.05 with 1 d.f., p = 0.815)			
B. Lifetime Non-Regular Drinker							
Yes	21.5	21.6	21.8	21.5	21.2	20.9	20.6
No	78.5	78.4	78.2	78.5	78.8	79.1	79.4
	(X^2 = 0.01, p = 0.934)			(X^2 = 0.200 with 1 d.f., p = 0.654)			
C. Non-Current Drinker							
Yes	36.8	34.9	36.8	36.1	35.3	34.5	33.7
No	63.2	65.1	63.2	63.9	64.7	65.5	66.3
	(X^2 = 0.72, p = 0.396)			(X^2 = 0.94 with 1 d.f., p = 0.333)			
D. Average Daily Ethanol Consumption							
Abstainer	35.0	33.1	35.2	34.2	33.2	32.2	31.2
Light	23.8	23.6	23.8	23.7	23.6	23.5	23.4
Moderate	26.7	27.7	26.6	27.2	27.7	28.2	28.7
Heavy	14.5	15.6	14.4	14.9	15.5	16.1	16.7
	(X^2 = 1.07, p = 0.301)			(X^2 = 2.29 with 1 d.f., p = 0.131)			
E. High Consumption							
Yes	3.9	5.6	4.0	4.5	5.2	5.9	6.7
No	96.1	94.4	96.0	95.5	94.8	94.1	93.3
	(X^2 = 3.89, p = 0.048)			(X^2 = 4.52 with 1 d.f., p = 0.034)			

probability of Southeast Asia service in non-responders. Likewise, there was no difference in Southeast Asia service by age at enlistment in responders, but in non-responders there was an increased probability of serving in Southeast Asia for veterans who enlisted after the age of 19.

Bias due to self-reported military service experiences is unlikely to provide an explanation for our results. The measurement of Southeast Asia service is in accord with Southeast Asia service as determined from the military record (kappa = 0.86). We have previously demonstrated that the index of combat exposure is reliable and valid.[15] Likewise, the five alcohol drinking measures used in this study demonstrated acceptable reliabilities.

It is difficult to relate our findings to previous research on alcohol drinking among Vietnam era veterans[7,8,11,20,21] because of the major methodological differences among studies. The definition of "exposed" and "unexposed" cohorts is highly variable across studies. Some compare Vietnam era veterans with non-veterans without any measure of actual service in Southeast Asia[22] while others compare combat exposed and non-exposed Southeast Asia veterans without any non-veteran comparison group.[20] Each of the studies uses a different set of indicators for alcohol drinking behavior, ranging from a diagnosis of alcohol abuse and dependence,[8] to measures of usual alcohol beverage consumption.[21] The time interval between exposure and the measurement of alcohol drinking behavior also displays great variability across studies. For example, Robins'[10] initial data collection took place within two years of discharge from active duty while Kulka, *et al*,[12] collected data more than 15 years following discharge. Finally, and perhaps most importantly, there are major differences in the way each study addresses the issue of confounding. If factors related to both military service and alcohol consumption are not adequately controlled in the study design or analysis, then the observed results could be wholly or partially confounded.

Boscarino,[21] using data collected in 1977, found a positive association between service in Southeast Asia and three measures of alcohol consumption (usual daily intake, heavy drinking during the past 30 days, and binge drinking). That study is especially troublesome since it failed to exclude females from the analysis and the confounder adjustment was based solely on demographic factors. In a recent study, the Centers for Disease Control[8] found an increased prevalence of alcohol abuse and dependence in Southeast Asia and combat exposed veterans compared with non-Southeast Asia veterans. That study controlled for potential confounders by restricting the study participants to male Army veterans and by matching Southeast Asia veterans to non-Southeast Asia veterans based on period of service and age. During the analysis numerous potential confounders were examined and found to have no effect. However, the analysis failed to account for familial alcohol drinking patterns.

Card[9] used a single question on heavy alcoholic beverage intake to compare an age-matched cohort of non-veterans, veterans who did not serve in Southeast Asia, and veterans who served in Southeast Asia. Using a complex research design that involved matching on over 50 premilitary service factors, she found no difference in the prevalence of heavy drinking according to service in Southeast Asia. Roth[11] found a small but significant association between military service in Southeast Asia and a composite measure of alcohol intake and problem drinking, after multi-factor adjustment; she found no association between combat exposure and the same composite drinking measure. Yager, *et al*,[7] using a slightly different measure of alcohol drinking in this same cohort, came to a different conclusion; neither an indicator of heavy daily alcohol consumption nor a problem drinking scale was related to service in Southeast Asia. A significant association was found between combat and a measure of heavy daily

consumption. These conflicting reports obtained from the same data decrease the credibility of both studies.

Based on our findings, and the inconsistent evidence from previous research, we conclude that service in Southeast Asia and exposure to combat have relatively modest long-term effects on the alcohol drinking patterns of Vietnam era veterans.

APPENDIX A

Eighteen Indicators of Combat Exposure

1. In an artillery unit which fired on the enemy
2. Flew in an aircraft (reconnaissance, or fixed wing F-14, B-52, etc.)
3. Flew helicopter attack gunships or medivacs
4. Stationed at a forward observation post
5. Tunnel rat checking enemy base camps
6. Served on river patrol or gunboat
7. Demolitions expert in the field
8. Assigned to graves and registration to retrieve dead bodies from the field
9. Served as a medic in combat
10. Received incoming fire
11. Encountered mines and booby traps
12. Received sniper or sapper fire
13. Unit patrol ambushed
14. Flew in aircraft (fixed wing or helicopters) and was shot down
15. Engaged VC and/or NVA in firefight
16. Saw Americans killed, and/or saw Vietnamese killed
17. Wounded
18. Captured by the enemy

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Violence during Pregnancy and Substance Use

HORTENSIA AMARO, PHD, LISE E. FRIED, MSPH, HOWARD CABRAL, MPH, AND BARRY ZUCKERMAN, MD

Abstract: Violent incidents were assessed as part of a prospective study of 1,243 pregnant women. Participants were predominantly poor, urban, minority group women. Seven percent (n = 92) of women reported physical or sexual violence during pregnancy. Most of the women (94 percent) knew their assailant. Victims of violence were at greater risk of having a history of depression and attempted suicide, having more current depressive symptoms, reporting less happiness about being pregnant, and receiving less emotional support from others for the current pregnancy. Comparisons of victims and non-victims showed that victims were more likely to be users of alcohol and drugs. In addition, partners of victims were more likely to use marijuana and cocaine. When possible confounders were controlled using multivariable analyses, a woman's alcohol use during pregnancy and her partner's drug use were independently associated with an increased risk of being a victim of violence during pregnancy. Results of this study highlight the importance of assessing exposure to violence during prenatal care, especially among women who are heavy users of alcohol or drugs or whose partners use these substances. (*Am J Public Health* 1990; 80:575–579.)

Introduction

A substantial proportion of women in the general population experience abuse and violence.[1–8] Two recent studies[6,7] indicate that 4 to 8 percent of pregnant women experience abuse during pregnancy and that this problem may be largely unreported to health care providers. Since the risk of abuse is higher for women in young adulthood and this time coincides with women's most active childbearing years,[9] the experience of violence and abuse during pregnancy deserves special attention.

In spite of the prevalence of violence experienced by women, and its damaging impact on women's health and mental health, few studies have attempted to identify the factors associated with violence during pregnancy. One such study found little difference between abused and non-abused pregnant women in race, age, marital status, employment or educational status.[6] Another study[7] found that, compared to non-abused women, women who experienced abuse during pregnancy were of lower socioeconomic status, higher parity, more likely to be single, and had a history of depression or other psychiatric symptoms. Abused women were also more likely to report alcohol use, and a trend in the data indicated that they may also have higher levels of illicit drug use.[7] While this preliminary finding is consistent with previous associations between alcohol and drug use and family violence in the general population,[8] methodological problems such as small numbers of abused women in the sample and reliance on self-reported drug use[10] limit firm conclusions about the role of these factors during pregnancy.

The aims of the present analysis are: to describe the prevalence and patterns of violent incidents during pregnancy; to describe the association between demographic and psychosocial characteristics and violence during pregnancy; to investigate the association between the experience of violence during pregnancy and the use of alcohol and illicit drugs by pregnant women and their partners; and to investigate the association between the experience of violence during pregnancy and newborn outcomes.

Methods

Sample Recruitment

Subjects were consecutively recruited from July 14, 1984 through June 30, 1987 in the Women's and Adolescent Prenatal Clinics of Boston City Hospital. Eligible subjects were English- or Spanish-speaking women who were willing to give informed consent for study participation. The protocol was approved by the Human Studies Committee of Boston City Hospital. Subjects were protected from the use of data for criminal prosecution by a Writ of Confidentiality obtained under Title 42 of the United States Code Section 242A.

Assessment of Participants

All participants were interviewed during the prenatal and postpartum period by a trained bilingual interviewer. To minimize attrition, participants were paid $10 for each interview. The first interview assessed violent incidents occurring in the time period from three months prior to the calculated day of conception through the day of the interview. The postpartum interview determined violent incidents from the time of the previous interview through the time immediately prior to the delivery of the baby. Violent incidents were ascertained by asking the following question, "Were you physically threatened or abused, or were you involved in any fights or beatings?" The respondents were then asked to "describe what happened," and probed to find out whether the assailant was known to the victim, the type of injury, whether they had seen a doctor, and whether they were hospitalized overnight. Victims of violence were defined as those women who experienced physical or sexual violence during their pregnancies. Verbal threats or emotional abuse were not included as an episode of violence in this analysis.

A close-end, forced-choice interview elicited sociodemographic characteristics and the timing and frequency of the use of cigarettes, alcohol, marijuana, cocaine, and other illicit psychoactive substances prior to and during pregnancy. Data on drug and alcohol use were obtained for the same time periods as were data on episodes of violence. Participants were asked to furnish urine samples at the time of each interview and were informed that their urine would be assayed for "marijuana metabolites and prescription and nonprescription drugs," but that these results would remain confidential and not become part of their clinical record. The

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use of other substances such as alchohol and other drugs was ascertained by self-report alone. Urine assays were conducted by Enzyme Mediated Immunoassay Technique (EMIT),[11] and positive results were confirmed by Mass Spectrometry/Gas Chromatography for cocaine metabolites and by High Pressure Liquid Chromatography[12] for marijuana metabolites. Use of these drugs was determined by both positive self-report or positive urine assay. A full description of the procedure for the urine assays has been published elsewhere.[13]

During the prenatal interview, respondents were also asked to report whether their partners used alcohol, marijuana or cocaine. Quantity measures of partner's drug use were also asked but proved to be difficult for the women to answer and were therefore not included in the analysis.

Life stress was measured by the Life Experiences Survey (LES) whose reliability and validity has been demonstrated.[14] For this analysis, life stress was measured by the Negative Life Events subscale of the LES, which is a summary scale of the number and negative effect of life events experienced in the past year.

Depressive symptoms were measured by the Center for Epidemiological Studies—Depression Scale (CES-D)[15] whose validity and reliability for the CES-D have been demonstrated and reported.[15–22] Past history of depression and suicide were measured by asking respondents, "In your lifetime, have you had two weeks or more during which you felt sad, blue, depressed, or when you lost all interest and pleasure in things that you usually cared about or enjoyed?" History of suicidal feelings and attempts were assessed by asking "Has there ever been a period of two weeks or more when you felt like you wanted to die?" and "Have you ever attempted suicide?"

Following delivery, trained record reviewers, without knowledge of the hypotheses of this analysis, abstracted medical records using a precoded protocol, which documented participants' reproductive and general medical health histories.

Statistical Analysis

Odds ratios and 95% confidence intervals (CI) were used to compare victims of physical or sexual violence during pregnancy to non-victims of violence on demographic, medical and psychosocial characteristics and on drug and alcohol use.[23] Least square multiple regression was used to determine the association between neonatal growth parameters and violence during pregnancy while controlling for potentially confounding variables.[24] Multiple logistic regression was used to assess the multivariable relationship between violence and drug use. Adjusted odds ratios and 95% CI were calculated from this model.[23]

Results

Sample Characteristics

Eighty-six percent of 1,932 eligible women participated in the study; 144 (8 percent) refused to participate and 124 (6 percent) who had agreed to participate left after the prenatal clinic visit prior to the interview. Non-participants were more likely than participants to be White (19 vs 9 percent), older (26 years vs 24 years), cigarette smokers (41 vs 34 percent) or third trimester registrants (40 vs 22 percent). The two groups were similar in parity, use of alcohol, marijuana, cocaine, or other drugs.

Seventy-eight percent (n = 1,303) of the women who were recruited completed the study. Of the 361 women who were lost to follow-up, most (81 percent) delivered in another hospital or had an elective abortion. Sixty (5 percent) of the 1,303 who completed the study were excluded from the analysis because of a missing medical record review or infant examination, leaving 1,243 completed cases. Completed cases (n = 1,243), when compared to those lost to follow-up or with incomplete infant data (n = 421), had similar sociodemographic characteristics and similar patterns of alcohol, marijuana, cocaine, and other drug use.

The sample of 1,243 participants with complete data reflects the low income (48 percent with household income ≤ $500 a month), predominantly ethnic minority (North American Black = 55 percent, foreign-born Black = 19 percent, Hispanic = 18 percent, White = 8 percent), single (62 percent), and primiparous (55 percent) population served at the prenatal clinic. While 16 percent of the participants were less than 18 years old, 66 percent were between 19–29 years of age.

Violence during Pregnancy

Seven percent (n = 92) of women in the study reported physical or sexual violence during pregnancy. An additional 3 percent of the study population (n = 37) reported incidents of violence in the three months prior to the pregnancy but not during their pregnancy. Less than one percent (n = 11) of women in the sample (12 percent of victims) reported incidents both three months before and during pregnancy.

Sixty percent of the victims were subjected to one incident of violence during pregnancy, 25 percent were victimized twice and 15 percent experienced three or more incidents. More incidents occurred in the first trimester (55 percent) than in the second (40 percent) or third trimesters (25 percent). Most of the women (94 percent) knew their assailant. Thirty-six percent of the victims saw a doctor for at least one of the violent incidents during pregnancy and 10 percent were hospitalized overnight as a result of one of these incidents.

Comparison of Victims and Non-Victims

Compared to non-victims (Table 1), victims had higher relative odds of identifying themselves as White (7 vs 15 percent), born in the US (65 vs 76 percent), and single (62 vs 75 percent). They were also more likely to be on Medicaid (60 vs 76 percent), to have had a history of sexually transmitted diseases (17 vs 30 percent) and to have had an elective

TABLE 1—Relative Odds of Selected Demographic and Medical Differences of Victims of Violence Compared to Non-victims (n = 1,243)

Variable/Comparison	Relative Odds	95% Confidence Interval
Ethnicity		
North American Black/Other Black	1.56	0.81, 3.00
North American Black/Hispanic	1.54	0.78, 3.03
North American Black/White	0.48	0.25, 0.92
North American Black/Other	0.75	0.37, 0.54
Marital Status		
Living with Partner/Married	1.86	0.80, 4.34
Single/Married	2.52	1.28, 4.97
Born in the US/Born outside	1.70	1.04, 2.79
Medicaid Recipient/Non-recipient	2.12	1.23, 3.66
History of Sexually Transmitted Disease*/None	2.13	1.33, 3.41
Prior Elective Abortion/None	1.68	1.01, 2.57

*Syphilis, gonorrhea, herpes, pelvic inflammatory disease, chlamydia

abortion (38 vs 50 percent). The two groups had similar levels of educational attainment (42 percent of victims had 12 years of school vs 39 percent of non-victims), mean ages ($x = 24$ for each group), employment status (24 percent not employed in each group) and parity (53 percent of victims were primiparas vs 52 percent of non-victims).

Victims of violence differed from non-victims on a number of psychosocial measures (see Table 2). Compared to non-victims, victims were at greater risk of having reported a history of one or more depressive episodes (33 vs 56 percent) and to have attempted suicide (5 vs 17 percent), reporting unhappy feelings about their current pregnancy (20 vs 33 percent), perceiving their partners (11 vs 22 percent) and families (14 vs 27 percent) as unhappy about their pregnancy, and feeling a lack of support during their pregnancy (13 vs 36 percent). Victims reported more depressive symptoms (mean difference = 5.6, 95% CI = 3.35, 7.86) and greater numbers of negative life events in the past year (mean difference = 13.16, 95% CI = 10.01, 16.31).

These demographic and psychosocial characteristics did not differ between women who experienced one compared to two or more incidents of abuse during pregnancy (data available on request to authors).

Drug Use among Victims and Non-victims

Women were divided into three categories (nonusers, light users, and heavy users) based on their frequency of use and the number and type of different drugs used during pregnancy. Since we have shown that among marijuana and cocaine users, a positive urine assay is associated with more frequent drug use,[13] we designated women who had a positive urine for either drug and those who self-reported using opiates and/or marijuana or cocaine at least weekly during pregnancy as heavy users. Users of any illicit drug who did not meet the heavy use criteria were categorized as light users.

Victims of violence during pregnancy were at greater risk than non-victims of being heavy users of alcohol (OR = 2.43, 95% CI = 1.71, 3.46) and illicit drugs (OR = 2.68, 95% CI = 1.72, 4.17). A comparison of frequency of use by victims and non-victims showed that victims were heavier substance users in all categories of use than non-victims (see Table 3).

Compared to non-victims, women who were victims of violence had greater odds of having a male partner who was a marijuana (OR = 2.27, 95% CI = 1.46, 3.53) and/or cocaine (OR = 2.35, 95% CI = 1.81, 4.48) user. Further, odds ratios demonstrate a two-fold increase in the use of two or more drugs (OR = 3.2, 95% CI = 1.92, 5.48) by partners of victims when compared to partners of non-victims. Alcohol use by partners was not an increased risk for victims (OR = 1.03, 95% CI = 0.63, 1.69).

Alcohol and drug use did not differ between women who experienced one versus two or more incidents of abuse during pregnancy (data available on request to authors).

A multiple logistic regression (Table 4) shows that the risk of being a victim of violence was associated with a woman's alcohol use during pregnancy and drug use by her partner even when controlling for race, age, marital status, education, and history of violence in the three months prior to pregnancy. While heavy drug use during pregnancy (use of opiates or weekly use of marijuana or cocaine or positive urine assay) was associated with a 39 percent increase in the odds of being a victim of violence, chance cannot be ruled out as the explanation for this finding. Violent incidents in the three months prior to pregnancy have strong predictors of violence during pregnancy.

Birth Outcomes

Multivariable regression analyses were conducted in

TABLE 2—Relative Odds of Selected Psychosocial Differences of Victims of Violence Compared to Non-victims (n = 1,243)

Variable/Comparison	Relative Odds	95% Confidence Interval
History of Depression		
Yes/No	2.60	1.69, 4.00
History of Suicide Attempts		
Yes/No	3.85	2.08, 7.14
Unhappy Feelings about Pregnancy		
Yes/No	1.91	1.21, 3.02
Partner's Unhappy Feelings about the Pregnancy		
Yes/No	2.40	1.37, 4.19
Family's Unhappy Feeling about the Pregnancy		
Yes/No	2.62	2.29, 5.73
Little or No Emotional Support During the Pregnancy		
Yes/No	2.30	1.37, 3.86

TABLE 3—Relative Odds of Frequency of Psychoactive Substance Use of Victims of Violence Compared to Non-victims (n = 1,243)

Variable/Comparison	Relative Odds	95% Confidence Interval
Maximum Average Daily Alcohol Use		
< 1 Drink/None	2.06	1.24, 3.44
1–2 Drinks/None	5.34	2.30, 12.40
2+ Drinks/None	5.15	2.14, 12.41
Frequency of Marijuana Use[a]		
< 1× a Month/None	1.98	0.81, 4.81
Monthly/None	2.13	0.93, 4.91
Weekly/None	2.56	1.53, 4.29
Frequency of cocaine use[1]		
< 1× a Month/None	1.67	0.70, 4.03
Monthly/None	2.69	0.90, 8.02
Weekly/None	2.91	1.56, 5.44

[a]Based on self-reported data only.

TABLE 4—Results of Multiple Logistic Regression Analysis on Experience of Violence During Pregnancy (n = 1,153)

Variables	Relative Odds	95% Confidence Interval
Average daily alcohol use (2 drinks/day vs none)	1.87	(1.24, 2.80)
Average daily alcohol use 1 drink/day vs none)	1.37	(1.12, 1.67)
Heavy illicit drug use	1.39	(0.79, 2.46)
Light illicit drug use	0.87	(0.36, 2.12)
Partner's alcohol use	1.41	(0.92, 2.43)
Partner's illicit drug use (2 or more vs none)	2.26	(1.19, 4.30)
Partner's illicit drug use (1 vs none)	1.51	(1.09, 2.07)
White vs US Black	1.95	(0.97, 3.93)
Foreign-born Black vs US Black	0.86	(0.41, 1.77)
Hispanic vs US Black	1.02	(0.49, 2.11)
Other race vs US Black	0.72	(0.20, 2.59)
Age (30 years vs 18 years)	0.90	(0.51, 1.60)
Marital status (married vs single)	0.60	(0.29, 1.23)
High school graduate vs not	1.12	(0.67, 1.85)
Violent incidents—3 months prior to pregnancy	5.87	(2.73,12.60)

order to investigate the relationship between violence during pregnancy and birthweight, infant length, head circumference, and gestational age. Each regression model included ethnicity, age, pre-pregnancy weight, weight gain during pregnancy, parity, number of prenatal visits, history of sexually transmitted diseases, marijuana use, cocaine use, alcohol use, opiate use, and cigarette use. Once confounders were controlled, the association of violence to birth outcomes, while generally in the expected direction, was weak: birthweight: −19 g, (95% CI = −115, 78); length: 0.15 cm, (95% CI = −0.68, 0.38); head circumference = −0.14 cm, (95% CI = −0.48, 0.19); gestational age: 0.13 weeks, 95% CI = −0.29 to 0.55).

Discussion

The findings of this study support previous reports on the prevalence of violence during pregnancy.[6,7] Of greater importance are the results which provide evidence of a strong relation between violence during pregnancy and the use of alcohol by the pregnant woman and the use of illicit drugs by her male partner.

A number of previous studies have shown that abused women are at greater risk of using heavier quantities of alcohol and prescription and non-prescription drugs than women who are not abused.[4,7] While the cause and effect relationship between use of alcohol and experience of violence is unclear, there are indications that women who are abused may self-medicate with alcohol, illicit drugs, and prescription medication in order to cope with the violence.[4,5,25] For example, in a review of medical records, Stark, *et al*,[4] determined that increased alcohol and drug use followed the first incident of abuse.

The relation between the male partner's drug use and violence during pregnancy has not received as much attention. In general, among men who batter their female partners, substance use has been found to frequently accompany battering.[7,26–28] One author[29] has suggested that drinking is used by men who batter to disavow their violent behavior and disclaim responsibility for their actions. Findings from the present study indicate that multiple drug use among male partners is independently associated with over a two-fold increase in women's experience of violence during pregnancy. Since this study did not directly investigate whether the assailant was the male partner, it is not possible to identify the perpetrator of violence as the male partner. Anecdotal evidence from the interviews suggests that a large proportion of the perpetrators were male partners, however. Further research is needed to determine whether the use of illicit drugs actually promotes violent behavior, is employed as a culturally sanctioned justification for engaging in violence without taking responsibility for it, or is associated through some third causal factor to violence.

The results of this study indicate that once sociodemographic factors and drug use are taken into account, the experience of violence during pregnancy is weakly, if at all, associated with newborn size or gestational age. It is possible that infrequent but severe injuries are associated with adverse pregnancy outcomes. However, the small number of women reporting such injuries did not allow us to assess this possibility.

A number of important limitations should be considered when interpreting the results of this study. First, the subjects were primarily poor, Black, and Hispanic women living in the inner-city. The prevalence of violence and drug use in other populations may be different. Second, our assessment of abuse was limited because it did not evaluate the full psychological impact of an abusive relationship that typically involves a climate of threat and fear. Third, the assessment of violence through limited questions about current experiences of violence does not allow for an understanding of the causal ordering of the relationship between violence, drug use, and depression. Finally, validity of the assessment of the male partner's drug use may be reduced since it was conducted through the female participant, rather than through direct self-report and/or urine assay from the male partner.

With these limitations in mind, prenatal health care providers should assess women's exposure to violence prior to and during pregnancy. In addition, assessment of psychoactive drug use among women and their partners is critical, since such behaviors may be employed as a marker for risk for violence during pregnancy. Further, the possibility of continued violence against a woman after delivery and the potential extension of this violence to the child have important public health implications. Since violence and drug use reflect a wider set of environmental conditions and psychosocial factors that create a context that is detrimental to a mother's and infant's physical and mental health, prevention and intervention efforts will need to be set within a framework of services to address these needs. Training of health care professionals about the problems of drug abuse and family violence is a critical and necessary first step in early identification and intervention efforts.

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NCI Announces Research Opportunities in Cancer Prevention

The Division of Cancer Prevention and Control (DCPC) of the National Cancer Institute has announced it is accepting applications for the next Cancer Prevention Fellowship Program. Funding permitting, as many as 10 fellows will be accepted for up to three years of training, beginning July 1, 1991. The *deadline* for applications is September 1, 1990.

The purpose of this program is to attract individuals from a multiplicity of health science disciplines into the field of cancer prevention and control. The program provides for:

- Participation in the DCPC Cancer Prevention and Control Academic Course;
- Working at NCI directly with individual preceptors on cancer prevention and control projects;
- Field assignments in cancer prevention and control programs at other institutions.

Benefits include selected relocation and travel expenses, paid federal holidays, and participatory health insurance. Those eligible for the program include those with MD or DDS degree from a US, territorial, or Canadian medical school. Foreign medical graduates must have current ECFMG/FMGEMS certification and appropriate experience (e.g., one year residency) in a training program approved by the Accreditation Council for Graduate Medical Education. Also eligible are those with PhD, DrPH, or other doctoral degree in a related discipline (epidemiology, biostatistics, and the biomedical, nutritional, public health or behavioral sciences). Foreign education must be comparable to that received in accredited US, territorial, or Canadian institutions. Applicants must be US citizens or resident aliens eligible for citizenship within four years.

For more details and application catalog, send a postcard or letter with your name and home address to: Douglas L. Weed, MD, MPH, PhD, Director, CPFP, DCPC, National Cancer Institute, Executive Plaza South, T-41, Bethesda, MD 20892. Further inquiries should be directed to Barbara Redding at NCI; (301) 496-8640 or -8641.

Risk Factors for Depressive Symptomatology in a Drug Using Population

JOHN C. BUCKNER, PHD, AND WALLACE MANDELL, PHD, MPH

Abstract: This study employs a prospective design to examine possible personality, drug use, stressful life event, and social support-related variables associated with the onset of a depressive episode in a cohort of psychoactive drug using young adults. Two waves of data, collected one year apart, were available on 942 individuals. Cases (n = 62) were free of depressive symptoms at time 1 but reported significant symptomatology at time 2 as measured by the depression subscale of the Brief Symptom Inventory. Controls (n = 490) were those free of depressive symptoms at both time points. In multivariate analyses, users of the central nervous system depressant methaqualone had a nearly four-fold elevated risk for depressed mood as compared to nonusers. Additional risk factors significant after multivariate adjustment included lower self-esteem at time 1 and negative life events. These results highlight the multifactorial nature of depressive symptomatology. (*Am J Public Health* 1990; 80:580–585.)

Introduction

Using predominantly case-control comparisons on general population samples, the association between negative life events,[1–9] lack of social support,[10,11,4,5,8,12] and self-esteem[13–18] with depressive symptoms and disorder has been well documented. Analogous prospective studies are rare.[17] While research has documented an association between alcohol use and depression,[19–21] much less is known about the potential effects of other drugs or about the relative and possibly interactive effects of life events and drug use on depression symptomatology. One obstacle to such studies is the low base rate of negative life events and/or heavy substance use in most samples making it unfeasible to examine their relationships. By focusing on a population of young adults who frequently use a variety of psychoactive substances, we estimated the adjusted relative risk of various drug use, personality, psychosocial, and negative life event variables for developing an episode of depressed mood.

Methods

We utilized a case-control methodology nested within a prospective study[22,23] to investigate risk factors for depressed mood using data from a study on the consequences of arrest for marijuana possession, a short-term longitudinal investigation of drug using young adults.*

Study Population

In 1981–82, 1,134 adults living in the State of Maryland were interviewed and 942 were relocated and agreed to a reinterview one year later. A primary aim was to evaluate the effects of having been arrested and tried for marijuana possession on subsequent drug use and mental health. An application for a certificate of confidentiality, which protects the identity of research study subjects, was approved by the US Secretary of Health and Human Services. Written consent was obtained of all research participants and each individual was informed as to the nature of the study and the certificate which protects the information they provide from seizure by police or use in any civil or criminal proceeding.

The panel of 942 individuals which forms the basis of the present study was comprised, in part, of a random sample of 621 individuals, stratified by month of arrest, from a roster of all adults who had been tried in 1981 for marijuana possession within the district courts of Maryland. An additional 321 non-arrested individuals, nominated by their arrested counterparts as being friends or acquaintances, were selected as a neighborhood-based comparison group. This nomination procedure was done to ensure comparability between the two groups on demographic characteristics and drug use patterns.

Operationalization of Variables

The presence of depressive symptomatology in the week prior to interview was assessed using the depression subscale of the Brief Symptom Inventory (BSI)—the short form of the Symptom Checklist-90 (SCL-90).[24,25] Internal consistency analysis of the depression sub-scale revealed coefficient alpha = .78 at the time of first interview (Time 1) and alpha = .80 at the second interview (Time 2). The depression subscale, consisting of six items, has been found to correlate (.95) with its corresponding longer version on the SCL-90.[25] There is evidence in support of the temporal stability and the convergent and discriminant validity of the BSI depression subscale.[26]

Individuals were classified as a case if they did not report depressive symptoms at Time 1 but reported symptoms at Time 2 that placed them in the 90th centile or above on the non-patient norms for the BSI depression subscale.[26] This cut-point was selected to serve as a strong contrast between cases and controls while still retaining a sufficient sample size for purposes of statistical power. While this depression measure does not identify a case of dysthymia or major depressive disorder according to DSM-III-R[27] criteria, an individual defined as a case in this study would present clinically with at least moderate symptoms of depression. All individuals who reported no or minimal symptoms of depression at both time points were selected to form a control group. Using these criteria a total of 62 cases and 490 controls were selected. Individuals excluded from analyses were those who reported mild-to-severe depressive symptoms at Time 1. Inspection of the depression scores at both time points revealed that those selected as cases went, on average, from the 50th percentile at Time 1 to the 95th percentile at Time 2. In contrast, those selected as controls were at about the 45th percentile at Time 1 and remained there at Time 2.

The wording of the BSI allowed for a determination of the current level of depressed mood experienced by a study participant, although, it could not help determine depressed

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mood in the months prior to each administration. While an imperfect and somewhat less specific measure for determining case status than use of an instrument such as the Diagnostic Interview Schedule,[28] it is unlikely that a bias was produced by this manner of case-control selection. Because the BSI was administered at Time 1, some control for prior depressed mood was achieved; more than is the case in many other case-control comparisons.

Participants were asked about frequency of use of a wide variety of licit and illicit drugs in the two months prior to each interview. Alcohol and marijuana were used with enough regularity to justify coding on an interval level (3 = daily; 2 = two–three times a week; 1 = one time a week or less; 0 = no use). Tobacco use was measured in packs of cigarettes smoked per day. The vast majority of use of such substances as amphetamines, barbiturates, methaqualone (Quaalude, Sopor), cocaine, heroin, hallucinogens, and anxiolytics (e.g., Valium, Librium) occurred on a weekly or less frequent basis. Therefore, these drug variables were dichotomously coded with 1 representing any reported use of the drug during the two months prior to interview and 0 indicating no use.

The occurrence of any of five negative stressful life events (arrest, conviction, hospitalization, traffic accident, fired from job) in the 12 months between interviews was assessed and a summative count score was created. Prior research has shown that weighting of events before creation of an overall score does not increase prediction of the dependent variable.[29-32] These negative life events are typical of those found on life event inventories[33] and represent life stressors that occur with relative frequency in this population.

Separate questions tapping perceived social support, or social embededness,[34] with family members and with peer group were asked of all respondents at both interviews: "Do you feel a sense of solidarity, closeness, belonging to your family?" Four dichotomous variables were created from the Time 1 and Time 2 questions representing perceived social embededness with family or friends or lack of support/embededness. The presence of a spouse or living together with a partner was also determined and dichotomously coded as another important indicant of social support.[32,35]

Finally, the Rosenberg Self-Esteem Scale,[36] a widely used 10-item measure of self-esteem, was administered at both interviews. Extensive research has demonstrated the predictive and construct validity of this instrument.[37] With the present sample, the instrument displayed good internal consistency (Coefficient alpha = .75) and the one year test-retest stability was r(938) = .49.

Univariate logistic regression models that estimate the crude relative risk ratio for the development of an episode of depression were first calculated for variables from each of the demographic, drug use, life events, social support, and personality domains. Using multivariate logistic regression techniques, variables predictive of case status in univariate analyses were simultaneously evaluated to estimate adjusted relative risks. The presence of multiplicative interaction was examined for pairwise combinations of variables that remained significant in the multivariate analysis.

Results

Descriptive statistics for the 62 depressed mood cases and 490 controls and crude relative risk estimates based on univariable logistic regression analyses are presented in Table 1 for categorical variables and Table 2 for variables measured on a continuous scale. Cases were fairly similar to controls with respect to age, race, sex, marital status, being employed or in school, recent history of marijuana arrest, and socioeconomic status (not shown in tables). Univariate relative risk (RR) estimates for the development of depressive symptomatology were found for any use during the two months prior to the second interview for methaqualone, anxiolytics, and less strongly for heroin, tobacco, and hallucinogens.

TABLE 1—Estimated Relative Risk for an Episode of Depressed Mood Based on Univariate Logistic Regression Analysis with Categorical Variables

Variables	Cases No. (%) (N = 62)	Controls No. (%) (N = 490)	Referent Category	Relative Risk Estimate (unadjusted)	(95%CI)	p value
Sociodemographic						
Male	52 (84)	382 (78)	female	1.47	(.72, 2.99)	.29
White	24 (39)	166 (34)	black	1.23	(.72, 2.12)	.45
Employed or in school	40 (65)	322 (66)	no	.94	(.54, 1.64)	.84
Recent marijuana arrest	45 (73)	321 (66)	no	1.39	(.77, 2.51)	.27
Drug Use						
Amphetamines (Time 1)	7 (11)	49 (10)	no use	1.15	(.49, 2.65)	.75
Amphetamines (Time 2)	9 (15)	52 (11)	no use	1.43	(.67, 3.06)	.36
Barbiturates (Time 1)	5 (8)	32 (7)	no use	1.26	(.47, 3.35)	.65
Barbiturates (Time 2)	6 (10)	28 (6)	no use	1.77	(.70, 4.45)	.3
Methaqualone (Time 1)	4 (6)	31 (6)	no use	1.02	(.35, 2.99)	.97
Methaqualone (Time 2)	10 (16)	23 (5)	no use	3.90	(1.76, 8.65)	.0008
Anxiolytics (Time 1)	3 (5)	29 (6)	no use	.81	(.24, 2.74)	.73
Anxiolytics (Time 2)	8 (14)	26 (5)	no use	2.64	(1.14, 6.12)	.02
Cocaine (Time 1)	16 (26)	96 (20)	no use	1.43	(.77, 2.63)	.25
Cocaine (Time 2)	14 (23)	99 (20)	no use	1.15	(.61, 2.17)	.66
Hallucinogens (Time 1)	6 (10)	26 (5)	no use	1.91	(.75, 4.84)	.17
Hallucinogens (Time 2)	7 (11)	25 (5)	no use	2.37	(.98, 5.73)	.06
Heroin (Time 1)	4 (6)	16 (3)	no use	2.04	(.66, 6.32)	.21
Heroin (Time 2)	7 (11)	24 (5)	no use	2.47	(1.02, 6.00)	.046
Social Support (Time 2)						
Family Embededness (yes)	53 (85)	453 (93)	no	.41	(.19, .91)	.03
Peer Group Embededness (yes)	42 (68)	400 (82)	no	.47	(.27, .85)	.01
Married or Living Together	10 (16)	120 (24)	no	.59	(.29, 1.20)	.15

TABLE 2—Estimated Relative Risk for an Episode of Depressed Mood Based on Univariate Logistic Regression Analyses with Continuous-scale Variables

Variables	Cases (N = 62) Mean (S.D.)	Controls (N = 490) Mean (S.D.)	Relative Risk Estimate (unadjusted)	(95%CI)	p value
Sociodemographic					
Age	24.50 (6.21)	24.92 (6.88)	.99	(.96, 1.04)	.92
Drug Use					
Alcohol (Time 1)*	1.51 (1.01)	1.37 (.96)	1.16	(.88, 1.53)	.28
Alcohol (Time 2)*	1.62 (1.02)	1.43 (.97)	1.22	(.93, 1.61)	.15
Marijuana (Time 1)*	1.85 (1.10)	1.77 (1.12)	1.04	(.84, 1.30)	.72
Marijuana (Time 2)*	1.81 (1.25)	1.56 (1.15)	1.20	(.95, 1.51)	.12
Tobacco (Time 2)**	3.89 (2.07)	3.35 (1.86)	1.17	(1.01, 1.35)	.04
Personality (coded 1–4)					
Self-esteem (Time 1)	3.24 (.49)	3.45 (.40)	.32	(.17, .59)	.0003
Self-esteem (Time 2)	3.08 (.45)	3.46 (.42)	.14	(.07, .26)	<.0001
Life Events: (count 0–4)					
Negative Events	.79 (.91)	.44 (.72)	1.67	(1.24, 2.26)	.0009

*These frequency codes range from 0–3: (0 = no use; 1 = 1 time a week or less; 2 = 2–3 times a week; 3 = daily use).
**These frequency codes range from 1–7: (1 = no use; 2 = >1 cigarette a day, 3 = 1–5 cigarettes a day; 4 = 10 cigarettes a day; 5 = 20 cigarettes a day; 6 = 30 cigarettes a day; 7 = 40 cigarettes a day).

TABLE 3—Estimated Relative Risk for an Episode of Depressed Mood Based on Multivariate Logistic Regression Model with Drug Variables Only

Variable	Referent Category	Estimated Relative Risk	(95%CI)	p value
Drug Use (Time 2)				
Amphetamines	no use	.81	(.29, 2.30)	.69
Barbiturates	no use	.54	(.14, 7.08)	.37
Methaqualone	no use	5.27	(1.86, 14.96)	.002
Anxiolytics	no use	2.07	(.75, 5.71)	.16
Cocaine	no use	.53	(.24, 1.19)	.13
Hallucinogens	no use	2.14	(.77, 6.00)	.15
Heroin	no use	3.32	(1.18, 9.30)	.02
Alcohol frequency	NA*	1.06	(.79, 1.43)	.67
Marijuana frequency	NA*	1.07	(.82, 1.40)	.59
Tobacco frequency	NA**	1.08	(.93, 1.25)	.34

*Not applicable. These frequency codes range from 0–3: (0 = no use; 1 = 1 time a week or less; 2 = 2–3 times a week; 3 = daily use).
**Not applicable. These frequency codes range from 1–7: (1 = no use; 2 = >1 cigarette a day; 3 = 1–5 cigarettes a day; 4 = 10 cigarettes a day; 5 = 20 cigarettes a day; 6 = 30 cigarettes a day; 7 = 40 cigarettes a day).

To statistically control for multiple drug use, a multivariate logistic regression model containing main effect terms for all Time 2 drug variables (see Table 3) was created. The relative risk estimates show a clear excess risk for those reporting any use of methaqualone in the prior two months (RR = 5.27; 95% CI = 1.86, 14.96) as well as those reporting any use of heroin (RR = 3.32; 95% CI = 1.18, 9.30). Two-way interaction terms were then evaluated for methaqualone and heroin with combinations of the other drug variables. None were found to be statistically significant at ($p < .05$) although the interaction term for methaqualone and barbiturate use was significant at ($p < .10$) and was further evaluated in subsequent analyses.

Crude relative risk estimates for the life event, social support, and self-esteem measures are apparent in Tables 1 and 2. Higher levels of self-esteem at Time 1 appear to act as a protective factor with the risk of depressed mood dropping by an estimated .32 (95% CI = .17, .59) with each unit increase in self-esteem. Conversely, individuals with lower levels of self-esteem appear more vulnerable to the development of an episode of depressed mood. A strong cross-sectional relationship between Time 2 self-esteem and depression was also present. Negative life events happening in the year before follow-up served to elevate the risk for depression with each occurrence of such an event (arrest, conviction, fired from job, or hospitalization) increasing the risk for developing depressed mood 1.67-fold (95% CI = 1.24, 2.26). Risk for depressed mood was less if the subjects reported that they felt embedded within their family (RR = .41; 95% CI = .19, .91) or within a peer group (RR = .47; 95% CI = .27, .85). Being married or living with someone was not associated with case-status.

In a multivariate logistic regression model (see Table 4) containing main effect terms for life events, social support and self-esteem, the relative risk estimate for family as well as for peer group embededness were sharply reduced suggesting that the univariate relationships were due to associations with other variables. Self-esteem at the earlier period and the occurrence of negative events during the year remained strong predictors of case status. All possible two-way interactions among statistically significant variables in this model were tested but no interaction was detected.

We then tested a multivariable function (table not shown) that combined the drug use and psychosocial varia-

TABLE 4—Estimated Relative Risk for an Episode of Depressed Mood based on Multivariate Logistic Regression Model with Social Support, Self-esteem, and Negative Event Variables Only

Variable	Referent Category	Estimated Relative Risk	(95%CI)	p value
Family embededness (yes)	no	.50	(.21, 1.16)	.11
Peer group embededness (yes)	no	.58	(.35, 1.22)	.18
Self-esteem (Time 1)	NA*	.39	(.21, .75)	.005
Negative events	NA**	1.60	(1.15, 2.15)	.005

*Not applicable. This variable is measured on a continuous scale.
**Not applicable. This variable ranges from 0–4 and was not categorized.

bles that had retained p values of .05 or less in the prior multivariate models as well as the interaction term for methaqualone and barbiturate use. The prior association between heroin use and depressed mood no longer held in this model nor did the methaqualone-barbiturate interaction term. Negative events, self-esteem, and methaqualone use maintained their independent effects. A final model was tested that retained these three main effect terms and produced the following estimates: negative events (RR = 1.45; 95% CI = 1.06, 2.00); self-esteem (RR = .32; 95% CI = .17, .59); and methaqualone use (RR = 3.70; 95% CI = 1.59, 8.63). Interaction was not detected among pairwise combination of these three variables.

Discussion

Prior research evidence has suggested associations between self-esteem, the occurrence of negative life events, and indices of social support with depressed symptomatology. With the exception of social support, which only evidenced a trend in the predicted direction in multivariate analysis, this was corroborated in our findings; and, importantly, our results indicate that, when controlling for each other, these variables remain independent predictors for who will develop a new episode of depression. It is conceivable that a more refined measure of social support than the ones used in this study might have detected a support-depression association in multivariate, not just univariate, analyses. The measure of life events was limited by the available data to a count of five negative occurrences (arrest, conviction, hospitalization, traffic accident, fired from job). A more exhaustive index of life events would likely have produced an even stronger association.

This study has documented that a similar pattern of risk factor relationships for depressed affect exists among a sample of psychoactive drug users as has typically been found in general population samples.[1–9,15,17] The final model suggests that individuals who have low self-esteem (measured a year prior), or who experience negative life events, or who use methaqualone, are at elevated risk for developing depressed mood. An issue to consider was the effectiveness of the nested case-control design in controlling for Time 1 depressed mood. We examined this by re-analyzing all models reported in Tables 1–4 and the final model. While the Time 1 measure of depressed mood was a strong predictor of the same variable at Time 2, the same findings emerged from the reanalysis with only slight and trivial changes in the relative risk estimates. Thus, sufficient control for prior depressed mood appears to have been achieved in the design alone.

In this sample, methaqualone use appears to substantially increase the risk for depressed symptoms independently of self-esteem or negative life events. The majority of methaqualone users who became depressed at Time 2 may have initiated their use of this substance during the study period: Of the 10 individuals with depressed symptoms who reported use of methaqualone at Time 2, seven (70 percent) had not reported use at Time 1 while three (30 percent) reported use at both time points. This methaqualone-depression association cannot be explained by the potential confounding effects of other drug use assessed in this study or other predictive variables. In fact, the adjusted relative risk estimates for methaqualone use were higher than the crude estimate. Nevertheless, the existence of a third variable which causes both a propensity to use methaqualone and to become depressed cannot be ruled out. When examined with other drug variables, heroin use was also related to depression. However, when considered together with psychosocial variables, heroin use failed to retain its importance in predicting an episode of depression.

Our review of the literature indicates that the association found between methaqualone use and depressed mood in this drug using sample is a new finding and, therefore, deserves further comment. Methaqualone is a synthetic central nervous system depressant compound that is similar to barbiturates in its sedative and hypnotic action.[38,39] Until January 1984, when it was made a controlled substance,[40] the drug was marketed in the United States under the trade names Quaalude, Sopor, Parest, Optimil, and Somnafac.[41] Its use reached epidemic proportions in the 1970s and early 1980s [42–46]; perhaps, due in part to its purported aphrodisiac qualities.[47] Although its use today has diminished as a result of discontinuation of legal production in the United States,[48] quantities produced by clandestine domestic or small foreign laboratories are still available.[47,48]

Methaqualone users are reported to be more likely to also use other illicit and licit psychoactive substances than are non-users of the drug.[44,49] This was confirmed in the present sample. The multi-drug involvement of methaqualone users could account for this study's finding of univariable associations between depressed mood and various other substances (marijuana, tobacco, hallucinogens) but lack of association when controlling for methaqualone use. Our results suggest the possible prudence of adjusting for the potential influence of methaqualone use when examining the relation between other drugs and depression.

A relation between methaqualone use and depressed symptomatology has been hinted at in two prior studies. Schwirian and colleagues,[49] in a study of personality characteristics of methaqualone users, noted a cross-sectional association between duration of methaqualone use and mea-

sures of "lethargy and introspectiveness." Craig and Van Natta[50] looked at cross-sectional, unadjusted associations between recent use of a variety of prescription medicines and the Center for Epidemiologic Studies' depression (CES-D) scale. Users of anxiolytics and sedatives (which included but were not limited to methaqualone) had significantly greater proportions of high CES-D scores than did non-users of either class of drugs with stronger associations for females than for males. Barbiturates, a class of drugs with a similar sedative-hypnotic action to methaqualone, have also been linked to depression. Recent case reports indicate that phenobarbital may cause severe depression in patients taking the drug to control seizure disorders.[51,52] At a more general level, Judd and Grant[53] found that central nervous system depressant users, especially sedative users, displayed pronounced neuropsychological deficits on cognitive tasks which assessed abstract thinking, nonverbal learning, eye-hand coordination, and perceptual accuracy.

The temporal ordering of methaqualone use and depressed mood is important in terms of etiology. In a survey of college student methaqualone users that is relevant to this issue, Kochansky and colleagues[44] report that while no particular emotion encouraged the use of methaqualone, "moods named as clearly aversive to use were depression and anger." This study suggests that use of methaqualone is more likely to be an antecedent of depressed mood than a consequence. Until more conclusive research is conducted, it would be premature to infer that methaqualone use causally contributes to the development of depressed mood. Also, it should be noted that methaqualone is an infrequently used drug. Therefore, despite the almost four-fold relative risk for depressed mood, its attributable risk both in the general population and among drug user samples would be less than that of other explanatory variables including other potential etiologic factors examined in this study.

In this sample, lower self-esteem was associated with the development of a depressive episode one year later regardless of drug use behavior or the occurrence of negative life events. Likewise, the occurrence of negative life events also independently predicted depressed mood. Such findings may suggest alternative pathways to depressed mood in drug users, with those individuals low in self-esteem, or who use methaqualone, or who experience one or more stressful life events at higher risk. A combination of these risk factors would appear to increase risk in a multiplicative manner. Each one of these three variable's ability to predict subsequent depressed affect does not depend on a particular level of the other two; that is, effect modification was not detected.

A study limitation was reliance on a measure of depressed symptomatology as opposed to a diagnostic classification for depressive disorder. Nevertheless, a strong consistency exists between psychosocial risk factors for depressed symptoms and unipolar depressive disorder indicating a possible continuity between these two conditions.[54]

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European Charter on Environment and Health

Following 12 months of intense diplomatic activity and detailed technical consultations among the 32 countries of the European Region of the World Health Organization, a ministerial conference was held in Frankfurt on December 7–8, 1989, which formally approved a Charter on Environment and Health. The European Region of WHO represents 825 million people and covers an area from Greenland in the west to the Pacific coast of the Soviet Union in the east. The conference was attended by Ministers of both Health and Environment; observers were present from Canada and the United States.

Efforts are underway to secure adoption of the Charter by all sections of society in the countries of the WHO European Region.

The decision to develop the European Charter was based on a growing perception that links between the environment and health were not clearly perceived at international, national, and local levels and that insufficient attention had been given to the integration of health dimensions into policy decisions in such fields as physical planning, energy, transport, and agriculture, nor were potential health consequences always adequately addressed at the planning stage of new developments.

The conference not only addressed specific biological, chemical and physical hazards but placed considerable emphasis on the importance of a salubrious overall environment in terms of housing, the community, and the workplace. In brief, the Charter:

- Acknowledges the dependence of human health on a wide range of environmental factors;
- Devotes a section to entitlements and responsibilities, i.e., of individuals as well as public and private bodies;
- Encompasses principles for public policy—including new policies, technologies and developments—that safeguard the environment and human health;
- Enumerates strategic elements essential to its objectives, i.e., control measures, standards, information systems, and the appropriate use of fiscal/administrative/economic instruments and land-use planning;
- Delineates priority areas to which governments, the European community, and intergovernmental organizations should pay particular attention;
- Lays down steps necessary to reverse negative trends and maintain and increase improvements already taking place, in a section titled "The Way Forward."

A copy of the Charter, as well as a detailed commentary and other relevant materials, may be obtained from the WHO Regional Office of Europe, 8 Sherfigsvej, 2100 Copenhagen, Denmark.

HIV Antibody Testing among Adults in the United States: Data from 1988 NHIS

ANN M. HARDY, DRPH, AND DEBORAH A. DAWSON, PHD

Abstract: Data collected from 21,168 adults using the 1988 AIDS supplement to the National Health Interview Survey were examined to determine awareness of and experience with HIV antibody testing in the United States. Three-fourths of adults knew of the blood test for HIV antibodies; awareness was lower among Blacks, Hispanics, older adults, and those less educated. Overall, 17 percent of adults had been tested; of these, 73 percent because of blood donation, 14 percent through other non-voluntary programs (such as military induction), and 16 percent sought testing voluntarily. While a smaller proportion of Black and Hispanic adults had been tested, they were more likely than their White non-Hispanic counterparts to have been tested voluntarily. Persons who reported belonging to groups with high-risk behaviors were also more likely to have been voluntarily tested. Most of those tested voluntarily received their test results, but only one-third also received prevention information. Three percent of adults plan to be tested voluntarily in the next year; about half will seek testing through their doctor or health maintenance organization. (*Am J Public Health* 1990; 80:586–589.)

Introduction

Counseling and testing for antibodies to human immunodeficiency virus (HIV) are important parts of the US Public Health Service plan for the prevention and control of HIV infection and AIDS (acquired immunodeficiency syndrome).[1] Throughout the United States, confidential and/or anonymous counseling and testing sites have been established to provide these services. Between 1985 and September 1988, over 1.4 million persons were counseled and tested in publicly funded test sites.[2] HIV antibody testing may also be obtained at a variety of other health care settings, but the number tested in these settings is not known. In some instances, such as blood donation and physical examination for military induction, individuals are routinely screened for HIV antibodies.

It is important to have information on the number of persons tested under all the above mentioned circumstances to help evaluate the extent to which these particular prevention strategies have been implemented. To address this need, data were examined on the adult population's experience with the HIV antibody test from the National Health Interview Survey of AIDS Knowledge and Attitudes.

Methods

The National Health Interview Survey (NHIS) is a cross-sectional survey of the US civilian, non-institutionalized population, conducted annually since 1957.[3] Each year, members of 40–50,000 households across the country are interviewed in person by Census Bureau personnel. In addition to basic health and demographic information which is collected every year, supplemental surveys on special health topics which change periodically are also included.

Since August 1987, the NHIS has included a set of questions on AIDS. Data collection with the version of the AIDS survey analyzed in this report began in May 1988 and continued throughout 1988 and 1989. The questionnaire, administered to one randomly selected adult 18 years or older in each family included in the NHIS sample, was designed primarily to estimate adults' knowledge about AIDS, HIV transmission, and prevention of HIV infection. The information obtained has been summarized in previous reports.[4–10] Questions on knowledge about and use of the HIV antibody test are also included in the survey[4–9] and form the basis of this report.

The results are based on interviews conducted from May through October 1988 with 21,168 adults or about 90 percent of eligible respondents. The data were weighted to provide national estimates. These estimates are provisional because the 1988 AIDS records were processed before the other NHIS records and have not yet been merged with them. However, the estimates should differ only slightly from those which will be made using all of the 1988 data. Provisional standard errors for these estimates were calculated by applying a design effect of 1.3 to the standard errors obtained by assuming a simple random sample. These provisional estimates of variance were used to determine the significance of differences observed between proportions; all the differences noted are significant at the 0.05 level.

For analytic purposes, persons tested for HIV antibodies included those who reported they had had their blood tested and those who did not report being tested but who had donated blood since March 1985, when routine screening for HIV antibodies in donated blood began. Information on HIV antibody testing was examined in various demographic subgroups. In addition, two other groups were examined based on information obtained in the survey. The first were persons who reported belonging to one or more of the groups with behaviors associated with an increased risk for acquiring HIV infection (such as men who have had sex with other men or persons who have used illegal drugs by needle). This question is phrased similarly to that used by the Red Cross to screen potential blood donors. Respondents were given a list of statements about risk behaviors and were asked only to indicate if at least one was true for them; they did not indicate which were true. Two percent of respondents reported that at least one of these statements was true. The second group examined included persons who felt they had a high or medium chance of having or getting HIV infection; 3.5 percent of adults fell into this category.

Results

Overall, 75 percent of adults in the US had heard of the HIV antibody test (Figure 1). The percent who were aware of the test varied by age, race/ethnicity, and education, with lower levels of awareness noted among those over 50 years

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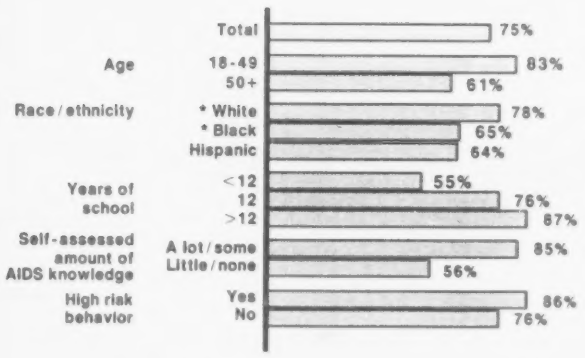


* Non-Hispanic ethnicity
SOURCE: NCHS, National Health Interview Survey, 1988

FIGURE 1—Percent of Adults Who are Aware of the HIV Antibody Test, by Demographic Characteristics

of age, among Black and Hispanic adults, and among those with less than 12 years of education. People who were more knowledgeable about AIDS and those who reported belonging to one or more of the high-risk behavior groups were more likely to know about the test.

Seventeen percent of adults had been tested for antibodies to HIV (Table 1); the majority because of blood donation since March of 1985. Men were more likely to have been tested than women, Black and Hispanic adults were slightly less likely than White non-Hispanic adults to have been tested. However, Black and Hispanic persons who were tested were more likely to have voluntary testing and testing by other non-voluntary means than their White non-Hispanic counterparts. The group with the highest proportion tested

TABLE 1—Percent of Adults Undergoing HIV Antibody Testing and Distribution of Those Tested by Circumstance

Group	Percent Tested	Circumstance of Testing for Those Tested*		
		Blood Donation	Voluntary	Other non-voluntary
		Percent		
Total	17	73	16	14
Sex				
Male	20	72	15	16
Female	13	74	17	10
Race/Ethnicity				
White, non-Hispanic	17	76	14	12
Black, non-Hispanic	14	55	26	23
Hispanic	14	58	22	23
Age (years)				
18–29	24	70	17	17
30–49	20	74	16	12
Over 49	7	75	12	10
Region				
West	17	66	21	15
All others**	16	74	14	14
High-risk Behavior				
Yes	35	40	46	14
No	16	74	14	14
Chance of HIV Infection***				
High/medium	24	51	27	23
Low/none	17	74	15	13

*Multiple responses may sum to more than 100%.
**No differences between other regions (Northeast, South, Midwest).
***Chance of having or getting HIV infection.

(35 percent) were those who reported high-risk behaviors for AIDS/HIV and voluntary testing was the most common circumstance for this group.

The characteristics of those tested voluntarily, those tested by all other means (including blood donation), and those not tested are summarized in Table 2. Persons who were tested, regardless of the circumstance were more likely to be younger and better educated than those not tested. Persons tested voluntarily were more likely to be Black and to report high-risk behaviors and a greater chance of HIV infection than those tested under other circumstances and those not tested.

Most (73 percent) of those tested voluntarily were tested only once and 82 percent reported receiving results of their testing. However, of those who got their results, only 32 percent also reported receiving information on how to avoid getting or transmitting HIV.

Six percent of adults planned to be tested in the next year; 45 percent of these had not been tested previously. About half of those with such plans indicated that they would seek testing voluntarily. Figure 2 shows where those with plans for voluntary testing will reportedly go to be tested. Over half (52 percent) will get tested through a doctor or at a health maintenance organization (HMO).

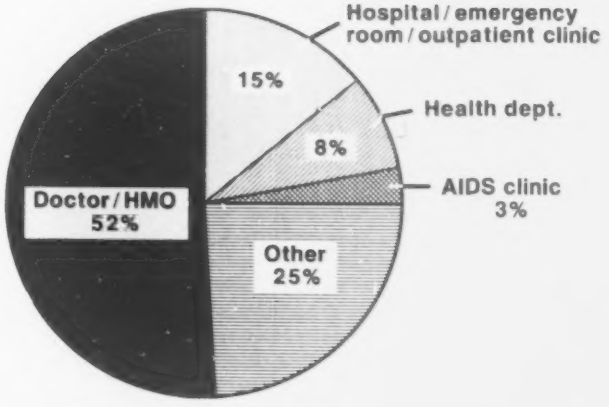
Discussion

Some caution is necessary when interpreting data from the NHIS. While the complex sampling methods used in the survey assure a representative national sample and the response rate to the survey is good, much of the data presented are based on self-report by the participants and cannot be verified. This is especially important when dealing with a sensitive topic such as HIV testing which may affect the person's willingness to respond honestly. However, the figures on HIV antibody testing obtained with the survey have remained relatively stable throughout 1988.[9] Also, the

TABLE 2—Comparison of Demographic Characteristics of Those Tested Voluntarily for HIV Antibodies, Those Tested under Other Circumstances, and Those Not Tested

Characteristics	Circumstance of Testing		
	Voluntary	Other*	Not Tested
	Percent with characteristic		
Race/Ethnicity			
White, non-Hispanic	78	85	79
Black, non-Hispanic	15	8	11
Hispanic	7	5	6
Other	5	2	3
Age (years)			
18–29	40	37	24
30–49	48	47	38
Over 49	12	16	39
Years of School			
Less than 12	14	10	24
12 or more	85	90	74
High-risk Behavior			
Yes	14	3	2
No	86	96	96
Chance of HIV Infection**			
High/medium	9	4	3
Low/none	89	95	94

*Includes those tested because of blood donation and as part of other activities which required testing.
**Chance of having or getting HIV infection.



SOURCE: NCHS, National Health Interview Survey, 1988

FIGURE 2—Distribution of Reported HIV Antibody Testing Sites for Those Planning Voluntary Testing in the Future

information on blood donation is consistent with other data on blood donors.[11–13]

In general, awareness of the HIV antibody test is high. However, it is lower than desired in certain important groups such as racial/ethnic minorities. This accounts for at least some of the deficit in testing rates among these groups; when testing rates were examined among those aware of the test, the racial/ethnic differences were smaller and no longer statistically significant. These lower levels of knowledge about the HIV antibody test and lower testing rates for Black and Hispanic adults are particularly important because of the higher rates of AIDS and HIV infection in these two populations.[14] There are also differences in AIDS rates among various subgroups within the Hispanic population.[15] We were unable to examine differences among these various subgroups because of the small numbers in our data set. The same survey instrument will ultimately be used for 20 months (May 1988 through December 1989) making it possible to examine HIV antibody testing experience within Hispanic subgroups in the future. Awareness of the test increased slightly from 1987 to 1988 among adults in the US[6]; however, it remained the same throughout 1988.[9] Efforts to increase the use of HIV antibody testing should also include ways to increase awareness about the test.

As of October 1988, approximately one in six civilian adults had been tested for HIV antibodies, primarily through blood donation. While those with high-risk behaviors who were tested were more likely to be tested voluntarily, 40 percent were tested as a result of blood transfusion. The observation that some individuals with high-risk behaviors for HIV infection have donated blood despite attempts to encourage self-deferral is not new. Further interpretation of these data is limited because the survey was not designed to address this particular issue. First, we have no information on whether any of these high-risk blood donors indicated at the time of their donation that their blood should not be used for transfusion. Thus, we cannot estimate how much of this donated blood was actually transfused. Secondly, we have no information on when these individuals engaged in high-risk behavior or on the specific dates of blood donation. For some of these donors, the high-risk behaviors may have occurred after their donation.

The characteristics of those seeking voluntary testing indicate that a higher proportion were in groups targeted for testing (e.g., those reporting high-risk behaviors) than those tested by other means or those not tested. However, many in these targeted groups have yet to be tested and most of those voluntarily tested perceived themselves to be at low or no risk of having or getting HIV infection and did not report belonging to groups with high-risk behaviors. This may be one reason why a large percentage reported not getting prevention counseling along with their test results. However, counseling is at least as important as testing in helping to prevent the spread of HIV and even low-risk individuals benefit from having such information. Also, some individuals who present for testing may be unwilling to admit risky behavior to health care providers. Post-test counseling on HIV prevention should be provided to all who are tested.

Of those tested voluntarily, 18 percent did not get the results of their tests. This figure includes those who were tested blindly and could not get their results and those who had not yet received their results when interviewed, as well as those who chose not to get their results. Presumably, most were in the latter category, this figure is similar to the 10–20 percent non-return rate reported to the Centers for Disease Control's Counseling and Testing Statistical Reporting System for persons tested at publicly funded HIV counseling and testing sites (K. Cahill, CDC, personal communication). At present the small size of the group who did not get their test results identified in our survey makes it difficult to further characterize them reliably; it will be important to do so in the future when the number of sample cases is larger.

The information on the number who plan to be tested in the coming year can be used to help ensure that the future demand for testing can be met. According to the figures obtained in the survey, approximately five million adults may voluntarily seek HIV antibody testing in the next year. Many of these persons indicated that they would go to their doctor or HMO to be tested. Evidence from other studies suggests that at least some physicians are reluctant to discuss AIDS and associated risk behaviors with their patients.[16,17] Thus, there continues to be a need to educate physicians and other primary health care providers so that they will have the information and skills necessary to provide adequate pre- and post-test counseling for their patients who request HIV testing.

Counseling and testing for antibodies to HIV will continue to be important parts of the effort to control the spread of this infection. The recent recommendation that HIV-infected individuals be immunologically monitored to identify candidates for prophylaxis against *Pnemocystis carinii* pneumonia may lead to an increase in testing.[18] The NHIS AIDS survey will continue to provide a way to monitor the national experience with HIV antibody testing. A public use data tape containing the data collected in 1987 is currently available; the 1988 data tape was to be available at the end of 1989. During 1990, the survey will be expanded to include more questions about HIV antibody testing.

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May 7 Designated as National Nurses' Day

Each year, a day is set aside to recognize the outstanding contributions made by the nursing profession. This year, May 7 has been designated "National Nurses' Day" and a nationwide observance will highlight the important work and health care provided by the more than two million registered nurses in the United States. The American Nurses' Association (ANA) and its 53 constituent nurses' associations will take part in activities to recognize the numerous contributions nurses make to the health of citizens of the US.

"Nurses Together in Caring" is the theme adopted by ANA for this year's nursing celebration. Nurses play a pivotal role in enhancing and promoting health care and the well-being of patients and family members. The US is experiencing a nationwide nursing shortage at the present time. During the next decade, more than 600,000 additional nurses will be needed. ANA and the state nurses' associations are working together to combat this shortage and misperceptions about nursing as a career. ANA is calling for organized groups to work together to solve the nursing shortage problem, help improve nurses' salaries and working conditions, and provide a greater role for nurses in decision-making processes. For additional information, contact: ANA, 2420 Pershing Road, Kansas City, MO 64108.

National Nurses' Day • May 7, 1990

Persistence of Anti-HBs among Health Care Personnel Immunized with Hepatitis B Vaccine

MARY T. PASKO, PHARM D, AND THOMAS R. BEAM, JR., MD

Abstract: Health care personnel who received the hepatitis B vaccine (Heptavax-B^R, MSD) were followed for persistence of hepatitis B surface antibody (anti-HBs). Response occurred in 135/146 (92.5 percent) vaccinees. Loss of anti-HBs (<72 RIA units; 10 S/N) occurred in 35.9 percent during the 36-month surveillance. Stepwise discriminant analysis found age and magnitude of initial antibody level, but not weight-height index, to be predictive of antibody loss over the 36 months. Twenty-four of 27 employees (88.9 percent) who lost anti-HBs responded to a fourth vaccine dose. In contrast, three of eight initial non-responders (37.5 percent) developed antibody after a fourth vaccine dose. (*Am J Public Health* 1990; 80:590–593.)

Introduction

Hepatitis B virus (HBV) infection is a significant occupational risk to health care personnel.[1–3] Safety and immunogenicity of the hepatitis B vaccine have been demonstrated in immunocompetent health care personnel.[4] However, current evidence from studies of healthy vaccinees and male homosexuals suggests serum antibody will not be maintained indefinitely.[5–9] Whether loss of demonstrable antibody can be equated with loss of protection against disease is not known.

The objectives of the present study were to determine prevalence of baseline antibody to hepatitis B surface antigen (anti-HBs) in our population, to evaluate response rate, and to monitor persistence of anti-HBs serially over three years. Non-responders and those responders who lost antibody (transient responders) were also evaluated and compared to the persistent responder group.

Methods

Participants were health care personnel at the Veterans Administration Medical Center, Buffalo, New York. The study was approved by the Human Studies Subcommittee and each participant signed informed consent prior to enrollment.

The clinic nurse recorded height, weight, health problems and/or medications upon entry into the study. A baseline serum sample was obtained and three doses of Heptavax-B^R (MSD) (20 mcg) were administered intramuscularly according to current recommendations; second and third doses were given one and six months after the first.[10]

Serum samples were collected 7, 12, 24, and 36 months following the first vaccine dose for determination of anti-HBs. Non-responders and those employees with demonstrable loss of anti-HBs to levels less than or equal to 72 radioimmunoassay (RIA) units were given a fourth vaccine dose (20 mcg). Fourth doses were given to non-responders two to three months after the third dose. Transient responders received fourth doses at variable intervals with respect to the third dose but within two to three months of a serum sample demonstrating anti-HBs ≤ 72 RIA units. Serum anti-HBs was then determined one month later. All sera from non-responders were tested for the presence of hepatitis B surface antigen (HBsAg).

Participants were asked to report any known or suspected exposure to HBV. Exposures were investigated by study personnel to determine whether a true HBV exposure occurred.

Serum Studies

Serum anti-HBs was determined in duplicate by radioimmunoassay (AUSAB, Ausria II; Abbott Laboratories, North Chicago, IL). Anti-HBs levels were quantitated according to manufacturer's directions and reported as radioimmunoassay units (RIA units).[11] In our laboratory, 72 RIA units correlated with 10 S/N units (ratio of sample counts per minute to mean counts per minute in antibody negative controls).

Vaccine response was defined as an anti-HBs value of greater than 72 RIA units (10 S/N). Those with a maximal anti-HBs value of 72 or less either 7 or 12 months after the first vaccine dose were considered nonresponders. Persistent responders were those whose anti-HBs values exceeded 72 RIA units (10 S/N) throughout the period of study. Those responders whose anti-HBs values initially exceeded 72 RIA units but declined below this value during the course of the study were considered transient responders.

Weight-height index was calculated as weight (kg)/{height (m)}^{p}, where p equals 2 for males and 1.5 for females.[12]

Statistical Analysis

Data analysis was accomplished with the use of the Statistical Package for Social Sciences (SPSS-PC) (v.2.0; SPSS Inc., Chicago, IL; 1988). Students t-test for unpaired data and stepwise discriminant analysis were performed; 95% confidence intervals (CI) for mean differences were calculated.[13]

Results

Baseline Screen for Anti-HBs

Of 184 employees screened for the presence of serum anti-HBs, 14 (7.6 percent) were found to be positive (>8 RIA units; 2.1 S/N). Anti-HBs levels in six of the 14 sera (42.9 percent) were 72 RIA units or less while eight sera (57.1 percent) exceeded this value. Those with baseline anti-HBs > 8 RIA units were excluded from further analysis.

Response to Vaccination

Of the 170 employees with negative baseline serum, 17 employees either did not complete vaccination or were lost to

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follow-up and seven employees received injections in the buttocks. All of these were excluded from further analysis.

Among the 146 employees remaining, 135 (92.5 percent) responded while 11 (7.5 percent) were non-responders. The mean age of the study population was 36.0 ± 9.9 years. Non-responders (mean 40.5 years) were slightly older than responders (mean 35.7 years; 95% CI of difference = 2.8, 12.4). The mean calculated weight-height index for responders (29.6 ± 6.3) and non-responders (28.9 ± 5.3) was similar.

Persistence of anti-HBs

Antibody concentrations declined to 72 RIA units (10 S/N) or less in 6 of 133 employees (4.5 percent) at 12 months, 21 of 115 (18.3 percent) at 24 months, and in 33 of 103 (32.0 percent) at 36 months after the first vaccine dose (Figure 1). The duration of anti-HBs was related to the magnitude of the antibody response measured seven months after the first vaccine dose (Table 1).

To further characterize the persistent and transient responders, 92 individuals were evaluated from whom serial anti-HBs values were available 7, 12, 24 and 36 months after the first vaccine dose. These 92 individuals were similar with respect to age, initial anti-HBs level and weight-height index when compared to the 43 responders for whom follow-up was incomplete. Anti-HBs levels were maintained above 72 RIA units (10 S/N) in 59 individuals (64.1 percent) for the 36-month period, while 33 (35.9 percent) were transient responders.

Stepwise discriminant analysis revealed that magnitude of the initial anti-HBs concentration and age were significant discriminators of antibody persistence or loss within the 36-month period. Transient responders were older than persistent responders and the magnitude of initial anti-HBs was substantially higher among persistent responders. The weight-height index (WT-HT) was not found to be of predictive value (Table 2).

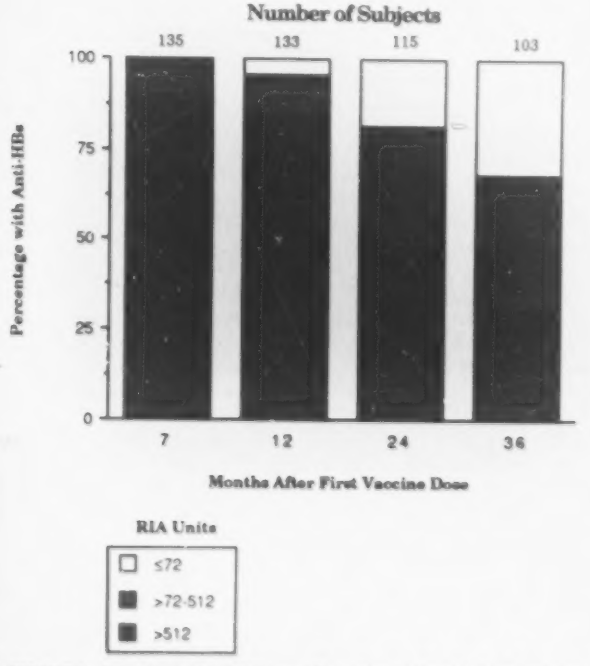

FIGURE 1—Persistence of Anti-HBs over Three Years in Health Care Personnel Who Responded to the Three-dose Vaccine Series

TABLE 1—Loss of Anti-HBs 36 Months after Vaccination (N = 103)

Initial Anti-HBs[a]		No. < 10 S/N	Duration of Anti-HBs (months)[b]
RIA Units	S/N	/total (%)	
72–512	10–50	11/11 (100)	16.3 ± 6.2
>512–5000	50–1000	14/21 (66.7)	25.5 ± 7.1
>5000	>1000	8/71 (11.3)	32.6 ± 3.4

[a]measured 7 months after first vaccine dose.
[b]mean ± standard deviation of time (months) that anti-HBs ≥ 10 S/N.

Exposure to HBV

Twenty-five employees reported 110 exposures to potentially HBsAg positive blood or body fluid. Exposures involved direct contamination of mucous membranes or abraded skin with blood or other body fluids; none was an accidental needle stick. Antigen status of the patients was determined or known in 104 of the 110 (95 percent) incidents and 30 (29 percent) involved HBsAg positive patients. The 30 documented exposure events occurred among nine employees, two of whom were non-responders. None of the nine employees developed HBsAg.

Revaccination of Non-responders and Transient Responders

Response to a fourth vaccine dose was assessed in 27 transient responders and eight non-responders. In comparison to the response elicited following the initial three-dose series, anti-HBs following the fourth dose was of similar or greater magnitude in 24 of 27 (88.9 percent) transient responders. Anti-HBs exceeded initial magnitude by three-fold or greater in 13 individuals (48.2 percent), was of similar magnitude in 11 (40.7 percent), and declined in two individuals (7.4 percent). In one case (3.7 percent), the fourth dose failed to evoke any anti-HBs response. However, this individual initially demonstrated a low antibody response to the three-dose series (anti-HBs = 112 RIA units; 18 S/N). Persistence of anti-HBs following the fourth dose in this subpopulation is under continued evaluation.

In contrast, five of eight non-responders (62.5 percent) given a fourth dose failed to respond. Among the three individuals who responded, anti-HBs persisted above 72 RIA units (10 S/N) for 9, 27, and 36 months, respectively.

Discussion

The results of this study corroborate the findings of others but also provide important additional information about persistence of antibody levels and response to booster injections in recipients of plasma-derived hepatitis B vaccine (Heptavax-B). Since we found a low prevalence of hepatitis B markers among our employees (7.6 percent), these observations support the contention that antibody response and decline in our employee population was indicative of the vaccination process and not inadvertent exposures to contaminated blood and body fluids.

In the present study, initial response rate, magnitude of mean peak anti-HBs and loss of antibody within the first year were very similar to that reported by Dienstag, *et al*,[4] (Table 3), although the relation to age was not as strong as relation to initial response. Other studies[12,14,15] have found age to be an important factor distinguishing responders and nonresponders.

TABLE 2—Comparison of Persistent and Transient Responders followed Serially for 36 Months* (N = 92)

	Persistent	Transient	Difference (95% CI)	p-value[b]
anti-HBs[a]	29951 ± 19409	7470 ± 14450	22481 (14896, 30066)	.00001
age	33.9 ± 8.7	41.7 ± 9.7	7.8 (3.9, 11.7)	.0002
WT-HT	29.4 ± 4.8	31.1 ± 8.7	1.7 (−1.1, 4.4)	.2128

*data expressed as mean ± standard deviation.
[a]initial antibody level at 7 months post dose 1 expressed in RIA units.
[b]stepwise discriminant analysis.

TABLE 3—Response to Vaccination among Two Populations of Health Care Personnel

	Dienstag et al[a] (N = 645)	VAMC (N = 135)
Initial Anti-HBs[b]		
≥100,000	43.1%	NA
≥51,200	NA	23.8%
≥1,000	79.2%	79.4%
≥512	87.0%	90.4%
Mean Peak Anti-HBs[b]	26,718	26,606
Transient Responders[c]	2.3%	4.5%

[a]reference number 4.
[b]RIA units.
[c]Anti-HBs loss within 12–13 months of the first vaccine dose.

Although the influence of injection site on response rate is well established,[16] the role of obesity as a factor in response rate is less clear. In our study, no association was found between likelihood of response and weight-height index as a measure of obesity. In a previous study reporting an association,[12] all vaccine was administered in the buttocks with a 1-inch needle and thus the overall seroconversion rate was low as might be expected (55.7 percent). Horowitz, *et al*,[7] found an association between body mass index (weight/height^2) greater than 25 and prevalence of low antibody levels (<10 mIU/ml) at a median time of 39 months post-vaccination. The subpopulation with low anti-HBs (<10 mIU/ml) included non-responders as well as responders who lost antibody since vaccination. A recent case control study reported by the Centers for Disease Control (CDC) used an age-adjusted logistic model and found an association between obesity and nonresponse for females over the age of 19 years. A similar association was not found for males.[17]

Antibody persistence among vaccine responders in health care populations is of particular interest. During our 36-month surveillance, 32 percent of responders lost antibody (<10 S/N). Jilg, *et al*,[5] in a brief report of "healthy vaccinees" (inclusion groups not further characterized) found antibody in 27.5 percent of the study population fell to 10 IU/l (equivalent to 10 S/N) or below after four years. The authors do not comment on site of vaccine injection, age of their study population, or HBV exposure events.

Several additional studies have attempted to evaluate either initial response or antibody persistence following vaccination of health care personnel. However, vaccine was administered via gluteal injections in two reports[14,15] and response rate was low as expected. In an additional two reports,[9,10] initial antibody levels post vaccination (deltoid injections) were not obtained. Both authors assumed a response rate of 90–95 percent and thus the subset of employees reported to be negative for anti-HBs at the respective serum sampling times included non-responders and low level responders. Additionally, since baseline prevalence of anti-HBs is also unknown in these populations, those with persistent antibody as a result of natural infection cannot be distinguished from vaccine responders.

Extensive long-term follow-up of vaccinees has been reported by Hadler, *et al*, in a study of male homosexuals[6] and by Wainwright, *et al*, in an Eskimo population.[9] In the Hadler study, anti-HBs was maintained above 10 SRU (sample ratio units; equivalent to 10 S/N) in 93, 70, 65, 60, and 58 percent of the population at 12, 24, 36, 48, and 60 months of follow-up, respectively. Similar to Hadler's data, anti-HBs in our study was maintained above 72 RIA units (10 S/N or 10 SRU) in 95, 82, and 68 percent of vaccinees at 12, 24, and 36 months of follow-up. Preliminary review of our 48-month data indicates 52 percent of vaccinees are above 10 S/N (unpublished observation). This percentage is similar to that of Barnes, *et al*,[8] at 48 months (55 percent > 10 S/N). In contrast, Wainwright, *et al*,[9] report 73 percent above 10 S/N at five years post-vaccination for ages 20–49 and 63 percent for those over 50 years of age. Similar to our finding, initial antibody level was a stronger predictor of antibody persistence than age.

Characterization of responders who subsequently experience a loss of anti-HBs has not been previously reported. We found these transient responders to be somewhat older and have lower initial anti-HBs levels than those with persistent antibody. Evaluation of the response to booster doses and persistence of antibody is in progress. Preliminary results from a small group of our transient responders indicate that a single booster dose elicits a response equal to or greater in magnitude than the initial response in 89 percent of recipients. Continued follow-up of this group will provide further information about persistence of antibody.

While the majority of our transient responders developed anti-HBs after a fourth dose of vaccine, the need for booster doses of vaccine is controversial. Although progressive loss of anti-HBs occurs over time, immunity may outlast demonstrable antibody. Among male homosexuals in the Hadler study,[6] the risk of hepatitis B infection was greatest among vaccine non-responders or low level responders. Of the hepatitis B infections that occurred, 76 percent were in individuals whose anti-HBs was below 10 SRU (equivalent to 10 S/N). Only two cases of clinically important hepatitis developed among responders. Likewise, Wainwright, *et al*,[9] found only four cases of HBV infection over five years. All had anti-HBs level < 20 SRU (equivalent to 20 S/N), developed anti-HBc, remained negative for HbsAg, and were asymptomatic. Thus while current evidence suggests that maintenance of demonstrable antibody levels within five years of vaccination may be unnecessary for prevention of symptomatic disease, further studies are required to investigate the issue of life-long protection.

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Maternal and Child Health Law Changes Explained

Recent legislative changes in the maternal and child health block grant program were spelled out in a letter sent by Dr. Robert G. Harmon, HRSA Administrator, to state health officers. The letter is intended to assist states in applying for fiscal 1991 maternal and child health block grant funds and in submitting the fiscal 1989 annual report on state activities funded under this program.

The law was amended by the Omnibus Budget Reconciliation Act of 1989 (Public Law 101-239). The amendments "create a new framework for action on maternal and child health for the 1990s," Dr. Harmon noted.

The amendments, which increase the authorization for appropriations to $686 million for fiscal 1990 and subsequent years, are designed to:

- Promote the PHS Health Objectives for the Nation for the Year 2000;
- Assure expenditure of funds for preventive and primary care services for children and for services for children with special health care needs;
- Improve data collection and analysis; and
- Increase overall accountability in the administration of the program.

The amendments also make structural changes in the block grant to improve state planning, accountability, and targeting of federal funds to priority populations. The changes:

- Prescribe new requirements for the use of allotment funds;
- Establish a new application requirement;
- Establish new state and federal reporting on services, expenditures and health status of target populations; and
- Mandate additional state and federal activities to promote improved access to maternal and child health services.

The changes reflect "work remaining to be done by federal and state authorities in developing effective statewide systems of comprehensive, community-based family-centered continuous care," Dr. Harmon wrote. "They encourage using resources better through effective coordination of MCH block grant activities with those of other major maternal and child health assistance programs providing related services to similar populations."

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Carbon Monoxide in Indoor Ice Skating Rinks: Evaluation of Absorption by Adult Hockey Players

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Abstract: We evaluated alveolar carbon monoxide (CO) levels of 122 male, adult hockey players active in recreational leagues of the Quebec City region (Canada), before and after 10 weekly 90-minute games in 10 different rinks. We also determined exposure by quantifying the average CO level in the rink during the games. Other variables documented included age, pulmonary function, aerobic capacity, and smoking status. Environmental concentrations varied from 1.6 to 131.5 parts per million (ppm). We examined the absorption/exposure relationship using a simple linear regression model. In low CO exposure levels, physical exercise lowered the alveolar CO concentration. However, we noted that for each 10 ppm of CO in the ambient air, the players had adsorbed enough CO to raise their carboxyhemoglogin (COHb) levels by 1 percent. This relationship was true both for smokers and non-smokers. We suggest that an average environmental concentration of 20 ppm of CO for the duration of a hockey game (90 minutes) should be the reference limit not to be exceeded in indoor skating rinks. (*Am J Public Health* 1990; 80:594–598.)

Introduction

Over the past few years numerous cases of carbon monoxide (CO) intoxication in indoor skating rinks have been described in medical literature.[1–9] Most often, the victims have been hockey players, and the source of the contamination has been the ice resurfacing machine.

When inhaled, CO quickly enters the bloodstream where once having displaced the oxygen, it becomes associated with hemoglobin to form carboxyhemoglobin (COHb). The percentage of COHb is the main indicator of the absorption of this toxic gas. It is generally equal to 1 percent among non-smokers[10] and may reach 5 to 10 percent among smokers.[11]

Following acute exposure to CO, there is a rapid increase in the COHb level. At the 3 to 4 percent level, the first signs of adaptation in healthy individuals take the form of an alteration of alertness[11] and arterial dilation in response to a drop in blood oxygenation.[12] These levels are sufficient to worsen coronary symptoms among persons with cardiac problems.[13–16] At 4 percent, careful driving skills are affected,[17] and, between 5 and 10 percent, visual acuity is decreased.[12] Levels between 10 and 20 percent cause headaches and difficult breathing.[12] At 20 percent, headaches worsen, nausea appears and manual dexterity is affected.[12] At 40 to 50 percent, all symptoms intensify, and between 50 and 60 percent, there is a possibility of coma.[12]

Gasoline or propane-driven ice resurfacers give off combustion by-products in their exhaust fumes. Depending on how well the unit operates, there will be a variable level of CO emitted. The problem is generally aggravated by poor ventilation in indoor rinks. The combination of these two factors often results in high levels. The CO reaches its maximum concentration when the ice resurfacer is in operation and gradually drops afterwards.[18]

Extremely high levels of CO have been documented in skating rinks with some levels exceeding 100 parts per million (ppm).[1,3,6,8,9,18–21]

CO absorption is up to three to four times higher during exercise than during rest.[22] We conducted a study among male adult hockey players of the Quebec City area to evaluate the CO absorbed by the players at the end of a 90-minute game. The data were collected between October 26, 1988 and January 28, 1989.

Methods

We chose 10 of the 16 voluntary hockey teams, each playing in its own indoor rinks, guiding our choice on various indicators such as the age and size of the rink, the type of resurfacing machine, and whether or not there was a mechanical ventilation.

Measuring Absorption

To measure absorption, subjects, standing up, took a deep breath which they held for 20 seconds. They then exhaled first into one polyvinyl chloride 900 mL bag and then into two other bags joined by plastic tubing (Tygon). The second sample filled with alveolar air was analyzed within 12 hours using a Bendix 8501-BA infra-red CO analyzer. The quantification technique was developed beforehand, and the correlation between the CO concentration in the alveolar air in ppm and the percentage of COHb was established using a sample of 24 volunteers (13 smokers and 11 non-smokers).[23] The square of the linear correlation coefficient (r^2) was .99, the intercept was −.003 ppm with a standard error of .005 ppm, the slope was 5.39 ppm (5.4 ppm of alv CO ≈ 1% COHb) and the standard error of the slope was .13 ppm.

For each player, the alveolar CO was measured within 15 minutes before and after the game. The difference between the two results represented the CO absorbed by the subject during the match in ppm (ΔalvCO).

Measuring Exposure

Exposure was measured by collecting seven samples of air at 15 minutes intervals, from the players' arrival on the ice rink to their departure. The samples were taken from the penalty box located 1.2 meters above the ice using a Gilian HFS-113A pump and Calibrated Instrument 5 L aluminized bags. Pump flow was set at one liter per minute. These samples were analyzed within 12 hours on the Bendix spectrometer. The average of the seven samples was con-

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sidered the level of exposure by players during the game ([CO]).

Other Variables

Player's age and whether a smoker or non-smoker were obtained by questionnaire. The pulmonary function was quantified with a Roxon Compact vitalograph using the standardization criteria of the American Thoracic Society.[24] We determined the forced expiratory volume in one second (FEV_1), the forced vital capacity (FVC), the mean forced expiratory flow (FEF_{25-75}) and the FVC/FEV_1 ratio. The expected values were estimated using the Knudson method.[25] Finally, players' aerobic capacity ($\dot{V}O_2max$) was evaluated using a sub-maximum progressive effort test on an ergometric bicycle. The initial work load was set on the basis of the weight of the individual and increased progressively by 240 kiloponds meter (Kpm) for four two-minute levels. The $\dot{V}O_2max$ was estimated based on the equation suggested by the American College of Sports Medicine (26).

Statistical Analysis

The data were stratified according to smoking habits (smokers vs non-smokers). In addition to CO exposure, we examined the influence of age, $\dot{V}O_2max$ and the various spirometric indices on absorption, in a multiple regression model using a backward elimination procedure.[27] The pre-selected critical value of the F distribution was set for a significance level of .05. The resulting relationship between absorption and exposure was evaluated with a simple linear least squares regression model using individual values. When heteroscedastic, it was weighted by the inverse of the variance of the means to confirm the validity of the statistical tests.[27] To verify the possible parallelism between smokers and non-smokers, the coefficients were compared with the addition of a term of interaction.

Results

We evaluated 122 players in 10 teams, 32 smokers and 90 non-smokers. Their age varied between 19 and 59 years old (mean = 34.04 years, standard deviation = 7.10 years).

The various spirometric parameters (FEV_1, FVC, FEF_{25-75}, FVC/FEV_1) as well as the age and the $\dot{V}O_2max$ were eliminated by the backward elimination procedure (data available on request to authors). It seems that these variables have very little effect on CO absorption, whatever the smoking habits. As a result, these variables were not considered factors likely to alter the exposure/absorption relationship.

Tables 1 and 2 describe by indoor rink, for non-smokers and smokers respectively, exposure concentrations in ppm, the number of players, the average quantity of alveolar CO (mean and variance) absorbed during the game and the estimated equivalent in percentage of COHb (5.4 ppm alv CO ≈ 1% COHb).

The exposure levels varied from 1.6 to 131.5 ppm. Seven rinks were below 20 ppm, whereas three exceeded 75 ppm.

Table 1 shows that with low exposures (rinks 1 and 3), non-smokers had, at the end of their game, lowered their COHb concentration. In seven other rinks there was absorption and it was proportional to the level of exposure. The concentration of 4 ppm noted in rink 4 represented a balance threshold where there was neither absorption nor elimination.

Table 2 shows an average drop in COHb among smokers at exposure levels as high as 15.1 and 16.2 ppm. This phenomenon is attributable to high basic levels among smokers.

TABLE 1—Variation in the Alveolar Absorption of Carbon Monoxide among Players during a 90-minute Hockey Game (ΔalvCO) and the Estimated Equivalent in Percentage of Carboxyhemoglobin (COHb) based on the Concentration of Carbon Monoxide ([CO]) in the Indoor Skating Rinks among Non-smokers

Arena	Players N	[CO] (ppm)	ΔalvCO Mean Variance (ppm)	(ppm²)	COHb %
1	8	1.6	−.46	.10	−.09
2	12	9.3	3.48	.46	.66
3	13	1.6	−.24	.10	−.04
4	9	4.0	−.04	.44	−.007
5	10	9.5	2.39	.93	.45
6	5	15.1	4.51	1.56	.85
7	14	131.5	69.79	128.90	13.17
8	8	107.3	57.36	35.07	10.82
9	5	16.2	7.81	.70	1.47
10	6	79.3	46.10	40.42	8.70

TABLE 2—Variation in the Alveolar Absorption of Carbon Monoxide among Players during a 90-minute Hockey Game (ΔalvCO) and the Estimated Equivalent in Percentage of Carboxyhemoglobin (COHb) Based on the Concentration of Carbon Monoxide ([CO]) in the Indoor Skating Rinks among Smokers

Arena	Players N	[CO] (ppm)	ΔalvCO Mean Variance (ppm)	(ppm²)	COHb %
1	3	1.6	−6.38	46.32	−1.20
2	2	9.3	−27.72	20.58	−5.23
3	3	1.6	−10.68	11.30	−2.00
4	1	4.0	−18.13	—	−3.42
5	1	9.5	−13.79	—	−2.60
6	4	15.1	−2.38	31.42	−0.45
7	6	131.5	51.50	299.45	9.72
8	6	107.3	45.66	98.87	8.61
9	4	16.2	−9.93	1.81	−1.87
10	4	79.3	34.80	357.27	6.57

Figures 1 and 2 show the relationship between exposure and absorption using a simple linear regression model.

Figures 1 illustrates this model among non-smokers. The r^2 was equal to 0.97 and the intercept to −1.72 ppm (standard error = .69 ppm). This small negative intercept reflected the CO elimination in low exposure concentrations for the non-smokers. The slope at 0.55 ppm (standard error = .01 ppm) indicated that for every 10 ppm of exposure, an adult hockey player (non-smoker) had, at the end of a 90-minute game, absorbed enough CO to raise his alveolar CO concentration by 5.5 ppm (≈1% of COHb). The F ratio was 2681.36 for a significance level below 0.0001. The test had to be interpreted with caution given the heteroscedasticity of the data. Indeed, there was an increase in the variability of absorption proportional to the level of exposure (see Figure 1 and Table 1). For exposures of 1.6 and 131.5 ppm, absorption varied respectively from −0.9 ppm to 0.1 ppm (range = 1 ppm, variance = 0.10 ppm²) and from 51.1 ppm to 87.4 ppm (range = 36.3 ppm, variance = 128.90 ppm²). To correct this situation, the results were weighted with the inverse of the variance of the means. The results of the regression and the test continued to be very significant ($F = 1454.70$, $p < 0.0001$).

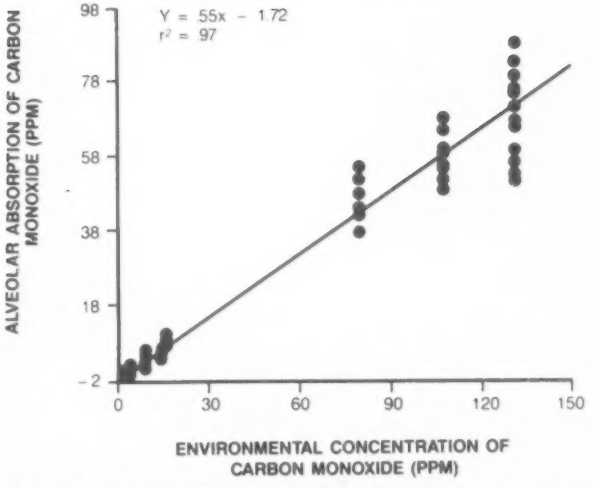

FIGURE 1—Relationship between Alveolar Absorption of Carbon Monoxide among Players during a 90-Minute Hockey Game based on Carbon Monoxide Concentrations in Indoor Skating Rinks (non-smokers).

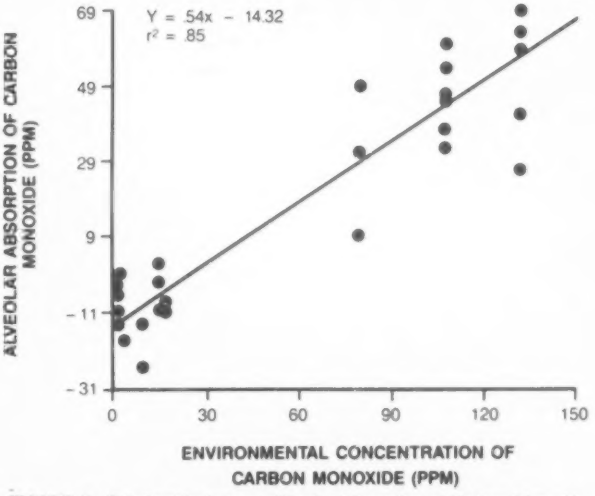

FIGURE 2—Relationship between Alveolar Absorption of Carbon Monoxide among Players during a 90-Minute Hockey Game Based on Carbon Monoxide Concentrations in Indoor Skating Rinks (smokers).

Among smokers (Figure 2), the r^2 was equal to 0.85 and the intercept to −14.32 ppm (standard error = 3.1 ppm) indicated the effect of exercise on CO elimination with elevated basic levels of alveolar CO. The F ratio was 169.50 ($p < 0.0001$). The slope at 0.54 ppm (standard error = .04 ppm) was not considered statistically different to that of non-smokers ($p = .6607$). As a result, after a 90-minute game of hockey, smokers and non-smokers alike increased their concentration of COHb by 1 percent for every 10 ppm of exposure to CO in the indoor rink.

Discussion

There are two types of standards for CO: standards for workers and standards for the public in general.

In an occupational environment the National Institute for Occupational Safety and Health (NIOSH) recommends a maximum time-weighted average (TWA) of 35 ppm for an eight-hour work day and a ceiling concentration of 200 ppm.[10] In the province of Quebec (Canada), the prevailing regulation stipulates 50 and 400 ppm for the same parameters.[28]

These values cannot apply to a hockey player playing in a recreational league. The latter, besides practicing his sport during his leisure time, is involved in an activity that required an energy expenditure greater than the workers. For example, the average energy expenditure of professional activities like lumberjack, mason, miner or carpenter is between 5.0 and 6.4 Mets (1 Met = 3.5 mL $O_2 \times Kg^{-1} \times min.^{-1}$).[29] For a hockey player, it is 12.9 Mets (min. = 9, max. = 15), a greater quantity than a soccer player (10.3 Mets, min. = 8, max. = 12) or a basketball player (11.1 Mets, min. = 9, max. = 13).[29] It is therefore necessary to look to criteria that apply to the general public.

Table 3 gives a summary of Canadian[30] and American[31] standards and of the recommendations of the World Health Organization (WHO).[32] These levels have been deduced using empirical equations based on the controlled exposure of volunteers. Apart from the desirable Canadian standard, they aim at maintaining the COHb concentration for non-smokers at 2 percent, thus protecting the most vulnerable segment of the population from the harmful effects of CO. The desirable Canadian standard aims at allowing an average COHb content below 1 percent, namely the equivalent of the endogenic production of a non-smoker.[30]

In indoor rinks, the British Columbia Department of Labor (Canada) has proposed a reference limit of 25 ppm[8] and, following a review of the literature on the subject, Luckurst and French suggested CO concentrations of 25 and 12 ppm for one hour and eight hours, respectively.[33]

The study that we conducted allowed us to ascertain extremely high CO concentrations in three indoor rinks out of 10. These levels even exceeded the standards set for workers.

Beyond environmental measurements, the best way to describe the risk involves using biological measurements. Only one author has shown interest in this aspect. Spengler evaluated alveolar breath samples among 12 Harvard University players before and after 90 minutes of exercise. An average concentration of 22.5 ppm of CO in the air was sufficient to raise the average COHb level by 1.1 to 3.2 percent.[18]

We succeeded in determining the absorption curve of adult hockey players who practice their sport on a recreational basis, and we noted that absorption was directly related to exposure.

There was great individual variability of absorption at the same CO exposure. For the non-smokers, at low level of CO, the intrateam variability was very low (variance = .316 ppm^2 for teams 1 and 3). At higher exposures, the observed

TABLE 3—Recommended Carbon Monoxide Levels for Ambient Air

	Average per 1 hour (ppm)	Average per 8 hours (ppm)
WHO* (1979)	25	13
United States (1985)	35	9
Canada (1987)		
Desirable	13	5
Acceptable	30	11

*World Health Organization

variability may be explained in part by the analytical error on the measure of alveolar CO itself.[23] Also, other variables undocumented here can probably affect the individual CO absorption at these levels. The ratio of the tidal air to the dead space of the lung and the diffusion constant of the lung can explain some divergencies in the CO uptake.[22] Moreover, factors which affect the players' activity may be important. For example, Green showed differences in the time-motion characteristics (actual playing time, number of playing shifts, recovery time between shifts, etc.) between players depending on the position played (center, wing, defense).[34] In addition for the smokers, the variability of the pre-exposure alveolar CO level is certainly an important factor.

In the more exposed teams (#10-8-7), the average increase of COHb were respectively 8.70 percent, 10.82 percent, and 13.17 percent. Some players had an increase up to 17 percent. Levels like this are certainly sufficient to cause headache, breathing difficulties and, for some people, nausea.[12] They can also alter the choice reaction time,[11] the driving skills,[17] and the visual acuity.[12] All these toxic actions can contribute to accidents on the skating rinks and on the road at the end of the game.

At the present time there are no reliable data on cardiac pathology among hockey players. However, sudden increases in COHb as noted here may have a significant impact on the triggering of an irreversible ischemia. Particularly, if they are within the framework of intense physical exercise. Montgomery estimated for 12 adult recreational players that the intensity of hockey play assessed by the Karvonen method was 85 percent.[35] This represents a strenuous activity and, by itself, it can strain the cardiovascular system.[35] All these considerations make CO a significant threat for the population under study.

Generally, these players drop out of hockey several years and then return to improve their physical fitness or for the friendship obtained.[35] They are often ill-prepared for such intense physical exercise and, of all the groups frequenting indoor rinks, they are probably the most susceptible to the harmful effects of CO given the high level of absorption conferred by the intensity of their activity. If a standard is to be issued, we believe that they should serve as a reference.

We find absurd the idea that a hockey player must adapt to a toxic gas. A 3 percent COHb threshold, which is sufficient to induce physiological compensation mechanisms, must be the reference index. Considering that non-smokers have COHb levels of 1 percent and less, an increase of more than 2 percent cannot be accepted during the hockey game. This corresponds, for the duration of the hockey game (90 minutes), to an average concentration of 20 ppm in the air of the indoor rink, and as a result, it is the limit we propose.

If we rely on the data of our study, this norm is realistic since seven indoor rinks in 10 fell within it. When an ice resurfacing machine with a non-polluting motor (electrical, hydrogen) is not available, there are three means of respecting this norm: good maintenance of the ice resurfacing machine, sufficient mechanical ventilation used wisely, and routine monitoring of CO levels in indoor skating rinks.[7,8,20,36]

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AADS to Offer Oral Health Education for Elderly

The American Association of Dental Schools (AADS) is preparing an education packet to help older persons take better care of their oral health. Funding for the program is being provided by a $49,479 grant from the W. K. Kellogg Foundation.

Through the AADS project, copies of the oral health education packets will be distributed to 55 dental schools and 200 dental hygiene education programs nationwide. Beginning in the Fall 1990, the dental and dental hygiene students will give one-hour presentations at senior centers, clubs, churches, and volunteer organizations, with a special emphasis placed on reaching minority older persons.

The educational materials will be prepared by a team of dentists, dental hygienists, a social worker, and dental educators. By conducting the training, the students will gain first hand experience about oral health and its relation to the aging process, as well as learn to communicate effectively with elderly persons and to network with other health professionals.

For more information, contact the American Association of Dental Schools, 1625 Massachusetts Avenue, Suite 502, Washington, DC 20036; (202) 667-9433.

Passive Smoking and 20-Year Cardiovascular Disease Mortality among Nonsmoking Wives, Evans County, Georgia

CHARLES HUMBLE, MS, JANET CROFT, MPH, ANN GERBER, MSPH, MICHELE CASPER, MSPH, CURTIS G. HAMES, MD, AND HERMAN A. TYROLER, MD

Abstract: The association of passive smoking and cardiovascular disease (CVD) mortality was assessed in a cohort of 513 rural, married Black and White women who were disease-free and self-described as never-smokers at baseline in 1960. Over a 20-year period, 76 of 147 total deaths were attributed to CVD. Relative risk estimates adjusted for age, cholesterol, blood pressure, and body mass from proportional hazards models were 1.59 for CVD (95% CI = 0.99, 2.57) and 1.39 (CI = 0.99, 1.94) for all cause mortality among women with husbands who smoked cigarettes. (*Am J Public Health* 1990; 80:599–601.)

Introduction

Cardiovascular diseases account for about one-half of all deaths in the United States annually.[1] Although active smoking is well-established as a CVD risk factor,[2] the risk for all CVD mortality associated with passive smoking among nonsmokers has not been previously investigated. Recent studies of risks for coronary heart disease,[3–8] stroke,[4,5] or all cause mortality[7,9,10] associated with passive smoking generally have reported weak and/or statistically nonsignificant results.

The 20-year mortality experience of nonsmoking women in Evans County, Georgia was used to assess the association of passive smoking with CVD and all cause mortality. This is the first report that includes data on both Blacks and Whites and on the consistency of self-reported smoking behaviors over time.

Methods

In 1960–61, 92 percent of all residents ages 40–74 years and a 50 percent sample of individuals ages 15–39 years in Evans County, Georgia participated in a cardiovascular disease study that included risk factor measurements, complete physical examinations, and a demographic and medical history interview.[11] Detailed descriptions of the Evans County study design and the 20-year mortality follow-up of the cohort have been reported elsewhere.[11–13] At baseline, 554 (82 percent) White and 389 (83 percent) Black women, among a total of the 1,127 women ages 40–74, reported that they had never smoked. The present study was restricted to the 328 White women and 185 Black older women who had never smoked and were married to male examinees who reported they either had never smoked or were current smokers at baseline. Women married to ex-smokers were excluded from the analyses as the probability for misclassification of these subjects' own smoking habits and those of their husbands was judged to be higher than for spouses of never smokers.[14] A second survey of study subjects in 1967 provides data on the stability of reported smoking status.

Vital status was determined as of May 1, 1980. Underlying cause of death was abstracted from death certificates with codes 390-456 (ICD 8th Revision) defining CVD. All CVD mortality was chosen as an endpoint given the limitations of death certificate data and the small number of deaths attributed to each specific CVD entity.[13] Three subjects who did not have follow-up information were excluded.

Analyses for White women were stratified by social status because of its inverse relationship with smoking status and CVD mortality in this cohort.[13] White women were divided into high social status and low social status groups based on the median of the McGuire-White index of social status for all Evans County Whites. This index, based on occupation, level of education, and source of income of the head of household, was developed for use in rural settings.[15] Since only 5 percent of the Black women in the Evans County population had a social status score above the median for Whites, Blacks were not stratified by social status. Exposure to passive smoking was defined by husband's smoking status (current, never) at the time of the baseline interview.

Mean baseline characteristics by passive smoke exposure were compared using t-tests. Cox proportional hazards models[16] were used to estimate the association of passive smoking with time to all CVD, smoking-related CVD, and all cause mortality in this population while adjusting for age alone and for age, systolic blood pressure, serum cholesterol, body mass index (BMI), and a quadratic term for BMI. Relative risks (RR) and 95% confidence intervals (CI) were calculated using the SAS proportional hazards (PHGLM) modeling procedures,[17,18] and the statistical significance of trends was tested using a method proposed by Rothman.[19] Constancy of the relative risks over time was verified before the proportional hazards were modeled.

Results

Among nonsmoking married women, there were 179 (55 percent) of 328 White women and 117 (63 percent) of 185 Black women whose husbands reported current cigarette

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TABLE 1—Mean and Standard Error of Baseline Characteristics by Passive Smoking Status of Nonsmoking Wives, Ages 40–74 Years, Evans County, Georgia, 1960–61

	White Women				Black Women	
	High Social Status*		Low Social Status*			
	Exposed	Unexposed	Exposed	Unexposed	Exposed	Unexposed
N	(78)	(83)	(101)	(66)	(117)	(68)
Age	51.9 ± 1.0	54.9 ± 0.9	52.1 ± 0.8	53.9 ± 0.9	50.3 ± 0.7	55.5 ± 1.0
Systolic Pressure	145.5 ± 3.1	150.6 ± 2.9	151.6 ± 2.9	157.6 ± 4.3	170.6 ± 3.4	176.5 ± 5.0
Diastolic Pressure	88.4 ± 1.6	90.6 ± 1.4	92.2 ± 1.3	93.1 ± 1.7	103.1 ± 1.9	103.9 ± 2.5
Serum Cholesterol	231.9 ± 4.9	237.5 ± 4.5	227.0 ± 4.4	235.7 ± 7.3	216.5 ± 3.9	216.2 ± 4.6
Body Mass Index	26.3 ± 0.6	26.4 ± 0.6	27.0 ± 0.5	28.6 ± 0.9	29.2 ± 0.6	30.0 ± 0.9

*Based on the median of McGuire-White scores for all White subjects.

smoking behavior. Among both Black and White women there were no statistically significant ($p < 0.05$) differences by passive smoking status for systolic or diastolic blood pressure, serum cholesterol or body mass (Table 1). However, passively exposed Black women and high social status White women were younger on average than nonexposed wives by 5.2 years (95% CI = 3.0, 7.6) and 3.0 years (95% CI = 0.3, 5.5), respectively. For all Whites combined, nonexposed women were also more likely to be above the median SES (socioeconomic status) level than passively exposed women (55.7 percent vs 43.6 percent).

Comparison of self-reported smoking status in 1960 and 1967 showed 98 percent of wives again reported themselves as never having smoked in 1967. Similarly, 98 percent of never smoking husbands maintained their reported status in 1967 while 25 percent of husbands who smoked in 1960 described themselves as non-smokers in 1967.

Age-adjusted RRs for all CVD, smoking-related CVD, and all cause mortality among passively exposed wives were elevated in Blacks and high social status Whites and for all subjects combined (Table 2). The opposite relationship of mortality with passive smoking status was found for low social status White women. Adjustment for other established CVD risk factors (blood pressure, cholesterol, and BMI) generally caused modest elevations of the risk estimates (Table 3) but as with the age-adjusted estimates, the confidence intervals for all subject groups included unity. A trend in risk over level of husband's smoking as reported in 1960 was only seen among high social status Whites; RRs for both total and smoking-related CVD mortality among wives whose husbands smoked <10, 10–20, and >20 cigarettes per day as compared to wives of nonsmokers were 1.02, 2.11, and 2.55, respectively (p for trend <0.06). A marginally significant ($p < 0.09$) trend in risk for all CVD and smoking-related CVD over crude levels of duration of exposure was also apparent only among high social status White women.

TABLE 2—Age-adjusted Relative Risks and 95% Confidence Intervals for Total CVD, Smoking-related* CVD, and All-Cause Mortality for Wives Exposed to Passive Smoke in Evans County, Georgia, 1960–80

Cause of Death		All Subjects	Blacks	Whites	
				HSS+	LSS++
CVD Total	RR	1.34	1.69	1.66	0.60
	95% CI	0.84, 2.21	0.83, 3.46	0.64, 4.32	0.27, 1.34
Smoking-related	RR	1.29	1.57	1.67	0.61
	95% CI	0.79, 2.10	0.73, 3.37	0.64, 4.36	0.25, 1.47
All cause	RR	1.31	1.34	1.80	0.72
	95% CI	0.95, 1.82	0.79, 2.28	0.94, 3.47	0.41, 1.27

*ICD8 codes 410-456
+High social status
++Low social status

TABLE 3—Relative Risks* and 95% Confidence Intervals for Total CVD, Smoking-related+ CVD, and All Cause Mortality for Wives Exposed to Passive Smoke in Evans County, Georgia, 1960–80

Cause of Death		All Subjects	Blacks	Whites	
				HSS++	LSS~
CVD Total	RR	1.59	1.78	1.97	0.79
	95% CI	0.99, 2.57	0.86, 3.71	0.72, 5.34	0.32, 1.96
Smoking-related	RR	1.54	1.68	1.97	0.82
	95% CI	0.93, 2.55	0.76, 3.71	0.72, 5.34	0.31, 2.15
All cause	RR	1.39	1.33	1.97	0.87
	95% CI	0.99, 1.94	0.78, 2.28	1.00, 3.90	0.48, 1.59

*Hazards ratios adjusted for age, diastolic blood pressure, total serum cholesterol, body mass index (BMI = kg/meter^2), and BMI^2
+ICD8 codes 410-456
++High social status
~Low social status

Discussion

These data suggest an elevation of risk for death from CVD and all causes among non-smoking married women whose husbands described themselves as current smokers at the beginning of a 20-year follow-up period. Our findings for Blacks are the first report associating CVD with passive smoking in this racial group. Our observations that social status may modify the effect of passive smoke exposure may be due to chance, but a similar pattern of results for coronary heart disease (CHD) has been reported in other studies of passive smoking. Nonsignificant ($p > 0.05$) two-fold RRs for CHD among passive smokers were reported from studies of middle-class and upper-middle-class women[6] and men[7] while CHD risk was significantly but more modestly increased (RR = 1.2) among a much larger sample of predominantly blue collar Washington County, Maryland women.[8] No increased risk for CHD was reported among public hospital patients whose husbands smoked in four British hospital regions.[5]

It is unlikely that these results can be explained by a change in smoking habits since the minimum age of these women in 1960 was 40. We lack data to examine whether exposure status changed during follow-up due to remarriage. The absence of elevated risk among exposed low social status

White women may reflect a failure of our passive exposure index to measure exposure within the lower social stratum. Power to test for small differences in effect of passive smoking by race or social standing was lacking as were data to evaluate the role of other variables such as alcohol use or physical activity. Taken together with the results of previous studies[6–8,10] and laboratory results suggesting that passive smoke exposure causes decreases in energy production in the mitochondria of heart muscle[20] and increased platelet aggregability in nonsmokers,[21] our results support the health hazards of exposure to passive smoke.[20]

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Community Impact of a Localized Smoking Cessation Contest

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Abstract: The present study assessed the effectiveness of a localized community contest timed to coincide with a statewide smoking cessation contest. Follow-up interviews were conducted with 218 local contest participants and 198 participants from the statewide contest. Overall cessation impact (participation rate × abstinence) was 0.39 percent for the local contest and 0.09 percent for the statewide contest. Localized community contests offered in conjunction with statewide or national campaigns may represent cost-effective methods of reaching large numbers of smokers. (*Am J Public Health* 1990;80:601–603.)

Introduction

Contests to promote smoking cessation appear to represent cost-effective means of producing quit attempts in community settings.[1–3] Quit smoking contests have been offered on a number of occasions as part of the smoking intervention in the Minnesota Heart Health Program (MHHP), a 10-year research and demonstration project intended to reduce the prevalence of heart disease.[4,5]

Several smoking cessation contests have been timed to coincide with the Great American Smokeout conducted annually by the American Cancer Society (referred to as "D-Day" in Minnesota). The present study examined contest participation and outcome for samples of Twin Cities area residents in the 1984 Minnesota D-Day contest. Participants from one of the intervention communities (Bloomington) were compared with a random sample of those from other Minneapolis suburbs (not within the immediate Bloomington area). It was hypothesized that the overall impact of a contest, measured by participation and abstinence outcome, offered in conjunction with specific localized community recruitment and prizes would be greater than that of the statewide contest alone.

Method

Subjects were recruited for a statewide D-Day contest during the Fall of 1984. Recruitment began August 25, 1984

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optimistic to assume that similar results can be achieved elsewhere simply by adding a local promotion to a statewide contest.

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Smoking and Consistently High Use of Medical Care among Older HMO Members

DONALD K. FREEBORN, PHD, JOHN P. MULLOOLY, PHD, CLYDE R. POPE, PHD, AND BENTSON H. MCFARLAND, MD, PHD

Abstract: Smoking behavior of consistently high and low users of medical care services were compared in two groups of older health maintenance organization (HMO) members continuously enrolled for five years and a subgroup who were continuously enrolled for 10 years. Smokers and former smokers, combined, were more likely than never-smokers to be consistently high users of ambulatory services (52 percent vs 34 percent in the five-year group, and 45 percent vs 30 percent in the 10-year group). (*Am J Public Health* 1990; 80:603–605.)

Introduction

Although the increased risk of morbidity and mortality associated with cigarette smoking is well established, the effect of smoking on the use of health services is less clear.[1–3] A number of studies have examined utilization patterns among the elderly,[4–7] but few have examined the relation between smoking and utilization. To our knowledge, no research has examined the relation between smoking and long-term ambulatory utilization patterns among persons 65 years of age and older.

This study seeks to determine: whether smoking behavior is an independent predictor of consistently high use of ambulatory medical services over periods of five and 10 years; and whether, when individuals are categorized as consistently high or low users of services, smokers (defined as current and former smokers) *within* the consistently high or consistently low user group use more medical services and resources than non-smokers (defined as persons who have never been smokers).

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Methods

Data Sources

Study subjects were members of Kaiser Permanente (KP), Northwest Region, a large prepaid group practice HMO (health maintenance organization). Since January 1, 1967, medical record information has been routinely recorded for all medical contacts of a 5 percent sample of KP members.[8]

In 1970–71 the Center for Health Research at Kaiser Permanente conducted an extensive household interview survey of those subscriber units (families) in the 5 percent sample who were continuously enrolled in KP for the full two years of 1969 and 1970. The household survey was designed to obtain detailed individual and family demographic, social, economic, situational, behavioral, and attitudinal data, as well as health status data. The response rate was over 92 percent.[9] The survey and the outpatient utilization system for sample members age 65 and over (as of 1970–71) provide the data for this study.

Study Groups

Two study groups have been identified for this analysis:

- 312 sample members who were 65 years of age or older at the time of the survey, continuously enrolled in KP for the five-year period 1970–74, and alive at the end of 1975.
- A sub-group consisting of 203 sample members still alive at the end of 1980 who had been continuously enrolled for the 10-year period 1970–79.

Utilization Patterns

We determined the actual distribution of total ambulatory care contacts for each of the years 1970–74 for the first study group, and for the years 1970–79 for the second study group. Subjects were categorized as low, medium, or high users of services in each of the years according to the tertile into which their utilization fell.

TABLE 1—Background Characteristics by Medical Care Service Utilization Pattern

Characteristics	Consistently Low Users	Consistently Mixed Users	Consistenily High Users
Mean Age at Survey			
5-year group	70.7	71.5	71.2
10-year group	70.3	70.5	70.9
Percent Male			
5-year group	34.4	43.9	30.8
10-year group	27.7	34.1	18.2
Percent Married			
5-year group	54.7	63.3	65.4
10-year group	48.9	64.2	60.6
Percent High School Education or greater			
5-year group	29.7	38.1	42.3
10-year group	36.2	42.6	45.5
Income-Percent Less than $10,000			
5-year group	61.3	55.9	58.3
10-year group	65.2	55.1	60.7

TABLE 2—Smoking by Medical Care Service Utilization Pattern

	Consistently Low Users		Mixed Users		Consistently High Users		All Members	
	No.	%	No.	%	No.	%	No.	%
Group 1:1970–74								
Never smoked ($\bar{S}$)	42	65.6	127	66.1	25	48.1	194	62.9
Smoker, former smoker (S)	22	34.4	65	33.9	27	51.9	114	37.1
Total	64	100.0	192	100.0	52	100.0	308	100.0
High-Low Difference				17.5				
90% Confidence Interval				2.5–32.5				
Group 2:1970–79								
Never smoked ($\bar{S}$)	33	70.2	80	66.7	18	54.5	131	65.5
Smoker, former smoker (S)	14	29.8	40	33.3	15	45.5	69	34.5
Total	47	100.0	120	100.0	33	100.0	200	100.0
High-Low Difference				15.7				
90% Confidence Interval				−2.3–33.7				

Chi-squared test (Group 1) p = 0.05
Chi-squared test (Group 2) p = 0.19

For the first study group, those who were classified as high users in four or five of the years were defined as "consistently high users" (n = 52). Conversely, those classified as low users in four or five of the years were defined as "consistently low users" (n = 64). All others were defined as "mixed users" (n = 196).

For the second study group, those who were classified as high users in seven or more of the years were defined as "consistently high users" (n = 33). Those classified as low users in seven or more of the years were defined as "consistently low users" (n = 47). All others were defined as "mixed users" (n = 123).

Predictor and Control Variables

Both the predictor variable (smoking behavior) and the control variables (age, sex, marital status, and socioeconomic status) derive from the survey. Current and former smokers are combined in this study (and referred to as "smokers") because of the small number of cases.

In the five-year study group, 114 persons were included in the combined category of current and former smokers. Of this group, 67 percent were former smokers and 33 percent were current smokers. In the 10-year study group, the combined category included 35 persons (69 percent were former smokers and 31 percent were current smokers).

Analysis

To deal with potential confounding effects, we first examined the relation between the sociodemographic factors and the utilization patterns. None of the sociodemographic factors, individually or in combination, was associated with the utilization patterns (Table 1). The numbers, however, are small.

The analysis presented here is limited to the bivariate relationships between smoking behavior and patterns of use and, within categories of users, smoking behavior and resource use. The latter is defined as the number of office visits

TABLE 3—Medical Care Service Utilization by Utilization Pattern and Smoking Status

	Consistently Low Users			Mixed Users			Consistently High Users			All HMO Members		
Group 1; 1970-74 (N =)	Total (64)	S (22)	$\bar{S}$ (42)	Total (192)	S (65)	$\bar{S}$ (127)	Total (52)	S (27)	$\bar{S}$ (25)	Total (308)	S (114)	$\bar{S}$ (194)
Office visits per person per year	1.38	1.68	1.23	5.35	5.46	5.30	11.81	11.3	12.3	5.62	6.12	5.33
Hospital admissions per person per year	0.03	0.05	0.02	0.14	0.15	0.14	0.36	0.33	0.38	0.16	0.17	0.15
Average ambulatory costs per person per year+	$56	$72	$48	$208	$210	$207	$452	$440	$465	$218	$238	$206
Group 2: 1970–79	Total	S	$\bar{S}$	Total	S	$\bar{S}$	Total	S	$\bar{S}$	Total	S	$\bar{S}$
(N =)	(47)	(14)	(33)	(120)	(40)	(80)	(33)	(15)	(18)	(200)	(69)	(131)
Office visits per person per year	1.79	2.29	1.58	5.79	5.65	5.86	11.55	11.25	11.81	5.80	6.18	5.60
Hospital admissons per person per year	0.05	0.07	0.04	0.14	0.14	0.13	0.37	0.33	0.40	0.15	0.17	0.15
Average ambulatory costs per person per year+	$71	$90	$62	$225	$211	$232	$455	$414	$490	$227	$231	$225

+Costs are in terms of 1977 dollars

and hospital admissions per person per year, and the average per-person annual cost of ambulatory services.

Results

As shown in Table 2, for the five-year analysis, 52 percent of the high users compared to 34 percent of the low users were smokers (for the 10-year analysis, 46 percent vs 30 percent, respectively).

Table 3 shows that for both the five-year and the 10-year group, smokers had a higher rate of office visits and hospital admissions and greater average annual ambulatory costs per person than non-smokers. This pattern, however, does not obtain within the mixed user category.

Discussion

Elderly persons who were consistently high users of medical care were more likely than consistently low users to be smokers or former smokers; overall, smokers used more ambulatory care services than nonsmokers.

Limiting the study to long-term elderly HMO members may have understated the effects of smoking on the use of ambulatory medical services because of the differential survival rates of smokers and non-smokers. More smokers than nonsmokers may have been excluded from the study because of deaths. Nevertheless, the finding that, overall, non-smokers used less ambulatory services than smokers is consistent with the public health position that reductions in smoking will improve health status and reduce utilization.

Smoking cessation interventions can be successfully integrated into primary care practice and these interventions can be effective.[10] Long-term follow-up is needed to determine whether smoking cessation programs reduce subsequent utilization and the overall cost of medical care services.

ACKNOWLEDGMENTS

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Routine Communication in Sexually Transmitted Disease Clinics: An Observational Study

DEBRA L. ROTER, DRPH, NANCY KNOWLES, BA, MARK SOMERFIELD, MS, AND JULIE BALDWIN, BA

Abstract: Sixty STD patients were followed during the course of their clinic visit which averaged 2.75 professional contacts. Based on audiotape analysis, virtually all patients received a diagnosis; however, 25 percent of the patients did not receive any information about treatment or prevention, and only 57 percent of patients were given complete information. Patient recall of the information they were given averaged about 43 percent. Patients not seen by a physician during their clinic visit achieved higher recall rates than patients seen by a physician. (*Am J Public Health* 1990; 80:605–606.)

Introduction

Increasingly high rates of sexually transmitted diseases (STD) underscore the importance of Federal Quality Assurance Guidelines for STD Clinics.[1] According to guidelines, patient education should be routinely included in every patient visit and reinforced by every provider the patient sees. Validation of adherence to the Federal guidelines in STD clinics, however, has been infrequent.[2]

The current study was undertaken to observe directly provider-patient communication during routine STD clinic visits, to determine the extent to which diagnostic, treatment, and preventive information is given to patients as suggested by the Federal Guidelines, and to assess patient recall of this information.

Methods

Five health departments in the State of Maryland were asked to participate in a direct observation study of providers' practices in routine STD visits. One health department declined and another was dropped because of logistical difficulties. Observations were made in six clinics, representing three different county health departments between November 1985 and April 1986. Seventy-nine patients were enrolled in this study; this analysis is based on 60 patients for whom complete data were available. The patient sample was predominantly male (71 percent), and racially mixed (45

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Increase in Condom Sales following AIDS Education and Publicity, United States

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Abstract: Data from a national probability sample of drug stores show that condom sales rose from 240 million annually in 1986 to 299 million in 1988. The greatest increase occurred in 1987 after the Surgeon General's report on AIDS was released. Sales of latex condoms with spermicide rose 116 percent. Sales of other types of condoms increased less. These data suggest that Americans are using more condoms and probably more effective condoms in response to AIDS education. (Am J Public Health 1990; 80:607–608.)

Introduction

In his report on the acquired immunodeficiency syndrome (AIDS), the US Surgeon General advised persons whose sex partners may have been exposed to the AIDS virus to always use a condom during sexual intercourse.[1] An extensive public health campaign has since promoted condom use by persons who have sex outside of mutually monogamous relationships. Latex condoms, especially those containing spermicide, are believed to provide more protection than natural membrane condoms and have been the most highly recommended.[2]

Given the extensive public education effort and attendant publicity, we wondered if Americans were using more condoms. In a 1988 survey of urban teenage males, 58 percent reported using condoms during their last sexual encounter, compared to only 21 percent in 1979.[3] We analyzed a more objective measure, condom sales trends, to see how they have changed during the HIV (human immunodeficiency virus) epidemic.

Methods

Drug stores condom sales were estimated from bimonthly sales audits conducted by Nielson Marketing Research (Northbrook, IL). This national probability sample is stratified by size of store (four strata), geographic region (10 strata) and relative urbanization of the county where the store is located (four strata). Approximately 550 drug stores are audited, which compose 1 percent of drug stores and account for 2 percent of drug store sales. The same stores are audited every two months, making the system very sensitive to trends.

Condom sales in areas with a high incidence of AIDS were compared with sales in the remaining US. High incidence areas were defined as the television markets of the following metropolitan areas: Atlanta, Houston, Los Angeles, Miami-Fort Lauderdale, New York, San Francisco, and Washington, DC. These areas encompass one-fifth of the US population, one-third of all condom sales, and two-thirds of all AIDS cases. To reduce the tendency of seasonal variations in sales to obscure short-term trends, we compared sales for each two-month period with sales in the same period a year earlier.

To gauge condom publicity over time, we counted monthly condom citations using three indices: Newspaper Abstracts (University Microfilms International, Inc., Ann Arbor, MI), National Newspaper Index and Magazine Index (both produced by Information Access Company, Foster City, CA). All three showed the same pattern; only Newspaper Abstracts data are shown.

Results

Drug store condom sales grew slowly from 1984 to 1988 except for a 20 percent increase between 1986 and 1987 (Table 1). Sales of some styles grew more rapidly than others. Between 1986 and 1988, sales of *all* latex condoms increased 25 percent (226.1 to 283.4 million). The biggest percentage growth was in latex condoms with spermicide which increased 116 percent (23.2 to 50.1 million). Latex condoms without spermicide increased 15 percent (202.9 to 233.3 million). Natural membrane condom sales increased 7.8 percent (14.4 to 15.6 million).

Sales increased both in areas with a high incidence of AIDS and in the remaining US between 1986 and 1988 (Figure 1). Sales in the high incidence areas were growing throughout 17 of the 18 two-month periods. In contrast, sales were not growing in the remaining US until the beginning of 1987, and sales stopped increasing in July-August 1988. In both areas, condom sales grew rapidly throughout 1987 and early 1988 following the release of the Surgeon General's report in November 1986. Media attention to condoms also increased. Condoms were rarely mentioned before the report, but were increasingly cited in articles, editorials, and cartoons thereafter, reaching a peak in February 1987, when 182 items appeared in the 19 newspapers indexed by Newspaper Abstracts. (Forty percent of the February items concerned the controversy over whether condom advertisements were appropriate for television.) Throughout the remainder of 1987

TABLE 1—Annual Condom Sales in US Drug Stores, 1985–88, and Percent Change from Previous Year

	Sales in Millions (% change)			
	Latex			
Year	With Spermicide	Without Spermicide	Natural Membrane	Total
1985	22.4 (*)	201.4 (*)	13.9 (*)	273.7 (0.6)
1986	23.2 (3.2)	202.9 (0.7)	14.4 (3.9)	240.5 (1.2)
1987	35.0 (50.9)	237.1 (16.9)	17.3 (19.8)	289.4 (20.3)
1988	50 1 (43.2)	233.3 (−1.6)	15.6 (−10.0)	298.9 (3.3)

*data not available

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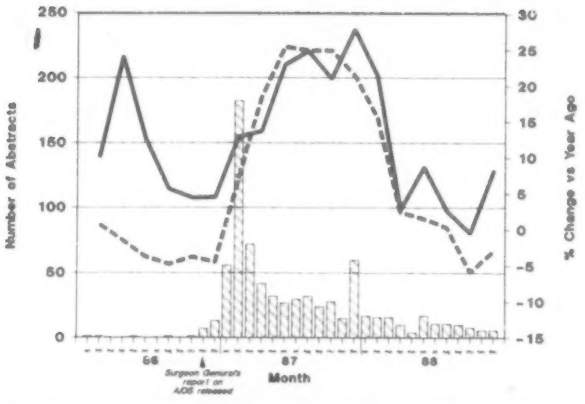

FIGURE 1—Annual Percentage Change in Condom Sales in Drug Stores in Seven Areas with High Incidence of AIDS (solid line) and remainder of United States (broken line) and Number of Newspaper Abstracts Mentioning Condoms (bars), 1986–88.
NOTE: Percent change in sales from the same period one year earlier are shown for two-month periods.

and 1988 media attention was greater than in 1986, but slowly diminished.

Discussion

The 20 percent increase in drug store condom sales in the year following the Surgeon General's report suggests that Americans responded to his message. In comparison, cigarette sales fell only 2.4 percent in the year following the Surgeon General's first report on smoking and health.[4]

The increase in sales was probably related to the recommendation that condoms be used to prevent HIV transmission. Sales of latex condoms, which were recommended for AIDS prevention, increased more than sales of natural membrane condoms. In addition, the greatest percent increase was in latex condoms with spermicide which cost more but may provide additional protection. The overall increase in drug store condom sales between 1984 and 1988 was 26 percent.

How much of the increase was for disease prevention? To answer this, we must estimate the proportion of condoms purchased primarily for contraception. According to studies of US women between 18 and 44 exposed to the risk of unintended pregnancy,[6] 14 percent of married women relied on condoms in 1982, making up 73 percent of the condom users in this population; in 1987, the proportion of married women using condoms was almost unchanged at 15 percent, suggesting that married women choosing condoms are mainly concerned with contraception, not disease prevention. In contrast, the percentage of unmarried women choosing condoms increased from 9 percent in 1982 to 16 percent in 1987, probably reflecting concerns about disease.[6] If all unmarried women choosing condoms do so for disease prevention, then there was a 78 percent increase in women using condoms for disease prevention.

How do sales compare with the number needed if all Americans who have intercourse outside of mutually monogamous relationships use condoms with every act of intercourse? Approximately 23 million Americans reported having more than one sex partner per year and 120 million reported a single sexual partner.[5] However, we do not know how many relationships are mutually monogamous or how often any of these people have intercourse. This lack of knowledge of Americans' sexual activity make it impossible to estimate the number of condoms needed to protect Americans from AIDS.

Other studies have shown increases in self-reported condom use among particular groups. However, behavior self-reports may be more affected by educational messages than behavior itself. Although this study has the advantage of using an objective measure of behavior, we have no evidence that the condoms purchased are actually used. Furthermore, the sales data are from drug stores only; condoms sold elsewhere or distributed free-of-charge are not included. Also, sales data give no information on who buys condoms or why.

This nationwide study of condom purchasing and other repeated surveys of sexual behavior show encouraging trends. But some populations are not being reached adequately. Despite the increase in sales and reported use of condoms, syphilis rates increased 25 percent between 1986 and 1987, with urban areas having the highest rates.[7] We need to learn how to increase the use of condoms among those who are at greatest risk of disease.

ACKNOWLEDGMENTS

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Car Design and Risk of Pedestrian Deaths

LEON S. ROBERTSON, PHD

Abstract: Fatal pedestrian injury rates by cars with relatively sharp front-corner designs were compared to such rates by cars of similar-size with relatively smooth front-corner designs. The relative risk of death by front-corner impact was 26 percent greater among the sharp-cornered cars. Pedestrian death rates from impact with other points on the cars and insurance claim frequencies among the studied cars were similar between the two sets of cars. (*Am J Public Health* 1990; 80:609–610.)

Introduction

In 1967, William Haddon, Jr., sent letters to 38 manufacturers of motor vehicles and their suppliers with pictures of designs that increased the severity of collisions with pedestrians.[1] In 1971, Haddon showed a committee of Congress the design of front ends of cars that resembled the design of ancient weapons—spears and battle axes.[2]

Despite Haddon's warnings, in the 1980s a number of large cars featured sharp protrusions as part of the design of their front corners. In impacts with pedestrians, these designs would be expected to increase the severity of injury because of their shearing effect.[3] The purpose of the research reported here was to investigate the extent to which such designs were associated with increased risk of pedestrian mortality in comparison with similar-sized cars that had relatively smooth front-corner designs.

Methods

A trade magazine with pictures of new cars introduced for sale in the United States during the model years 1980 through 1985 was examined to identify those that had sharp edges on their front corners.[4] Since all of the vehicles with sharp corners weighed more than 3,000 pounds, all cars from the same manufacturers that had relatively smooth front corners and weighed more than 3,000 pounds were chosen for comparison. The compared vehicles are listed in the Appendix.

Data on pedestrian fatalities in which the pedestrians were struck by the listed cars during the calendar years 1982–87 were obtained from the Fatal Accident Reporting System (FARS) computer files. These files contain data on virtually every fatal motor-vehicle injury in the US. The data include clock position of principal impact, eleven and one o'clock for front-corner impacts. Rates of pedestrian fatalities per vehicle years of use by principal point of impact were calculated using sales data[4] to estimate vehicle years of use.

To estimate vehicle use of vehicles sold during the calendar years of the study as accurately as possible, monthly sales data were used to calculate months of use during the year in which a given model was sold. The total years of use was thus the number of months from the sale in the first year divided by 12 plus the total number sold in the year times the number of years subsequently through 1987.

The rates of pedestrian deaths per 100,000 years of use from front-corner impacts and impacts at other points on the vehicles of the sharp-cornered relative to the smooth-cornered vehicles were compared. If differences in the pedestrian death rates in front-corner impacts of the two sets of vehicles were due to differences in driver factors, vehicle factors other than front-corner designs, or use in different environments, a similar difference in non-corner impacts should be found between the two sets of vehicles. There is no reason to believe that other risk factors would increase front-corner impacts but have no effect on pedestrian impacts at other points on the same vehicles.

Also, if the sharp-cornered vehicles were driven differently or had more defects that would increase their risk of collisions generally relative to the comparison vehicles, the insurance claim frequency for those vehicles should be higher. Therefore, the average insurance claim frequency of the two sets of vehicles was also compared using published data from insurance claims.

Results

The numbers of pedestrian deaths and deaths rates per years used in front-corner and other-point impacts of the cars with relatively sharp and relatively smooth front corners are presented in Table 1. The death rate of the relatively sharp-cornered cars in front-cornered impacts was 1.26 times that of the relatively smooth-cornered cars (95% CI = 1.05, 1.50). Thus the sharp-cornered vehicles had an average of 26 percent greater risk of pedestrian deaths in front-cornered impacts. In impacts with other points on the vehicles, the sharp-cornered cars had a relative risk of 0.98 compared to the smooth-cornered cars (95% CI = 0.86, 1.11), that is, virtually the same rates in non-cornered cars.

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TABLE 1—Relative Risk of Fatal Pedestrian Injury By Front-corner Design and Point of Impact—Large US Cars, 1982–87

Front-Corner Design	Deaths		100,000 Years Used	Death Rate		Relative Risk	
	Front Corner	Other		Front Corner	Other	Front Corner	Other
Smooth	589	1403	641.9	0.91	2.19	1.00	1.00
Sharp	160	296	138.8	1.15	2.13	1.26	0.98

95 percent confidence intervals (log odds method) (Front corner 1.05, 1.50) (Other 0.86, 1.11).

The insurance claim data were relative claim frequencies standardized so that the all-car average equals 100.[6] The smooth-cornered cars in this study had an average relative claim frequency of 86. The claim frequencies of the sharp-cornered cars had an average of 87, little different from the smooth-cornered cars' average.

Discussion

As physics and biomechanics predict, moving objects with sharp points do more damage when they strike people than do smooth objects.[3] The 26 percent average greater risk of fatal pedestrian injury from sharp-cornered compared to smooth-cornered cars is consistent with that knowledge. The rate of fatal non-corner impacts, comparing the same vehicles, were similar, suggesting that the major difference among the vehicles in risk to pedestrians is the sharpness of their front corners. The average insurance claim frequency of the two sets of vehicles were nearly the same. Therefore, it is unlikely that driver, environmental, or other vehicle factors accounted for the differences in pedestrian death rates from front-corner impacts.

Vehicle manufacturers often cite costs as the reason for refusing to design and build safer vehicles. Yet the sharp corners on the vehicles in this study require more material than would smooth corners.

The lack of difference in rates of non-corner impacts does not necessarily mean that other points on the vehicles were optimally designed. They were just not different, on average, among these two sets of vehicles. Low bumpers and hoods in combination with smooth, sloped hoods and energy absorbing material in the bumpers and windshield rims are the most efficacious designs.[7]

APPENDIX

Vehicles Studied

Sharp Corners	*Smooth Corners*
Buick Riviera	Buick Electra
Cadillac Eldorado	Buick LeSabre
Chrysler Cordoba	Buick Regal
Chrysler New Yorker	Cadillac Deville
Chrysler Fifth Avenue	Cadillac Fleetwood
Chrysler Imperial	Cadillac Seville
Dodge Diplomat	Chevrolet Caprice
All Lincolns except the 1983–85 Continental and Mark VII	Chevrolet Impala
	Chevrolet Malibu
Mercury Marquis	Chevrolet Monte Carlo
Plymouth Fury	Ford LTD
	Ford Crown Victoria
	1983-1985 Lincoln Continental and Mark VII
	1980–1982 Mercury XR-7
	1983–1985 Mercury Cougar
	Oldsmobile 88 and 98
	Oldsmobile Cutlass (except Ciera)
	Pontiac Bonneville
	Pontiac Gran Prix

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APHA Epidemiology Section Invites Abstracts for Poster Session

The Epidemiology Section of APHA will again be sponsoring a "Late-Breaker" poster session at the upcoming annual meeting. The session will take place on Wednesday, October 3, 1990, 12:30–2:00 p.m. Work completed in 1990 is eligible for consideration. Abstracts must be *received* by September 7 at the address given below. Students and recent graduates are particularly encouraged to submit abstracts. Submit an abstract of less than 200 words (any format) and a return envelope to:

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Males' Use of Public Health Department Family Planning Services

MICHAEL GLASSER, PHD

Abstract: Information was obtained from over 200 males visiting a family planning service either to obtain free-of-charge condoms or incidental to accompanying their female partner. The former group was younger and more likely to reside near the health department; over 40 percent of this group used the health department as sole condom source. Both groups indicated willingness to share in costs for male services. Future programs need to more effectively plan public health family planning for these males. (*Am J Public Health* 1990; 80:611–612.)

Introduction

A pilot study, summarized in detail elsewhere, presented the responses of 70 male clients to a brief questionnaire concerning sexual activity, contraceptive use, and health department visits. Results indicated the health department was serving a substantial population of males and was a central and often exclusive source of condoms. Methodologically, under conditions of anonymity, males were willing to answer questions regarding family planning and sexual activity.

The present research was conducted at the Winnebago County Public Health Department in Rockford, Illinois (population 139,712), with 279,514 living in the Standard Metropolitan Statistical Area. The family planning service of the health department has focused in the past nearly exclusively on female clients, consistent with national trends. Danielson, *et al*, point out that formulas for fund allocation are derived primarily from the number of women in need of services as well as actual number served; men are considered only more peripherally, if at all.

In the present case, a condom distribution service for males was made possible through health department commitment to providing total services related to birth control needs. The family planning clinic set up a distribution desk for condoms where males could request the contraceptive at specific times. Contraceptive availability was advertised in brochures and in the newspaper. Males had to be at least 13 years old to receive condoms. They also received literature regarding family planning and contraceptive use. This study was designed to further provide information on males' use of these services.

Method

Results of the pilot study revealed that there were two different "groups" of males at health department family planning: males using the free-of-charge condom service (Group C) and males attending the family planning visit of their female partner (Group P). We obtained information from both groups.

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Sampling focused on units of time broken down into three parts for each day that the condom distribution service and family planning service were open over a six-month data collection period. Nearly 30 percent of time periods were sampled. During these periods, all males requesting condoms or visiting with their partner were asked to complete a questionnaire or consent to an interview. At the time of this study, the Group P males were specifically visiting with their partners and did not request condoms from the distribution service during that visit. While some males visited family planning more than one time during the study period, they completed only one questionnaire or interview.

Results

A total of 207 males responded (74 percent from Group C and 78 percent from Group P). For Group C, there were 157 separate contacts during the study period, for Group P 144 encounters; altogether, an estimated 2,007 (±46 at 95% confidence intervals = 1961, 2053) contacts with males in one year.

Group C males using the condom service were younger than Group P males, less likely married, had a lower educational level, and lived nearer the health department. They reported a younger age of first intercourse—12.7 compared to 14.4 years. Over 47 percent of Group C had been sexually active by 12 years old compared to 26.2 percent of Group P. At first sexual encounter, nearly 60 percent of all males reported *not* using contraception.

When asked who has primary contraceptive responsibility, Group P males were more likely to indicate both the man and woman and relied less on condoms and more on other measures of birth control, such as the pill. Nearly one-half of Group C males reported condom use for two or more years but 15 percent used condoms for three months or less; over 45 percent indicated always using condoms while nearly 30 percent reported frequency of use as "usual."

Nearly two-thirds of Group C males were repeat visitors to the service, and over 40 percent had visited within one month (Table 1). Almost 42 percent reported the service as sole source for condoms. Main reasons for use were the service was free, friend's suggestion, and no questions asked. Nearly 70 percent visited the service with a friend.

For Group P males, about 60 percent were accompanying their partner to family planning for the first time. The males were there primarily to provide emotional support, give transportation, and be a part of the family planning process. Nearly one-fourth of Group P males had used the condom distribution service.

When asked if they would buy condoms from the service if necessary or share in expenses for services, 72 percent of Group C and 77 percent of Group P males replied yes (Table 2). Both groups reported satisfaction with health department services. The Group C males were a little more likely to indicate interest in additional services, including medical examinations, sexually transmitted disease (STD) programs, and support for adolescent fathers (66 vs 55 percent).

Discussion

The health department provides access to birth control

TABLE 1—Characteristics in Use of the Health Department Family Planning Condom Distribution Service by 100 Males Visiting only to Receive Condoms

First Visit to Condom Service	
Yes	33
No	65
No Answer	2
Length of Time Since Last Visit	
Less Than 2 Weeks Ago	10
2 to 4 Weeks Ago	18
1 to 2 Months Ago	18
More Than 2 Months Ago	19
First Visit	33
No Answer	2
Family Planning as Only Source for Condoms	
Yes	41
No	57
No Answer	2
Reason(s) for Using Condom Service	
Free of Charge	55
No Questions Asked	32
Male Friend Suggested	21
Female Friend Suggested	25
Sent by Health Personnel	3
Sent by Adult	9
Location was Convenient	14
Other	10
In Company of Another Person	
Yes	68
No	30
No Answer	2

both for men wanting condoms and those whose partners are seeking family planning. For males using the condom distribution service, nearly one-half reported exclusive use of condoms, two-thirds made more than one visit to the health department, and 40 percent viewed the health department as sole condom source.

Many Group C males were "repeat" service users. Of the 1,047 projected visits, one-third (346) were first visits. Although it is not clear how many different males are represented in the 701 remaining visits, it is apparent that a large proportion of males could be classified as regular clients.

Group P males comprise a population "in place" to benefit from public health services. They expressed interest in obtaining general as well as family planning services and seem relatively "untapped" as a target of programming.

TABLE 2—Evaluation of Health Department Services by Group C and Group P Males

	% (n = 100)	% (n = 107)
Willing to Purchase Condoms or Share in Costs of Family Planning	Group C	Group P
Yes	72	77
No	28	23
No Answer	(2)*	(4)
Satisfaction Level		
Very Satisfied	51	50
Satisfied	26	22
No Opinion	20	25
Somewhat Dissatisfied	2	3
Very Dissatisfied	1	0
	(3)	(3)
Health Department Should Offer More Services for Men		
Yes	66	55
Not Sure	30	24
No	5	21
No Answer	(3)	(2)

*( ) indicates number of missing cases.

Study limitations were that data were collected at one time rather than longitudinally and the focus was on one facility. Still, it was evident that there was a high frequency of male contacts, if not direct service utilization. In relation to Title X and to health care in general, it appears there is a large population that could be brought more formally under the "umbrella" of family planning.

ACKNOWLEDGMENTS

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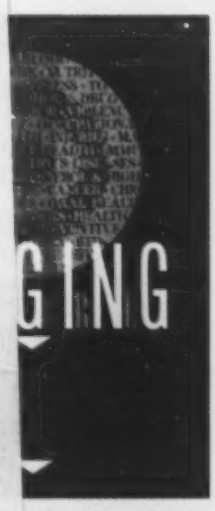

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The Prevalence of Severe Mental Disorder Among Male Urban Jail Detainees: Comparison with the Epidemiologic Catchment Area Program

LINDA A. TEPLIN, PHD

Abstract: This paper presents the prevalence rates of schizophrenia and major affective disorders by age and race among a random sample of male jail detainees. Subjects were administered the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS). The jail prevalence rates were then compared with general population data from the five-city Epidemiologic Catchment Area program using difference of proportion tests and loglinear analysis. After controlling for demographic differences between the jail and five-city samples, the jail prevalence rates were still two to three times higher than those in the general population. These findings suggest several public policy modifications concerning the psychiatric management of our burgeoning jail population. (*Am J Public Health* 1990; 80:663–669.)

Introduction

Mental health professionals speculate that the jails have become a repository for the severely mentally ill. Often referred to as the criminalization hypothesis, this trend is thought to be the unintended consequence of policy modifications, e.g., deinstitutionalization and more stringent commitment criteria.[1–3] Of particular concern are minor offenders (e.g., shoplifters) who could be treated in the mental health system but are instead managed by the criminal justice system. Criminal processing of the mentally ill may be most common among the individuals of the underclass because they have less access to treatment, fewer treatment alternatives, and less social support than wealthier persons.[4]

Jails, rather than prisons, are the critical point to gather psychiatric epidemiological data. Jail populations include detainees awaiting trial and convicted offenders serving sentences of less than one year, while prisons contain only convicted criminals serving longer sentences. Prison samples are biased because inmates are often diverted to forensic psychiatric facilities prior to conviction or imprisonment; prevalence rates of severe disorders in prisons* appear to be lower than those in jails[2] and those in the general population.[5,6]

The Appendix displays the findings of previous prevalence studies of mental illness among jail populations.[7–24] Interestingly, the studies listed show no increase in prevalence rates over time, and little consistency across studies. The tremendous variation in prevalence rates may be explained, in part, by three major methodological limitations:

- *Sample Selection*—To date, only four studies have used random samples.[11–13,23] One study used volunteers,[20] resulting in a plethora of potential biases. The most common type of study includes only persons who are referred for a mental health evaluation, and is obviously biased.
- *Measurement*—The variability in prevalence rates may be a result of the unknown reliability of the instrumentation used in many investigations. Since many studies used unspecified diagnostic criteria and/or nonstandardized instruments,[11,14,16] the diagnostic process is probably inconsistent across samples.
- *Sample Size*—Psychotic disorders have an extremely low base rate, between 1–2 percent.[5,6] The low base rate requires a relatively large sample size to generate accurate prevalence statistics.[25] No study incorporating a random sampling strategy used a large enough sample size.

None of these studies compare the jail prevalence rates with those in the general population. Moreover, because jail detainees are disproportionately young and minority group members[26]—characteristics correlated with severe mental disorder[27]—any comparison with baseline data must control for the jail's demographic composition.

The dearth of reliable psychiatric epidemiological data is critical because of the correctional system's overwhelming growth: between 1978 and 1983, the jail census rose by 40 percent, and by 1987, it increased by another 28 percent.[28–30] Research and intervention at the jail level are particularly important because not only are many jail detainees in need of ameliorative treatment, they are captive and physically amenable to intervention.

This paper provides an accurate estimate of severe mental disorder among male jail detainees in Cook County, Illinois, and compares the prevalence rate to that of the general population.

Methods

Subjects

Jail subjects studied were a stratified random sample (approximately one-half misdemeanants, one-half felons) of male detainees who entered Cook County Department of Corrections in Chicago, Illinois, directly from pretrial arraignment between November 1983, and November 1984 (N = 728). Persons charged with both misdemeanors and felonies were categorized as felons. Data were subsequently weighted to reflect the actual composition of misdemeanants and felons. The jail receives approximately 60,000 admissions per year[31] and is demographically similar to other large urban jails on such variables as race and age.[32] The size of the facility ensured adequate subject availability.

By assuming a binomial distribution for the frequencies of occurrence or non-occurrence, this sample size allows us to reliably detect events which have a base rate in the general population of less than 1 percent.[33]

Address reprint requests to Linda A. Teplin, Associate Professor, Northwestern University Medical School, and Coordinator, Psycho-legal Studies Program, Northwestern Memorial Hospital, 215 East Chicago Avenue, Room 708, Chicago, IL 60611. This paper, submitted to the Journal April 11, 1989, was revised and accepted for publication December 27, 1989.

Editor's Note: See also related editorial p 655 this issue.

*Collins JJ, Schlenger WE: The prevalence of psychiatric disorder among admissions to prison. Paper presented at annual meeting of the American Society of Criminology, Denver, CO, November 1983.

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The age range of subjects was 16–68 years, mean and median age of 26.3 and 25.0 years, respectively; 57.4 percent were employed at the time of arrest. The racial composition was predominantly Black (80.8 percent); 6.5 percent of the sample were Hispanic, 12 percent were other Whites, and the remaining 0.8 percent were largely Asian or American Indian. Educational level ranged from two to 16 years of schooling, mean 10.6 years, median 11.0 years. The sample demographic characteristics (including the skewed racial/ethnic breakdown) reflect the demographic composition of the Cook County Jail.

Procedures

All detainees, excluding persons with gunshot wounds or other traumatic injuries, were part of the sampling pool. Personnel at the jail referred all persons targeted for participation in the project, regardless of their psychiatric morbidity, state of drug or alcohol intoxication, potential for violence, or fitness to stand trial. Subjects were selected via random numbers as they waited to be processed in the jail intake area. To ensure that the sample contained approximately equal numbers of misdemeanants and felons, the interviewers alternated between them in the sampling process.

Interviewers were clinical psychologists who had extensive training in interviewing techniques, psychopathology, and the data collection instrument. Interviewer consistency was scrupulously maintained after the initial three-month training period via mock interviews with live subjects, spot checks, and videotape training. Detainees who consented to be subjects were paid $5 which was mailed to an address of their choice. Of 767 subjects approached, 35 (4.6 percent) refused to participate. The low refusal rate was probably due to the detainees' viewing the research project as a way to avoid the crowded and dismal conditions of the regular intake area. Four other subjects were excluded: two "duplicate" subjects (they were re-arrested and randomly selected again) and two others who appeared to be confabulating their responses. Thus, the final N was 728.

Subjects were interviewed in a soundproof, private glass booth within the jail intake area using the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS).[34] Empirical tests have documented the reliability of the NIMH-DIS in both institutionalized samples and the general population[35–38] (in contrast, see the report by Anthony, *et al.*[39] The DIS systematically differentiates between disorders that were ever manifest, even if currently remitted (lifetime disorders), and disorders in which symptoms have been recently experienced (current disorders).

The NIMH-DIS provides diagnostic categories rather than global psychopathology scores. DSM-III diagnoses are scored from the interview data by a computer program written expressly for this purpose.[40] Because of subject variance over time and the rarity of many disorders, it is difficult to assess the reliability and validity of psychiatric assessment instruments such as the DIS.[41] Nevertheless, a reliability check of twenty pairs of interviews found 93 percent agreement across all diagnoses; 85 percent were given nearly identical profiles. The interview lasted between one and three hours, depending on the number of positive symptoms of the detainee.

The baseline (general population) data were obtained from the NIMH Epidemiologic Catchment Area program[42] which, also using the NIMH-DIS, calculated the prevalence rates of mental disorders in five cities: Baltimore (n = 3,481), New Haven (n = 5,034), Los Angeles (n = 3,131), Raleigh-Durham (n = 3,921), and St. Louis (n = 3,004).[5]

Because the jail sample was male, female subjects were eliminated from the comparison data. We also omitted all Hispanics (as well as other subjects who were neither Black nor White) from both data sets because there was an insufficient number of Hispanic jail detainees to include in the analysis (n = 47). Since the five-city sample excluded persons under the age of 18, we eliminated 53 subjects under that age from the jail sample in the comparison. Because almost all the jail sample were under age 60, we also excluded subjects over age 60 from the comparison. The final sample includes 627 jail detainees and 3,481 from the general population in the five cities: Baltimore, n = 825; New Haven, n = 748; Los Angeles, n = 431; Raleigh-Durham, n = 832; and St. Louis, n = 818. The complex sampling design of the five-city sample[43] required adjustment of the variances and standard errors presented in this paper using design effects estimated with Taylor Series Linearization provided by the National Institute of Mental Health.[44]

We calculated the prevalence rates of three disorders: major depressive episode, manic episode, and schizophrenia (including schizophreniform disorder). For each disorder, separate analyses were conducted to differentiate between "current" (defined as symptomatic within two weeks of the interview) and "lifetime" diagnoses. We conformed with the Epidemiologic Catchment Area study in that the disorders are not mutually exclusive: a subject could meet criteria for more than one disorder.[45]

In order to attain adequate statistical power for the jail-general population comparison, the data from the five cities needed to be combined. Before doing so, we compared the prevalence rates of each disorder across the five cities. This precautionary measure ensured that any observed differences between the jail and the general population data were not merely an artifact of the idiosyncratic characteristics of one or two of the cities. A loglinear analysis indicated that there were no significant or substantial dissimilarities in prevalence rates across the five cities (data available on request to author).

Results

Tables 1 and 2 show the prevalence rates with their standard errors, differences between the jail and the five-city sample, and the ratio of the differences in prevalence rates to the standard error of the difference (differences of proportions test[46]) for current and lifetime disorders, respectively. The uneven partitioning of age is dictated by the limited number of jail detainees over age 32 (n = 117). We did not simultaneously cross-classify by race and age because the resulting decrease in cell size would reduce power to an unacceptable level. The tables do not present data on socioeconomic status (SES) because the only SES indicators used in the five-city general population study (income and occupation) measure the individual's *achieved* status. These variables are inappropriate for this analysis because they have inconsistent bidirectional and temporal effects on the dependent variable. Low income and unemployment may either precede or result from severe mental illness.[47,48] Race is an indirect indicator of SES because of the strong correlation between these two factors.[49,50]

Tables 1 and 2 show that, overall, prevalence rates of current and lifetime major depression, mania, and schizophrenia, and "any severe disorder" (that is, any of the three

TABLE 1—Current Prevalence, Standard Errors, and Standard Errors of Differences for Major Depression, Mania, Schizophrenia and "Any Severe" Disorder for Jail and Five-City Samples

		Major Depression				Mania				Schizophrenia				Any Severe Disorder			
	N	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE
All	4,281	1.49	0.36			0.30	0.19			1.18	0.32			2.50	0.47		
Non-Jail	3,654	1.07	0.22			0.12	0.08			0.91	0.22			1.84	0.31		
Jail	627	3.94	0.78	2.87	4.61**	1.36	0.46	1.24	4.48**	2.74	0.65	1.83	3.03**	6.36	0.98	4.52	5.33**
By race																	
Black	1,541	1.62	0.53			0.51	0.31			1.60	0.55			3.15	0.75		
Non-Jail	994	0.80	0.37			0.14	0.17			1.03	0.46			1.73	0.57		
Jail	547	3.11	0.74	2.31	3.12**	1.18	0.46	1.04	2.52*	2.63	0.68	1.60	2.01*	5.74	1.00	4.01	3.75**
White	2,740	1.42	0.62			0.19	0.32			0.95	0.43			2.14	0.69		
Non-Jail	2,660	1.17	0.27			0.12	0.10			0.87	0.26			1.89	0.37		
Jail	80	9.60	3.31	8.43	5.08**	2.55	1.77	2.43	3.81**	3.52	2.07	2.65	1.74	10.57	3.46	8.68	3.95**
By age (years)																	
18–22	706	0.82	0.60			0.11	0.24			1.90	0.91			2.61	1.06		
Non-Jail	511	0.43	0.38			0.00	—			1.51	0.77			1.94	0.85		
Jail	195	1.85	0.97	1.42	1.67	0.40	0.45	—	—	2.91	1.21	1.40	0.97	4.36	1.47	2.42	1.47
23–27	838	2.17	0.92			0.09	0.22			1.00	0.63			2.92	1.07		
Non-Jail	639	1.12	0.54			0.00	—			0.74	0.48			1.65	0.70		
Jail	199	5.56	1.63	4.44	3.35**	0.39	0.44	—	—	1.82	0.95	1.08	1.07	6.99	1.81	5.34	3.31**
28–32	736	2.14	1.06			0.56	0.66			1.72	0.96			3.70	1.39		
Non-Jail	619	1.29	0.59			0.08	0.16			1.17	0.61			2.36	0.85		
Jail	117	6.62	2.31	5.33	3.17**	3.10	1.61	3.02	3.81**	4.61	1.95	3.44	2.09*	10.80	2.88	8.44	3.64**
33–60	2,002	1.20	0.44			0.36	0.40			0.81	0.42			1.86	0.60		
Non-Jail	1,885	1.15	0.32			0.21	0.15			0.73	0.28			1.72	0.42		
Jail	117	2.01	1.30	0.86	0.65	2.85	1.54	2.64	3.65**	2.18	1.36	1.45	1.24	4.18	1.86	2.46	1.42

*Significant at .05.
**Significant at .01.

disorders) are significantly higher in the jail sample than in the five-city sample. The overall prevalence rates for current disorders are 1.24 percent to 4.52 percent higher among jail detainees than in the five-city sample; for lifetime disorders, the rates are 2.01 percent to 5.07 percent higher in the jail sample. In general, the differences in both current and lifetime prevalence rates between the jail and five-city samples hold when controlling for race. Except for current schizophrenia among Whites, and lifetime schizophrenia among both Whites and Blacks, the differences between the jail and the five-city sample remain statistically significant for Blacks and Whites, ranging from 1.04 percent (current mania among Blacks) to 8.43 percent (current major depression among Whites). The differences between the jail and the five-city sample are somewhat less robust when controlling for age, but this may reflect the small sample sizes.

The difference of proportions test (Tables 1 and 2) is less powerful with low prevalence rates than other techniques, e.g., loglinear analysis.[51] Given the limitations of the difference of proportions test, we reanalyzed the data using hierarchical loglinear modeling[52] (Table 3). This technique enables us to test the differences between the jail and five-city samples while holding both age and race constant.

Table 3 reports the parameters, the ratio of parameters to their standard errors (roughly equivalent to the likelihood ratio chi-square test), and model statistics for the final loglinear models for each current and lifetime diagnosis. Empty cells were set to .05, rather than the less conservative technique of setting them to structural zeros and adjusting degrees of freedom.[53] Notice that for each of the eight models, the site (jail/five-city sample) by diagnosis (present/absent) effect is retained and is in the hypothesized direction. In other words, we can reject the hypothesis of homogeneity for all current and lifetime disorders. In all cases, the coefficient for a diagnosis by site interaction exceeds its standard error by at least 2.58, or with a probability of less than .01. The analysis thus strongly supports our hypothesis: the prevalence rates of major depression, mania, and schizophrenic disorders are significantly higher in the jail than in the five-city sample, and are not an artifact of race or age differences between the two samples.

Discussion

This study provides reliable data concerning the relative prevalence of severe mental disorder among urban jail detainees in Cook County, Illinois, and the five-city Epidemiologic Catchment Area sample of the general population: for all race-age subgroups, the observed jail rates of schizophrenia, major depression, and mania were two to three times higher than in the general population. Moreover, these prevalence rates likely underestimate the true prevalence of mentally ill persons who are processed through the criminal justice system; samples obtained at the jail level omit all persons who are arrested but not incarcerated because they are diverted to a mental health facility during their pretrial hearing.

Our results do not enable us to ascertain whether mental disorder is a causal determinant of a criminal career, or merely a frequent trait among these offenders. Comparison with similar studies is of little utility. As demonstrated in the Appendix, their methodological limitations do not permit us to infer with any certainty whether our prevalence rates are higher than those found in prior studies. Our data thus provide necessary, but not sufficient, evidence that the mentally ill may be diverted into the criminal justice process.

In their exhaustive review of the literature, Monahan and Steadman[27] concluded that the apparently greater prevalence

TABLE 2—Lifetime Prevalence, Standard Errors, and Standard Errors of Differences for Major Depression, Mania, Schizophrenia and "Any Severe" Disorder for Jail and Five-City Samples

		Major Depression				Mania				Schizophrenia				Any Severe Disorder			
	N	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE	Rate %	SE %	Diff. %	Diff./ SE
All	4,281	3.53	0.50			0.64	0.27			1.99	0.40			5.15	0.63		
Non-Jail	3,654	3.15	0.37			0.32	0.14			1.70	0.30			4.41	0.47		
Jail	627	5.75	0.93	2.60	2.65**	2.51	0.63	2.19	5.26**	3.71	0.76	2.01	2.52*	9.48	1.17	5.07	4.09**
By race																	
Black	1,541	2.94	0.71			0.91	0.41			2.64	0.70			5.29	0.96		
Non-Jail	994	1.86	0.56			0.19	0.20			2.05	0.64			3.31	0.79		
Jail	547	4.90	0.92	3.04	2.99**	2.22	0.63	2.03	3.76**	3.70	0.81	1.65	1.57	8.89	1.22	5.58	4.00**
White	2,740	3.87	0.77			0.49	0.43			1.62	0.49			5.07	0.87		
Non-Jail	2,660	3.64	0.47			0.37	0.17			1.56	0.34			4.82	0.58		
Jail	80	11.54	3.59	7.90	2.84**	4.50	2.33	4.13	3.89**	3.52	2.07	1.96	0.98	13.49	3.84	8.67	2.55*
By age (years)																	
18–22	706	1.54	0.79			0.22	0.34			2.46	1.02			3.76	1.25		
Non-Jail	511	1.11	0.60			0.00	—			2.29	0.94			3.23	1.09		
Jail	195	2.66	1.16	1.55	1.28	0.80	0.64	—	—	2.91	1.21	0.62	0.37	5.16	1.59	1.93	0.96
23–27	838	5.17	1.31			0.40	0.44			1.38	0.72			6.42	1.50		
Non-Jail	639	4.28	1.04			0.00	—			1.21	0.62			5.26	1.23		
Jail	199	8.02	1.93	3.74	1.74	1.67	0.91	—	—	1.92	0.96	0.71	0.58	10.09	2.14	4.81	1.92
28–32	736	5.74	1.49			1.35	0.89			3.01	1.20			7.67	1.81		
Non-Jail	619	5.12	1.15			0.77	0.51			2.38	0.87			6.36	1.36		
Jail	117	9.04	2.66	3.92	1.36	4.44	1.91	3.67	2.56**	6.37	2.27	3.99	1.79	14.57	3.28	8.21	2.38*
33–60	2,002	2.74	0.63			0.62	0.52			1.70	0.64			4.19	0.91		
Non-Jail	1,885	2.68	0.48			0.36	0.20			1.47	0.39			3.80	0.61		
Jail	117	3.77	1.77	1.09	0.55	4.85	1.99	4.49	4.77**	5.44	2.11	3.97	2.38*	10.54	2.85	6.74	2.64**

*Significant at .05.
**Significant at .01.

of mental disorder among offenders disappears when sociodemographic factors are taken into account. In this study, however, the differences between the jail and general population persisted even after controlling for race and age.

It is also interesting to note that the observed ratio of current jail rates to current population rates is substantially higher than the comparable ratio of lifetime rates. This finding lends further support to the criminalization hypothesis because we know the arrest occurred during a period of active illness. Mentally ill persons with co-occurring substance abuse and personality disorders may be the most vulnerable to arrest because few treatment alternatives are available.**,[4] Clearly, further research is needed to disentangle the relationship between mental disorder and criminality, as well as to ascertain if mentally ill persons who are more appropriately treated within the mental health system are being funneled into the criminal justice system.

The finding that over 6 percent of all incoming jail detainees were suffering from a "current" psychotic illness suggests several public policy implications.

First, since disorders such as schizophrenia, major depression, and mania require immediate attention, jails must routinely screen all incoming detainees for severe mental disorder.[54] Interestingly, although the courts mandate that jails conduct routine mental health evaluations,[55–57] many jails do not do so.[58] The relative paucity of routine evaluation programs in jails is probably due to insufficient fiscal and mental health resources, as well as a dearth of screening instruments that are appropriate for the jail setting. Recent developments in screening techniques designed to be used within correctional settings[59] are likely to aid in the detection of mental illness. Jail administrators should incorporate these or similar instruments into their routine intake process to ensure that severely ill detainees will be detected and treated.

Second, the prevalence of detainees who are actively psychotic suggests that jail administrators must negotiate programmatic relationships with mental health facilities. Unfortunately, although the courts have stipulated that mentally ill prisoners must receive treatment for mental disorders,[60–62] many jails, particularly those in rural areas, have no mental health liaison.[63] Detainees can either be treated "in house," or referred outside.[64] Whichever system is used, it must be designed to minimize bureaucratic impediments to treatment.

Third, in accordance with the American Bar Association Mental Health Criminal Justice Standards,[65] mentally ill detainees who have committed minor crimes, e.g., trespassing and disorderly conduct, should be diverted to the mental health system. Clearly, persons whose offenses are more symptomatic of mental illness than of criminality should be treated as disordered rather than disorderly.

In sum, our results cannot be interpreted as evidence that the mentally ill are increasingly subject to incarceration. Nevertheless, the data document that the prevalence rate of severe mental disorder is significantly higher in a typical urban jail than in the general population. Additional epidemiological work is needed to ascertain the extent to which this finding is generalizable to other geographical areas. In accordance with court rulings, and in view of the number of severely disturbed detainees in our jails, we must allocate sufficient resources and develop and implement innovative treatment programs so that the mentally ill in jail may be treated expeditiously and in the most humane manner possible.

**Abram KM, Teplin LA: Co-occurring disorders among mentally ill jail detainees: Prevalence, patterns and implications. Manuscript submitted.

TABLE 3—Loglinear Modeling of Major Depressive, Mania, and Schizophrenia, and "Any Severe" Disorder with Site, Age, and Race

	Major Depressive				Mania				Schizophrenia				Any Severe Disorder			
	Current		Lifetime		Current		Lifetime		Current		Lifetime		Current		Lifetime	
	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE	Coeff.	Coeff/SE
Site	0.64716	7.79429**	0.72082	9.59050**	0.36704	2.06367**	0.36565	2.90012**	0.69515	7.37876**	0.77727	9.78684**	0.66814	0.98984	0.73620	11.82454**
Age	−0.08916	−1.98927*	−0.49406	−3.41389**	−0.08916	−1.98927*	−0.68544	−1.82100	−0.08914	−1.98884*	−0.08914	−1.98884*	−0.08917	−1.98941*	−0.28710	−2.94520**
	0.03183	0.72611	0.22196	2.40217*	0.03183	0.72611	−0.21753	−0.76806	0.03183	0.72596	0.03183	0.72596	0.03185	0.72654	0.09918	1.27409
	−0.24995	−4.93573**	0.01302	0.13690	−0.24995	−4.93573**	0.24017	1.14957	−0.24995	−4.93586**	−0.24995	−4.93586**	−0.24995	−4.93585**	−0.07200	−0.90589
Race	0.23301	6.22200**	0.06238	0.89179	0.23301	6.22200**	0.23304	6.22283**	0.23302	6.22212**	0.23302	6.22212**	0.23298	6.22283**	0.14572	2.51508**
Diagnosis	1.93002	24.72801**	1.58786	23.86679**	2.74907	15.65617**	2.51726	15.29506**	2.06427	22.97210**	1.83130	24.68055**	1.66601	27.18680**	1.32669	26.86695**
Age by diagnosis			0.43504	3.02526**			0.60959	1.61575							0.22683	2.35182*
			−0.20492	−2.27764*			0.25908	0.91142							−0.07513	−0.99454
			−0.28878	−3.19583*			−0.50552	−2.40392*							−0.20997	−2.82254*
Race by diagnosis			0.18961	2.85341**											0.10255	1.95333
Site by age	−0.34099	−7.60792**	−0.34967	−7.76005**	−0.34099	−7.60792**	−0.35214	−7.80981**	−0.34101	−7.60835**	−0.34101	−7.60835**	−0.34098	−7.60778**	−0.35156	−7.79333**
	−0.23844	−5.43896**	−0.23417	−5.32806**	−0.23844	−5.43895**	−0.24656	−5.58891**	−0.23844	−5.43881**	−0.23844	−5.43881**	−0.23846	−5.43939**	−0.23556	−5.34996**
	0.01154	0.22779	0.01890	0.37175	0.01154	0.22779	0.02443	0.47626	0.01154	0.22791	0.01154	0.22791	0.01154	0.22791	0.02311	0.45316
Site by race	−0.72519	−19.36411**	−0.73429	−19.40516**	−0.72519	−19.36411**	−0.72522	−19.36494**	−0.72519	−19.36423**	−0.72519	−19.36423**	−0.72515	−19.36845**	−0.73223	−19.39179**
Site by diagnosis	0.33326	4.26968**	0.27160	3.66280**	0.60486	3.44474**	0.61423	4.97229**	0.27986	3.11444**	0.19861	2.67667**	0.32106	5.23927**	0.26795	4.48820**
Degree of freedom	20		16		20		17		20		20		16		16	
Model G^2	23.95568		10.54066		26.69138		14.67989		17.75185		20.09493		17.80153		11.33859	
Model Probability	0.244		0.837		0.144		0.604		0.604		0.452		0.600		0.788	

*Significant at .05.
**Significant at .01.

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APPENDIX

The Prevalence of Mentally Disordered Persons in Jails: A Summary of the Most Recent Research

Study	Description of Sample	Findings
Bolton[7] 1976	N = 1,084 (gender unknown) from 5 county jails	6.7% psychotic
Petrich[8] 1976	N = 539 referred for evaluation; 80% male; 42% felons	49% psychotic 10% depression
Petrich[9] 1976	N = 122 referred for evaluation; 84% male; 80% felons	29% schizophrenia 12% depression 6% mania
Piotrowski, Losacco, & Guze[10] 1976	N = 50 selected from those referred for evaluation; 86% male	22% schizophrenia 10% bipolar
Swank & Winer[11] 1976	N = 445 referred for evaluation; gender unknown; 41% felons N = 100 randomly selected; gender unknown; 71% felons	26% psychosis 5% psychosis
Kal[12] 1977	N unknown; random sample	Any DSM-III diagnosis:50% females; 63% males
Schuckit, Herrman & Schuckit[13] 1977	N = 100 males randomly selected; no felony convictions; no current drug charge	3% affective 48% any diagnosis
Nielson[14] 1979	N unknown; sample referred	24% psychosis
Monahan & McDonough[15] 1980	N = 632 referred for evaluation; 82% male; 55.5% misdemeanants	32% schizophrenia
Whitmer[16] 1980	N = 500 (gender unknown) "in need of treatment"	Averaged 3 prior psychiatric hospitalizations
Morgan[17] 1981–2	N unknown; sheriff's perceptions of mental illness	Estimates from 4–50%
Lamb & Grant[18] 1982	N = 102 males; random sample from referrals for evaluation; drug/alcohol excluded; 53% felons	75% schizophrenia 22% affective disorder
Lamb & Grant[19] 1983	N = 101 females, random sample from referrals for evaluation; drug/alcohol excluded; 37% felons	59% schizophrenia 35% affective disorder
Ninzy[20] 1984	N = 50 volunteers; 74% males	26% psychosis
Virginia DMH[21] 1984	N = 171 "mentally ill" as identified by staff	40% schizophrenia 21% mania
Glaser[22] 1985	N = 50 referred for evaluation; Australians (gender unknown)	48% schizophrenia 16% affective disorders
Guy, Platt, Zwerling & Bullock[23] 1985	N = 486 males randomly selected; 96 given diagnostic interview	12% schizophrenia 4% affective disorder
Valdiserri, Carroll, & Hartl[24] 1986	N = 769 referred for evaluation; 86% male	17% psychotic 5.5% predicted morbidity rate: total jail

Case-Control Study of Lung Cancer and Truck Driving in the Teamsters Union

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Abstract: We conducted a case-control study of lung cancer deaths in the Teamsters Union to compare the risk of different occupations within the teamsters, after controlling for smoking and other confounders. Occupations with no presumed exposure to diesel fumes were used as the nonexposed group. The study population consisted of 996 cases and 1,085 controls who had died in 1982-83 after applying for pensions. Next of kin provided information on smoking, work history, and other potential confounders. Work history data were also obtained from the Teamsters Union. While no single job category had a significant excess risk compared to the non-exposed group, certain sub-groups were elevated. The odds ratio for those with long-term employment as long-haul truckers after 1959 (an approximate date for the introduction of diesel engines) was 1.55 (95% CI: 0.97, 2.47). Long-term drivers of primarily diesel trucks had an odds ratio of 1.89 (95% CI: 1.04, 3.42). Overall, our results suggest that diesel truck drivers have an excess risk of lung cancer compared to other teamsters in jobs outside the trucking industry. However, our findings were not uniformly consistent and our data have many limitations, the most important of which is the lack of data on exposure to diesel fumes. (*Am J Public Health* 1990; 80:670–674.)

Introduction

Inhaled diesel exhaust causes lung tumors in rodents,[1] and there is limited industrial hygiene data showing that truck drivers are exposed to diesel exhaust.[2] Recent human studies[3,4] also have indicated an excess lung cancer risk for diesel-exposed workers, after controlling for smoking. To test the hypothesis that truck drivers have an excess risk of lung cancer, we conducted a case-control study of lung cancer in the Teamsters Union. The risk for men in different occupations within the trucking industry was compared to that of men outside the trucking industry in jobs with no presumed diesel exposure. We had no direct data on level of exposure to diesel exhaust. A separate National Institute for Occupational Safety and Health/National Cancer Institute (NIOSH/NCI) project is now in progress to measure current exposure to diesel exhaust in the trucking industry, and to estimate historical exposures.

Methods

Study Population and Data Sources

Cases and controls were selected from the 10,699 male decedents from the Central States Teamsters files who had filed claims for pension benefits (requiring 20 years tenure in the union) and who had died in 1982 and 1983. We obtained death certificates for 10,485 (98 percent) of these men. Cases were all deaths from lung cancer, coded as ICD 162 or 163 for underlying or contributory cause on the death certificate (n = 1,288). Controls (n = 1,452) were every sixth death from the entire file (sorted by Social Security number), excluding deaths from lung cancer, bladder cancer, and motor vehicle accident.

Next of kin were questioned in detail on work history and potential confounders such as smoking, diet, and asbestos exposure. The response rate was 81 percent (see Figure 1). Most non-response was the result of our inability to locate the next of kin. Of the responses, 80 percent of the interviews were obtained by mail and 20% via follow-up phone calls; 76 percent were completed by spouses, and 24 percent were completed by some other next of kin. On the average, the next of kin had known the decedents for 39 years (38 for cases, 40 for controls).

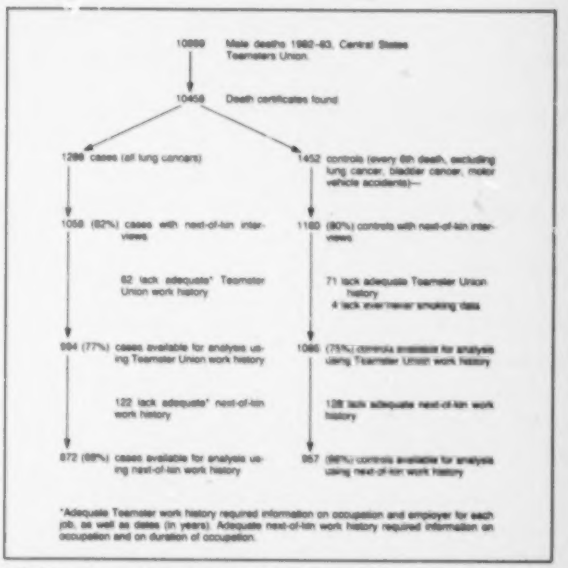
10699 Male deaths 1982-83, Central States Teamsters Union
10458 Death certificates found
1288 cases (all lung cancers)
1452 controls (every 6th death, excluding lung cancer, bladder cancer, motor vehicle accidents)
1058 (82%) cases with next-of-kin interviews
1160 (80%) controls with next-of-kin interviews
62 lack adequate* Teamster Union work history
71 lack adequate Teamster Union history
4 lack ever/never smoking data
994 (77%) cases available for analysis using Teamster Union work history
1086 (75%) controls available for analysis using Teamster Union work history
122 lack adequate* next-of-kin work history
128 lack adequate next-of-kin work history
872 (68%) cases available for analysis using next-of-kin work history
957 (66%) controls available for analysis using next-of-kin work history
*Adequate Teamster work history required information on occupation and employer for each job, as well as dates (in years). Adequate next-of-kin work history required information on occupation and on duration of occupation.

FIGURE 1—Cases and Controls Use in Study of Lung Cancer and Truck Driving in the Teamsters Union

Men were then classified into the job category in which they had worked the longest (diesel truck driver, gasoline truck driver, driver of both types of trucks, truck mechanic, and dock worker), according to 1980 US Census occupation and industry codes.[5] Those who had never worked in any of these job categories were classified either as non-exposed, or as men who had held other jobs with potential diesel exposure (see Appendix A). Approximately 12 percent of the study subjects could not be classified into a principal job category because of missing data (e.g., the number of years worked in specific jobs was unknown to next of kin).

A second work history file was created based on the Teamster Union pension application which listed all teamster

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jobs, as reported by the subject. Each job listed occupation, employer, and dates of employment. Both applicants and Teamster Union staff spent considerable effort to ensure the accuracy of these pension applications.

For the Teamster Union data, each job for each individual was assigned a three-digit US Census code (see Appendix A) for occupation and industry[5] (the latter obtained from commercial listings of employers which included type of industry). The four principal job occupations based on Teamster Union records were long-haul drivers, short-haul or city drivers, truck mechanics, and dock workers (see Appendix A). The Teamster Union work history did not provide information on whether men drove diesel or gasoline trucks. Men were again categorized according to the job category in which they had worked the longest. Most men had worked in only one exposed job category.

Diesel trucks were gradually introduced into the trucking fleet in the 1950s and 1960s in most of the country (before 1950s in Western states). Diesel trucks formed the majority of heavy duty truck sales for the first time in 1961.[6] However, many trucking companies (where most study subjects worked) had completed most of the dieselization of their fleets by 1960, while independent drivers and nontrucking firms may have obtained diesel trucks later (personal communication from companies in NIOSH/NCI industrial hygiene survey). We have chosen 1960 as the date used in our analysis, but we also report results using 1965 as the date for presumed dieselization.

Case-Control Analyses

The number of cases and controls available for analysis is shown in Figure 1. The method of analysis was unconditional logistic regression.[7] A series of dummy (0/1) variables was used to estimate the risk of a number of different job categories in relation to the same nonexposed group (see Appendix B).

All data on covariates, such as smoking and diet, were taken from the next-of-kin interview, while work history data were taken either from the Teamster Union pension applications or from next-of-kin interviews. Separate analyses were conducted for each type of work history data. Analyses considered employment (usual versus never) in a principal job category, as well as the number of years worked in that job. In some analyses using the Teamster Union work histories, only employment after 1959 (or 1964) was considered.

Details on covariate selection for inclusion in the model can be found in Appendix B. In summary, the only covariates that were included in the final model were age (five categories), smoking (six categories), asbestos (dichotomous), and a dichotomous variable for having held other jobs with potential diesel exposure. The fit of this basic model and four continuous exposure variables from the Teamster Union work history was evaluated via the method described by Lemeshow and Hosmer.[8] The chi square resulting from this test was 18.0, with 18 degrees of freedom ($p = .46$), indicating a good fit of the model.

Results

Table 1 shows the number of men and the average duration of employment in the different job categories. Table 2 shows the results of analyses when exposure was considered as a dichotomous variable (i.e., main job versus nonexposed), based on Teamster Union and on next-of-kin work history. No single job category had a highly elevated risk compared to non-exposed men, although most were somewhat elevated.

We further analyzed the Teamster Union work history data by estimating risk by duration of employment as a categorical variable (Table 3). When no cutoff date for employment was used, there was little indication of any trend of increasing risk with increasing duration of employment for any of the job categories. When only employment after 1959 was considered, both long-haul (test for trend, $p = .04$) and short-haul drivers (test for trend, $p = .22$) showed an increase in risk with increased years of exposure; truck mechanics showed no increase in risk with increased years of exposure; truck mechanics showed no increase in risk with time. If 1964 was used for a cutoff date instead of 1959, long-haul drivers continued to show a significant positive trend ($p = .04$), and the highest duration category (13+ years) had an odds ratio of 1.64 (95% CI: 1.05, 2.57). Short-haul drivers, however, no longer showed a positive trend. If duration of employment was used as a continuous variable, significant positive trends

TABLE 1—Number of Men and Average Years Worked in Different Job Categories by Case-Control Status, from Teamster Union Work History and Next-of-Kin Data

Teamster Union Data	Number Cases	Number Controls	Average Years in Job Cases	Average Years in Job Controls
Overall	994	1085	na	na
Long-haul truck driving	609	604	24.2	24.0
City truck driving	121	134	23.4	24.5
Truck mechanic	50	37	22.3	23.3
Dock worker	70	92	23.1	23.3
Other potentially exposed jobs	99	143	na	na
No potential for diesel exposure	45	75	na	na
Next-of-kin data				
Overall	994	1085	na	na
Primarily diesel truck driver	176	155	29.6	27.6
Primarily gasoline truck driver	245	309	29.0	29.7
Drove both types of truck	248	246	31.2	31.2
Diesel truck mechanic	43	41	32.4	32.3
Dock worker	28	40	25.4	25.1
Other potentially exposed jobs	87	128	na	na
No potential for diesel exposure	45	75	na	na
Missing required data	122		na	na

TABLE 2—Odds Ratios for Exposure Considered as Main Job (ever vs never)

Job category	Odds ratio* (95% CI)
Teamster Data	
Long haul driver	1.27 (.83–1.93)
Short haul driver	1.31 (.81–2.11)
Truck mechanic	1.69 (.92–3.09)
Dock worker	0.92 (.55–1.55)
Other potentially diesel exposed	1.44 (.88–2.39)
Next-of-kin data	
Truck driver, primarily diesel truck	1.42 (.89–2.26)
Truck driver, primarily gasoline truck	1.22 (.79–1.88)
Truck driver, both types of truck	1.25 (.81–1.95)
Truck mechanic (diesel)	1.35 (.74–2.47)
Dock worker	0.93 (.49–1.78)
Other potentially diesel exposed	1.54 (.93–2.15)

*Adjusted for age, smoking, and asbestos, the reference group is the non-exposed.

were observed both for long-haul drivers and mechanics, but only after 1959.

Similar trend analyses, based on next-of-kin data, are shown in Table 4. None of the job categories showed a significant increasing trend, although men whose main job was driving diesel trucks did show a marginal increase in risk with increasing duration of employment as a truck driver ($p = .12$). For truck drivers who drove 35 years or longer and drove primarily diesel trucks, the odds ratio for lung cancer was 1.89 (95% CI: 1.04, 3.42). If duration was considered as a continuous variable, only the trend for diesel truck drivers approached significance ($p = .09$).

Using either the Teamster Union work history or that supplied by next of kin, there were no significant interactions between age and exposure, or smoking and exposure. It did not appear that excess risks were confined to any specific category of smokers or non-smokers. The results for lagged analyses (discounting any exposure in the five years preceding death) were similar to the analyses with no lag.

Concordance between Teamster and next-of-kin job categories could not be easily evaluated because job categories were defined differently in each data set (e.g., diesel truck drivers based on next of kin, long-haul drivers based on Teamster Union data). Discrepancies between next-of-kin and Teamster Union work history could occur because next of kin were reporting lifetime work history, while the Teamster records only covered Teamster years. However, 90 percent of men identified by next of kin as primarily diesel truck drivers were long-haul drivers by Teamster Union data, and the corresponding proportions were 82 percent for mechanics and 81 percent for dock workers.

Discussion

The Teamsters Union keeps very thorough records regarding their members, which have not yet been fully utilized by epidemiologists. We were able to study Teamster pensioners using data on their vital status and work history which had been collected by the Union's Pension Fund. It is worth noting that the Union, jointly with employers, has created a Health and Welfare Fund which might be used for studying morbidity.

We observed positive trends in lung cancer risk with duration of employment for long-haul truck drivers after either 1959 or 1964 (Teamster work history), and for truck drivers who drove primarily diesel trucks (next-of-kin work

TABLE 3—Odds Ratios According to Length of Employment for Long-haul Truck Drivers, Short-haul Truck Drivers, and Truck Mechanics

Job Category	Length of Employment (years)	Odds Ratio (95% CI)*	Cases	Controls
No Cutoff Date				
Long-haul driver	1–21	1.25 (.78, 1.97)	205	218
	22–27	1.12 (.72, 1.77)	199	195
	28 or more	1.47 (.94, 2.31)	199	195
Short-haul driver	1–21	1.52 (.86, 2.71)	52	52
	22–27	1.73 (.92, 3.25)	40	34
	28 or more	0.83 (.45, 1.56)	29	57
Truck mechanic	1–21	2.23 (.97, 5.17)	22	13
	22–27	1.20 (.53, 2.72)	17	17
	28 or more	1.88 (.66, 5.35)	11	8
*Exposure after 1959***				
Long-haul driver	1–11	1.08 (.68, 1.70)	162	230
	12–17	1.41 (.90, 2.21)	228	203
	18 or more	***1.55 (.97, 2.47)	213	171
Short-haul driver	1–11	1.11 (.61, 2.03)	36	58
	12–17	1.15 (.63, 2.43)	37	45
	18 or more	1.79 (.94, 3.42)	40	31
Truck mechanic	1–11	1.83 (.80, 4.19)	19	16
	12–17	2.08 (.78, 5.52)	15	8
	18 or more	1.50 (.59,3.40)	16	13

*These data reflect two analyses, with employment restricted to after 1959 or without such a restriction. Each analysis included all the above exposure variables. Odds ratios were also adjusted for age, smoking, and asbestos. Variables for work as a dock worker or in other potentially diesel-exposed jobs were also included in the model. For all comparisons, the non-exposed were those who had never worked in principal jobs categories nor in any other job potentially exposed to diesel exhaust.

**For the 1959 cutoff, we have used different cutpoints, because the maximum possible length of employment in a given job category is 23 years (1960–83). Cutpoints were chosen a priori by dividing long-haul drivers into three groups of approximately equal size. See text for results using 1964 cutoff.

***The linear trend in odds ratios for long-haul drivers was significant ($p = .04$). The coefficient was 0.027, which increased to .037 when exposure was defined after 1964.

SOURCE: Teamster Union Work History.

TABLE 4—Odds Ratios According to Length of Employment for Men as a Truck Driver or Mechanic for Men Whose Main Job Was Diesel Truck Driver, Gasoline Truck Driver, Driver of both Gasoline and Diesel Trucks, or Truck Mechanic

Main Job Category	Years of Employment* (years)	Odds Ratio (95% CI)**	Cases	Controls
Diesel truck driver	1–24	1.27 (.70, 2.27)	48	52
	25–34	1.26 (.74, 2.16)	72	67
	35 or more	1.89 (1.04, 3.42)	56	36
Gasoline truck driver	1–24	1.24 (.74, 2.16)	72	87
	25–34	1.10 (.67, 1.80)	87	112
	35 or more	1.34 (.81, 2.22)	86	111
Drove both truck types	1–24	1.27 (.71, 2.26)	50	51
	25–34	1.15 (.70, 1.90)	95	100
	35 or more	1.34 (.81, 2.20)	102	95
Truck mechanic	1–24	1.69 (.61, 4.67)	11	9
	25–34	1.39 (.63, 3.07)	20	16
	35 or more	1.09 (.44, 2.66)	12	16

*Years of employment as a truck driver does not necessarily signify years of employment driving a particular type of truck.
**All the above exposure variables were included in the model together. In addition, odds ratios were adjusted for age, smoking, and asbestos. Dock workers and men in other potentially exposed jobs were also included in model. For all comparisons, the non-exposed were those who had never worked in principal job categories nor in any other jobs potentially exposed to diesel exhaust.
SOURCE: Next-of-Kin Data

history). These findings suggest that there may be a real increase in risk with increased employment driving a long-haul or diesel truck.

Mechanics showed marginal elevated risks of lung cancer, especially based on Teamster work history. Preliminary sampling results from NIOSH/NCI surveys indicate that mechanics are likely to have been exposed to relatively high levels of diesel exhaust, especially in winter. However, the risk for mechanics did not appear to increase consistently with duration of employment. Also, there is evidence in the literature that mechanics have been exposed to low levels of asbestos in the course of working on brakes.[9] While we included data from next of kin on asbestos exposure in the model, the data on asbestos may have been inadequate to control for confounding.

Dock workers showed a slight deficit in risk. It is possible that dock workers have had less diesel exposure than truck drivers and mechanics. Dock workers frequently unloaded trailers disconnected from the cabs with engines in open air terminals. Short-haul drivers in the Teamster data, but not gasoline drivers (next of kin data), showed some evidence of increased risk.

The principal limitation in this study is the lack of exposure data indicating level of diesel exhaust for the different job categories studied. Such data would enable us to draw firmer conclusions with respect to the role of diesel exhaust, rather than employment in presumably diesel-exposed jobs. Unfortunately, little data exist on historical levels of diesel exhaust in the trucking industry.

Other limitations include: 1) possible misclassification of smoking habits by next of kin, 2) misclassification of exposure by next of kin, 3) a relatively small non-exposed group (n = 120) which by chance might have had a low lung cancer risk, and 4) lack of sufficient latency (time since first exposure) to observe a lung cancer excess. On the other hand, next-of-kin data on smoking have been shown to be reasonably accurate[10], non-differential misclassification of exposure (between cases and controls) would only bias our findings toward the null hypothesis of no association, and the trends of increased risk with increased duration of employment in certain jobs would persist even if the non-exposed group had had a higher lung cancer risk. Finally, the lack of potential latency would also make any positive results more striking.

In view of the above limitations, the results of this study should be considered cautiously. However, other recent findings support our results. The study of Boffetta, *et al*,[3] based on data from the American Cancer Society, indicates that truck drivers show an increased risk of lung cancer after correction for smoking (RR = 1.24, 95% CI: 0.93, 1.66), and that this increase is primarily associated with truck drivers with long duration of diesel exposure (self-reported). Other recent data support an increased lung cancer risk due to diesel exposure in general. The Boffetta study cited above showed an increased in lung cancer risk for those with diesel exposure in any occupation (RR = 1.18, 0.97–1.44), and this risk increased with duration. Garshick, *et al*,[4] conducted a case-control study of 1,256 lung cancer cases and matched controls who had died in 1982–83 after railroad work. Industrial hygiene assessments were done to determine which job categories had significant exposure to diesel exhaust. Those men with 20 years or more duration of employment in diesel-exposed jobs after 1959 (date when locomotives were all presumed to be diesel) showed a significant excess risk (odds ratio = 1.41) compared to the non-exposed, after controlling for smoking and asbestos. These positive findings for truck drivers and other diesel-exposed workers lend plausibility to our own results.

ACKNOWLEDGMENTS

The NIOSH clerical staff, led in this study by Pauline Bishak, and the Central States Teamsters Pension Fund staff (particularly Alan Levy, Mary Balint, Viola Baba, and Mark Felke) performed invaluable service in collecting the raw data for this study. A preliminary version of this paper was presented at the American Public Health Association conference in Chicago, October 25, 1989.

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APPENDIX A

1980 Census[5] Occupation and Industry Codes Used in Defining Job Categories in Teamster Union Work Histories

Job category	Occupation Codes	Industry Codes
Long-haul driver	804 (truck driver, heavy)	any
City driver	805 (truck driver, light)	any
	806 (driver/sales for bakeries dairies, cleaners, or beverage company	111, 101. 771, 120, any if occupation is sales or route driver
Truck mechanic	507 (truck mechanic)	410 (trucking)
Dock worker	343 (checker)	410
	359, 363 (dispatcher)	410
	364 (shipping clerk)	410
	365 (stock clerk)	410
	368 (checker)	410
	843 (dock foreman)	410
	883 (dockman)	410
	856 (forklift operator)	410
	889 (warehouseman)	410
	859 (moving man)	410
Other jobs with potential diesel exposure	all except above jobs in 410 (trucking)	410
	503–517 (other mechanics)	any
	803–859 (other vehicle drivers)	any
	343 (checker)	any except 410
	359, 363 (dispatcher)	any except 410
	364 (shipping clerk)	any except 410
	365 (stock clerk)	any except 410
	368 (checker)	any except 410
	843 (dock foreman)	any except 410
	883 (dockman)	any except 410
	856 (forklift operator)	any except 410
	889 (warehouseman)	any except 410
	859 (moving man)	any except 410
	885 (service station work)	any
	019 (Teamster official)	410

APPENDIX B

Definition of Non-exposed and Selection of Covariates for Inclusion in the Model

The same non-exposed group was used for the analyses based on Teamster Union work history and the analyses based on next-of-kin work history. The non-exposed group was chosen from those who, based on Teamster work history, had never worked as either a long-haul driver, a short-haul driver, a mechanic, a dock worker, or any other potentially diesel-exposed job (Appendix A). Many of these men worked in dairies. Those with no potential diesel exposure via Teamster work history (n = 156) were then further reviewed to exclude anyone with any jobs reported by next of kin which might have had potential diesel exposure (e.g., men who drove trucks prior to joining the Teamsters Union and working in a non-exposed job). The classification of men into the non-exposed group, like all classifications into job categories, was done without any knowledge of case-control status.

Exposure variables and covariates were tested by adding them to a basic model, which included variables for smoking (six categories) and age (five categories). The odds ratios for lung cancer did not increase monotonically with age (using the youngest group as referent), prompting our use of categorical rather than continuous age variables (odds ratios, in reference to age less than 45 years, were 2.86 for ages 45–54, 3.42 for ages 55–64, 3.71 for ages 65–74, and 2.12 for ages greater than 75). Cigarette smoking was categorized into never smoker, current smoker (current at time of death) with unknown amount, current moderate smoker (one pack or less), current heavy smoker (more than one pack), former smoker who quit before 1963, and former smoker who quit 1963–79. The odds ratios for smokers, in reference to never smokers, were 5.64 for current light smokers, 8.85 for current heavy smokers, 4.48 for former smokers who quit in the 20 years before death, and 2.41 for former smokers who quit prior to 20 years before death.

Variables other than exposures, smoking, or age were not included in the model if they did not act as confounders (did not change the estimates of exposure effect), and if they did not interact significantly with the exposure variables. Trends with duration of employment were tested either via inclusion of continuous variables, or via categorization of duration and a test for trend described by Rothman.[11] Cutoff points for categories of duration were chosen a priori, based on dividing long-haul (or diesel when using next-of-kin data) truck drivers into three categories of duration with approximately equal numbers in each. These same cutpoints were then used for the other job categories.

The following variables were neither significant predictors of lung cancer nor acted as confounders: smoking a pipe or cigar, race, state of residence at death, coffee drinking, work in construction, work in shipyards, and driving a truck with a partner who smoked. The frequency of consumption of certain types of vegetables and fruits was of interest because of a possible protective effect of Vitamin A.[12,13] The only significant diet variable was consumption of deep yellow vegetables. The odds ratio for infrequent consumers (one or two times a month or less) of yellow vegetables was 1.38 (1.10–1.76), and for average consumers (one or two times per week) was 1.26 (.98–1.62), both compared to frequent consumers (three or more times per week). However, these variables did not confound the associations under study and were not included in the final model. Asbestos exposure was a significant predictor (odds ratio = 2.80), had some confounding effect on the exposure variables for mechanics, and was retained in the final model.

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Nongenetic Influences of Obesity on Other Cardiovascular Disease Risk Factors: An Analysis of Identical Twins

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Abstract: The importance of genetic influences on obesity has been emphasized recently. We conducted matched co-twin analyses of 250 pairs of White, male, monozygotic twins from the National Heart, Lung, and Blood Institute (NHLBI) Twin Study. Entirely in the absence of genetic influences, obesity was significantly associated with systolic and diastolic blood pressures; one-hour, post-load glucose; total, LDL-, and HDL-cholesterol; and triglycerides among these 42–55 year old men. Similar results were obtained in longitudinal analyses of weight change during adulthood (from mean age of 20 to mean age of 48 years) and risk factor status at middle-age. These results indicate that behaviors and environmental exposures that occur later in life are responsible, at least in part, for the associations between adult obesity and cardiovascular disease risk, supporting the appropriateness of weight reduction efforts during adulthood. (*Am J Public Health* 1990; 80:675–678.)

Introduction

Obesity, hypertension, glucose intolerance, and high lipid levels tend to cluster in families.[1,2] Obesity is also correlated with these other cardiovascular disease risk factors in individuals.[3,4] The extent to which this clustering is caused by common genetic influences on multiple risk factors or by shared environmental or behavioral factors is unknown. In a previous report,[5] investigators from the National Heart, Lung, and Blood Institute (NHLBI) Twin Study demonstrated that many cardiovascular disease risk factors have genetic determinants. Using another analytic approach with the same data, we sought to determine whether associations exist between obesity and the other cardiovascular disease risk factors independent of genetic influences.

Comparison within genetically identical, monozygotic twin pairs removes all genetic variability.[6] If intrapair differences in cardiovascular disease risk factors are related to intrapair differences in obesity, these associations must be attributable to environmental or behavioral, and therefore potentially alterable, influences. In this paper, we use longitudinal data from a group of White, male, monozygotic twins in a matched-pair analysis to assess the nature of the relationships of obesity and adult weight gain with blood pressure, glucose tolerance, and lipid levels at middle-age.

Methods

Study Subjects

Data collection for the NHLBI Twin Study took place between July 1969 and June 1973 in five locations: Framingham, Massachusetts; Indianapolis, Indiana; and Los Angeles, San Francisco, and Davis, California. Participants were 514 pairs of White male twins who were members of the National Academy of Sciences-National Research Council Twin Panel[7] and who lived in California, New England, or within 200 miles of Indianapolis. All twins were born during the years 1917 through 1927 and served in the US Armed Forces during the period from World War II through the Korean War. Twins were ages 42–55 years at the study examination. In this report, only the 250 monozygotic twin pairs were included.

Data Collection

After an overnight fast, both members of each twin pair received physical examinations on the same day by different physicians. Weight and height were measured in pounds and inches. Each individual's weight and height also were obtained from records of the physical examination prior to military induction (ages 17 to 28). After conversion to kilograms and meters, body mass index (BMI) was calculated as the measure of obesity (weight/height^2). Blood pressure was measured three times using a standard sphygmomanometer: first by a nurse, later twice by a physician. The participant was seated, and both systolic and diastolic (Phase V) blood pressures were recorded. The mean of the two physician readings was used in this report. Blood samples were collected to determine glucose tolerance, plasma cholesterol and triglycerides, and serotypes for assignment of zygosity. A 50-g oral glucose load was given one hour before blood was drawn unless the subject had diabetes and was currently using insulin or oral hypoglycemic agents or had glycosuria of 3+ or greater. Glucose levels were measured at the examination centers using an SMA 12/60 instrument (Technicon Instruments, Tarreytown, NY). Total, HDL-, and LDL-cholesterol[8] and triglyceride[9] levels were determined at three regional laboratories; specimens were exchanged to maintain standardization. Additional details of the recruitment process, response rates, zygosity determination, clinical examination, and questionnaire are available.[5,10]

Statistical Analyses

Matched-pair t-tests and linear regression models were computed using SAS computer programs.[11] Both members of a twin pair were included in both the unmatched and matched analyses. However, unmatched analyses were adjusted to avoid overestimating the statistical significance of parameter estimates due to correlated errors between co-twins.[12] For the matched analyses, regression models incorporated intrapair differences of the dependent and independent variables.

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The intrapair difference for each variable was obtained by subtracting the value for one twin from that of the other in each monozygotic twin pair. For analyses of weight change, the value of BMI at the military induction physical examination was subtracted from the value of BMI at the study examination.

Sample sizes varied between 219 and 245 pairs in the different regression analyses because of missing values and specific exclusions of individuals using medications that affect blood pressure, glucose, or lipid levels. Because the distribution of triglycerides was skewed toward higher values, we used the natural log transformation on this variable in all regression analyses.

Results

Means and standard deviations for obesity, weight change, and the other cardiovascular disease risk factors for the twins as individuals are presented in Table 1.

Within monozygotic twin pairs, heavier twins had significantly higher levels of systolic and diastolic blood pressures, glucose (one-hour, post-load), total cholesterol, LDL-cholesterol, and triglycerides and lower levels of HDL-cholesterol than their genetically identical, lighter-weight brothers (Table 2). Regression coefficients measuring the association of obesity with each risk factor are indicated in Table 3. Examination BMI was significantly associated with all the cardiovascular disease risk factors in the unmatched analyses, independently of age. Matched-pair regression analyses revealed even stronger, significant, cross-sectional relationships between BMI and the cardiovascular disease risk factors. Only the regression coefficient for HDL-cholesterol was somewhat reduced.

Obesity was then partitioned into two variables—BMI at military induction (average age 20 years), and change in BMI during the 20 to 34 years between the induction and study examinations. The independent influences of the two obesity parameters on the other cardiovascular disease risk factors are indicated in Table 4. In both the unmatched and matched analyses, weight gain during adulthood was significantly associated with all the cardiovascular disease risk factors measured. In addition, induction BMI was independently and significantly related to systolic and diastolic blood pressures, total cholesterol, and triglycerides in the matched analyses. The negative regression coefficient between change in BMI and HDL-cholesterol was the only coefficient smaller in the matched analysis than in the unmatched analysis; the inverse relationship between induction BMI and HDL-cholesterol in the unmatched analysis was reduced and no longer statistically significant in the matched analysis. The proportion of nongenetic variance explained by both measures of obesity in the matched analyses ranged from 4 percent for HDL-cholesterol to 10 percent for total cholesterol, 11 percent for systolic blood pressure, and 13 percent for diastolic blood pressure and triglycerides.

TABLE 1—Obesity Measures at Military Induction and at Study Examination and Cardiovascular Disease Risk Factors at Study Examination (1969–73) in a Sample of White, Male, Monozygotic Twins

Variables	Number of Individuals	Mean ± Standard Deviation
Body mass index (kg/m²)		
at military induction	499	21.9 ± 2.4
at study examination	499	25.7 ± 3.2
change between induction and examination	498	3.8 ± 2.9
Age at study examination (years)	500	47.8 ± 3.1
Systolic blood pressure (mmHg)	439	127.0 ± 16.3
Diastolic blood pressure (mmHg)	439	81.3 ± 10.2
One-hour, post-load glucose (mg/dl)	465	162.6 ± 51.9
Total cholesterol (mg/dl)	491	219.7 ± 35.0
HDL-cholesterol (mg/dl)	486	44.2 ± 12.8
LDL-cholesterol (mg/dl)	485	143.5 ± 33.5
Triglycerides (mg/dl)	490	136.1 ± 89.2

TABLE 2—Mean Levels of Cardiovascular Disease Risk Factors at Ages 42–55 Years Comparing Heavier and Lighter Brothers within Monozygotic Twin Pairs

Variables	Heavier Twin	Lighter Twin	Mean Intrapair Difference (95% CI)
Body mass index (kg/m²)	26.6	24.8	1.8 (1.6, 2.0)
Systolic blood pressure (mmHg)	129.6	124.5	5.1 (3.1, 7.1)
Diastolic blood pressure (mmHg)	82.7	80.0	2.7 (1.4, 3.9)
One-hour, post-load glucose (mg/dl)*	161.7	156.0	5.6 (0.3, 11.2)
Total cholesterol (mg/dl)*	222.6	217.1	5.5 (1.9, 9.1)
HDL-cholesterol (mg/dl)*	43.1	45.3	−2.2 (−3.5, −1.0)
LDL-cholesterol (mg/dl)*	145.7	141.6	4.2 (0.8, 7.5)
Triglycerides (mg/dl)*	147.6	124.9	22.7 (12.5, 33.0)

*Conversion factor to SI Units in mmol/L:
Glucose—0.05551
Cholesterol (total, HDL, LDL)—0.02586
Triglycerides—0.01129

TABLE 3—Regression Coefficients* (± standard errors) for Cross-sectional Relationships between Body Mass Index (BMI) and other Cardiovascular Disease Risk Factors in Middle-Aged, White, Male Monozygotic Twins

Dependent Variables	Unmatched Analysis	Matched Analysis
Systolic blood pressure	1.47 ± 0.28	2.23 ± 0.43
Diastolic blood pressure	1.06 ± 0.17	1.44 ± 0.26
One-hour, post-load glucose	3.95 ± 0.80	4.79 ± 1.22
Total cholesterol	2.43 ± 0.51	3.88 ± 0.74
HDL-cholesterol	−0.97 ± 0.18	−0.83 ± 0.27
LDL-cholesterol	1.13 ± 0.49	2.37 ± 0.71
Ln triglycerides	0.065 ± 0.008	0.073 ± 0.013

*Coefficients are age-adjusted: age was included in the unmatched regression models, but was not related significantly to any outcome; matched co-twin analyses control for age in that co-twins are perfectly matched on age.

Discussion

In response to the recent emphasis on the importance of genes in influencing adult obesity,[13–15] we used data from identical twins to address the following question: do nongenetic differences in obesity contribute to levels of blood pressure, glucose intolerance, and lipids? The results of our analyses indicate that significant associations exist between obesity and many cardiovascular disease risk factors entirely in the absence of genetic influences. These results were obtained from both cross-sectional analyses of obesity and concurrent risk factor status and longitudinal analyses of change in weight during adulthood and risk factor status during middle-age. Furthermore, because matched-pair analyses also control for any factors that are identical or very similar between co-twins, such as the influences of shared early environment and culture, the observed associations

TABLE 4—Regression Coefficients* (±standard errors) for Longitudinal Relationships between Cardiovascular Disease Risk Factors and Body Mass Index (BMI) from Military Induction to Middle-age in White, Male, Monozygotic Twins

Dependent Variables	Unmatched Analysis		Matched Analysis	
	Change in BMI	Induction BMI	Change in BMI	Induction BMI
Systolic blood pressure	1.66 ± 0.31	1.06 ± 0.39	2.27 ± 0.44	1.93 ± 0.79
Diastolic blood pressure	1.18 ± 0.19	0.79 ± 0.24	1.48 ± 0.26	1.09 ± 0.47
One-hour, post-load glucose	4.60 ± 0.87	2.19 ± 1.21	4.99 ± 1.25	3.22 ± 2.29
Total cholesterol	2.83 ± 0.55	1.33 ± 0.77	3.89 ± 0.76	3.80 ± 1.37
HDL-cholesterol	−0.97 ± 0.20	−0.97 ± 0.28	−0.84 ± 0.27	−0.75 ± 0.50
LDL-cholesterol	1.34 ± 0.54	0.54 ± 0.76	2.35 ± 0.72	2.55 ± 1.33
Ln triglycerides	0.075 ± 0.008	0.041 ± 0.011	0.075 ± 0.013	0.057 ± 0.023

*Coefficients are age-adjusted: age was included in the unmatched regression models, but was not related significantly to any outcome; matched co-twin analyses control for age in that co-twins are perfectly matched on age.

may be attributed more specifically to environmental exposures or personal behaviors of adulthood.

These results do not preclude genetic influences on obesity, blood pressure, glucose tolerance, or lipid levels.[5,16] Nor do they reveal the specific, nongenetic mechanisms underlying the associations of cardiovascular disease risk factors with obesity. Obesity may serve as a marker of an atherogenic lifestyle, or it may contribute directly to the increases in blood pressure, glucose intolerance, and lipid levels through mechanical or biochemical pathways. Regardless of the mechanisms involved, the effects observed here are not pleiotropic effects of genes regulating adult fatness. The generalizability of these findings is supported by the comparability of the twins participating in this study to White men in the general population[17–19] and to participants in similar studies.[20–23]

Weight change during adulthood was generally more important than baseline BMI at military induction in predicting cardiovascular disease risk factor status in the longitudinal analyses. However, for blood pressure, total cholesterol, and triglycerides, BMI in early adulthood also contributed independently to risk factor status in middle-age. When analyses for all cardiovascular disease risk factors were conducted including both examination BMI (middle-age) and induction BMI (early adulthood) in the models, only the examination BMI was statistically significant (data not shown). These results suggest that the attained level of obesity is important in elevating a person's risk for diabetes and cardiovascular disease, although duration of obesity may also play an independent role.

Although the influence of obesity on the other cardiovascular disease risk factors appears to be limited to less than 15 percent of the nongenetic variability of each risk factor, a significant, if moderate, effect of weight gain during adulthood was observed on each risk factor. The average man in our cohort was 69 inches tall and gained 28 pounds between age 20 and age 48. By middle-age, he had higher levels of each of the cardiovascular disease risk factors studied, except for HDL-cholesterol, than men who gained less weight. Associated with his weight gain were excesses of 9.5 mmHg systolic blood pressure, 6.2 mmHg diastolic blood pressure, 21.0 mg/dl glucose (one-hour, post-load), 16.4 mg/dl total cholesterol, 9.8 mg/dl LDL-cholesterol, and a decrease of 3.5 mg/dl HDL-cholesterol. Similarly, his triglyceride level was 40 percent higher than that of men who did not gain weight between military induction and middle-age; this represents roughly a 50 mg/dl excess. These excesses occurred independent of initial BMI and can be attributed solely to nongenetic influences on the basis of the matched co-twin analysis (Table 4). The cumulative impact of similar or larger weight gains on multiple risk factors clearly contributes significantly to an increase in cardiovascular disease risk.

The causes of adult obesity are complex and undoubtedly involve both genetic and nongenetic influences.[24,25] Bouchard, *et al*,[26] have shown that monozygotic twins generally gain similar amounts of weight as a result of similar diets that include excess calories. However, unrelated twin pairs respond very differently to the same diet. This interpair variability is a reflection of gene-environment interaction, where an individual's response depends both on genetic predisposition as well as specific environmental exposure. Moreover, BMI is only a surrogate measure of obesity and may not capture the most relevant aspect related to cardiovascular disease risk. Unfortunately, more detailed measures of total body fat or fat distribution were not available from the induction or 1969–73 study examinations.

Despite the etiologic complexity of adult obesity, and the well-documented difficulty in its control, our findings provide some reason for optimism. They indicate that behaviors and environmental exposures that occur later in life are responsible, in part, for the association between adult obesity and cardiovascular disease risk factor status. They emphasize the importance of attained weight in influencing that risk, thereby suggesting that weight reduction will be effective even at later ages, when most individuals tend to gain weight. Thus, they confirm the clinical impression that weight control and prevention of obesity are likely to be successful in reducing a person's risk of cardiovascular disease and diabetes.

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$3 Million Awarded to Public Health Schools

Almost $3 million in fiscal 1990 capitation grant funds were awarded by the Health Resources and Services Administration to 24 schools of public health.

The funds represent general institutional support which a school may expend for any purpose it judges will most effectively advance its educational program.

The grant recipients and the amounts awarded are:

- University of Alabama at Birmingham, $81,448;
- University of California, Berkeley, $172,670;
- Loma Linda University, California, $131,946;
- University of California, Los Angeles, $206,553;
- San Diego State University, California, $72,000;
- Yale University, New Haven, CT, $77,865;
- University of South Florida, Tampa, $60,272;
- University of Hawaii, Honolulu, $94,154;
- University of Illinois, Chicago, $71,674;
- Tulane University, New Orleans, LA, $124,127;
- Johns Hopkins University, Baltimore, MD, $223,494;
- University of Massachusetts, Amherst, $74,607;
- Boston University, Massachusetts, $76,887;
- Harvard University, Boston, MA, $152,471;
- University of Michigan, Ann Arbor, $224,472;
- University of Minnesota, St. Paul, $107,512;
- Columbia University, New York, NY, $127,059;
- University of North Carolina, Chapel Hill, $201,992;
- University of Pittsburgh, PA, $105,883;
- University of Puerto Rico, San Juan, $120,543;
- University of Oklahoma, Oklahoma City, $86,335;
- University of South Carolina, Columbia, $87,313;
- University of Texas, Houston, $155,729; and
- University of Washington, Seattle, $79,494.

More information is available from the Division of Associated and Dental Health Professions, Bureau of Health Professions, Room 8-101, 5600 Fishers Lane, Rockville, MD 20857, tel: 301-443-6853.

The Differential Effect of Traditional Risk Factors on Infant Birthweight among Blacks and Whites in Chicago

JAMES W. COLLINS, JR., MD, MPH, AND RICHARD J. DAVID, MD

Abstract: We analyzed 103,072 White and Black births in Chicago from the 1982 and 1983 Illinois vital records, using 1980 median family income of mother's census tract as an ecologic variable. Thirty-one percent of Blacks and 4 percent of Whites resided in census tracts with median family incomes ≤$10,000/year. Only 2 percent of Black mothers, compared to 16 percent of White mothers, lived in areas where the median family income was greater than $25,000/year. Among Blacks with incomes ≤$10,000/year, maternal age, education, and marital status had minimal predictive power on the incidence of low birthweight (LBW) infants. Among high-risk mothers in the poorest areas the proportion of LBW infants in Blacks and Whites was less divergent than in higher income areas. Independent of residential area, low-risk Whites had half the occurrence of LBW infants as Blacks. We conclude that the extremes of residential environments show dramatic racial disparity in prevalence, yet the few low-risk Blacks still do less well than low-risk Whites. Traditional risk factors do not completely explain racial differences in neonatal outcome. (*Am J Public Health* 1990; 80:679–681.)

Introduction

Despite the dramatic improvements in perinatal care during the past 30 years, Black infants are twice as likely as White infants to die during their first month of life.[1–3] This is primarily related to the high incidence of low birthweight (<2500 grams) infants among Black mothers.[4–6] Cities such as Washington, DC, Detroit, and Chicago with large Black subpopulations have the highest neonatal mortality rates (NMR) in the country and are in part responsible for the United States' relatively poor international ranking in neonatal survival.[7] The mechanisms underlying this well-documented phenomenon are not well understood.

Low maternal socioeconomic status (SES) worsens pregnancy outcome.[8,9] Black mothers are more likely to be young, unmarried, poorly educated, and receiving welfare support.[8] However, Spurlock, *et al*,[10] found that poor and non-poor Black infants in Kentucky had no significant difference in birthweight distribution. There is limited available information on mothers of high SES, especially among ethnic groups with widespread poverty. Shiono, *et al*,[5] reported that in a middle class community Black mothers still had a high incidence of LBW infants. Similarly, Kleinman and Kessel[11] found that the ratio of low birthweight for Blacks versus Whites was actually higher among college educated mothers than for women who did not complete high school. Another recent study indicated that a 5 percent racial difference in low birthweight persisted independent of maternal residence.[12] Few studies have addressed the possibility that mature, educated, Black mothers who reside in high income communities might be less likely to deliver low birthweight infants.

For these reasons, we utilized Illinois vital records and US census income data to determine the extent to which disparities in traditional risk factors affect racial differences in pregnancy outcome in Chicago. We particularly wanted to learn if low maternal risk status reduces the number of low birthweight Black infants.

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Methods

We analyzed all Black and White births in Chicago using a data set created by appending survival data from infant birth-death tapes as previously described[13] and income characteristics of mother's census tract (from 1980 census tapes) to records in the 1982 and 1983 birth files. Because of their strong association with neonatal outcome maternal age, education, and marital status were used as proxies of individual risk status. Median family income of mother's census tract residence was used as the primary proxy for environmental deterioration. Percent of families within each census tract living below the poverty level was used as an additional ecologic variable.

Among Blacks and Whites we calculated the proportion (per 100 live births) of LBW infants by census tract income, maternal age, and education for the entire study population. We stratified the study population by median family income of mother's census tract to examine the Black-White differential in residential environments. Within each grouped income area, we calculated the frequency of individual and ecologic risk variables. To explore the independent association of residential environment and pregnancy outcome, we determined the relation of maternal age, education, marital status, parity, and history of prior infant death to Black and White infant birth weight.

Relative risk with 95% confidence intervals are presented. Confidence limits were estimated by the Taylor Series method.[14]

Results

There were 51,827 Black and 51,245 White births in Chicago during 1982 and 1983. Black neonatal mortality was twice as high as that of Whites (16/1,000 vs 7/1,000) and the LBW proportions were twice as high in Blacks (14 percent vs. 6 percent). Only 2 percent of Black mothers, compared to 16 percent of White mothers, resided in census tracts in which the median family income was greater than $25,000/year. Conversely, 31 percent of Blacks lived in census tracts in which the average household income was less than $10,000/year; only 4 percent of White mothers resided in such impoverished neighborhoods (Figure 1).

The risk of LBW infants among Blacks remained essentially twice as high as that of Whites across all maternal income, education, and age groups. Although the higher

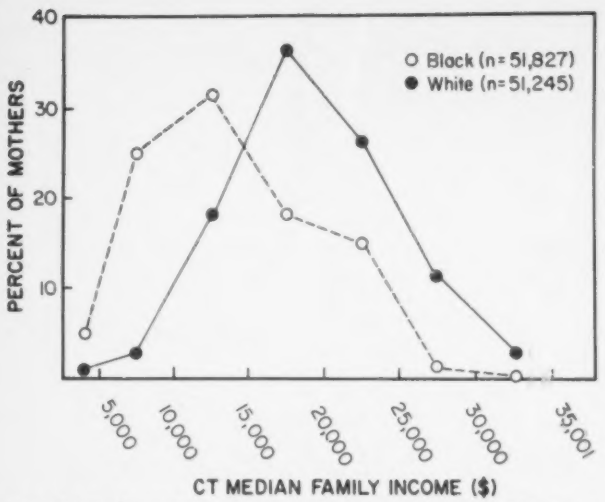

FIGURE 1—Census Tract Median Family Income Distribution of Black and White Mothers

Black risk appeared to be eliminated at very high incomes, the confidence intervals were broad because of the small number of Blacks (N = 200, or 0.4 percent) residing in census tracts with median family income over $40,000/year (Table 1).

Table 2 illustrates the racial distribution of risk factors by census tract income. White mothers were often Hispanic in low-income areas. Among mothers who resided in very low-income census tracts (≤$10,000/year) Blacks were almost 3.5-times as likely as Whites to reside in a census tract where more than one-half of the census tract cohabitants lived below the federally mandated poverty level (RR = 3.47, 95% CI 3.07, 3.92).

The differential effect of race on the LBW rates in low-income areas is apparent with maternal age. Among Black mothers who resided in higher income communities, the high proportion of LBW was minimally reduced and remained twice that of Whites independent of maternal education, age, and marital status (Table 3).

TABLE 1—Proportion of Low Birthweight (<2500 grams) and Relative Risk in Black and White Infants according to Income, Maternal Education, and Age

Variables	% LBW		Black Relative Risk (95% CI)
	Black	White	
Income (per year)			
≤$10,000	15	8	1.92 (1.64, 2.26)
$10,001–$20,000	14	6	2.12 (2.01, 2.24)
$20,001–$30,000	12	6	2.16 (1.99, 2.39)
$30,001–$40,000	10	5	2.26 (1.39, 3.54)
>$40,000	4	4	.98 (.13, 7.29)
Maternal Education (years)			
<12	16	7	2.40 (2.26, 2.55)
12	14	7	2.13 (1.93, 2.42)
13–15	12	5	2.14 (1.93, 2.42)
16	10	5	2.13 (1.76, 2.57)
≥17	9	5	1.87 (1.38, 2.53)
Maternal Age (years)			
≤19	14	8	1.44 (1.37, 1.55)
20–35	14	6	2.41 (2.30, 2.52)
>35	14	8	1.98 (1.64, 2.39)

TABLE 2—Distribution of Individual and Environmental Risk Factors by Census Tract Average Annual Income and Race

Variables	≤$10,000		$10,001–$20,000		$20,001–$30,000	
	Black	White	Black	White	Black	White
Age (years) % ≤19	34	19	27	3	22	8
Education (years) % <12	54	55	38	60	23	21
Marital Status % Unmarried	82	36	71	88	59	12
Maternal Ethnic Origin % Hispanic	<1	25	<1	23	<1	2
Poverty Level*	43	13	1	0	0	0

*Incidence rate of residing in a census tract in which more than 50% of families have annual incomes below the federally mandated poverty level.

Discussion

We found that high-risk Whites and Blacks had less divergent LBW rates in the poorest areas than in higher income areas while low-risk Whites had half the occurrence of LBW infants as Blacks regardless of the income of the area in which they lived. In Chicago, the percentages of Black and White mothers at the extremes of residential environment are dramatically different. Contrary to Spurlock, *et al*,[10] who reported that poor and non-poor Black infants in Kentucky had no difference in the incidence of LBW, we found that among all mothers, Black and White, low income is associated with a greater risk of low birthweight.

A wider Black-White gap at the upper end of the socioeconomic spectrum has been described in earlier studies such as that by Kleinman and Kessel,[11] but this could have been an artifact of the differences in the economic value of education (the usual proxy for income) between Blacks and Whites in the US. For example, the income disparity between college educated Blacks and Whites is greater than it is for those who did not finish high school.[13] Notwithstanding the limitations of the available grouped income data, our results confirm the results of Kleinman and Kessel and highlight the difficulty in defining a truly low-risk group of Black mothers using traditional risk factors.

Our data suggest that residential environment is an important risk factor that researchers need to take into account when examining the relation between race and neonatal outcome. Residing in a very low-income urban neighborhood is such a strong proxy of low birthweight for Blacks that traditional indicators of favorable outcome (education, age, marital status) fail to identify clearly a low risk subgroup. The intense concentration of extreme poverty combined with the related issues of disintegrating social networks, substance abuse, poor nutrition, smoking, and inadequate prenatal care may produce such a powerful negative force that isolated changes in the classical risk factors do not dramatically reduce the high percentage of low birthweight infants.

Racial differences in intrauterine growth rates exist.[16,17] However, when Wilcox and Russell controlled for Black-White disparities in birthweight distribution, the excess in mortality among Black infants was not eliminated.[17] The study found that a high incidence of small births among Blacks accounted for the majority of Black-White differences in survival.

The mechanisms of racial disparity in health in this

TABLE 3—Proportion of Low Birthweight (<2500 grams) Infants by Census Tract Average Annual Income and Race

Variables	<$10,000			$10,001–$20,000			$20,001–$30,000		
	Black (N = 15,889)	White (N = 1,851)	Black RR (95% CI)	Black (N = 27,754)	White (N = 27,867)	Black RR (95% CI)	Black (N = 8,936)	White (N = 20,199)	Black RR (95% CI)
					%LBW				
Maternal Age (years)									
≤19	14	13	1.02 (.78, 1.35)	14	8	1.72 (1.53, 1.93)	13	5	2.70 (2.15, 3.40)
20–35	16	7	2.40 (1.97, 2.93)	14	6	2.24 (2.10, 2.39)	12	6	2.20 (2.02, 2.41)
Maternal Education (years)									
≤12	16	9	1.85 (1.56, 2.21)	15	7	2.08 (1.97, 2.21)	14	6	2.21 (2.01, 2.43)
>12	12	5	2.43 (1.54, 3.82)	11	5	2.08 (1.80, 2.40)	11	5	2.14 (1.87, 2.46)
Marital Status									
Unmarried	16	11	1.20 (.96, 1.44)	15	9	1.76 (1.51, 2.05)	14	8	1.75 (1.51, 2.05)
Married	14	6	2.29 (1.80, 2.40)	11	6	2.00 (1.84, 2.18)	10	5	1.87 (1.67, 2.09)
Parity*									
Primaparity	13	8	1.64 (1.27, 2.12)	9	7	1.27 (1.14, 1.41)	12	6	1.99 (1.75, 2.74)
Low parity	14	6	2.44 (1.83, 3.25)	14	6	2.26 (2.11, 2.47)	13	5	2.62 (2.35, 2.93)
High parity	18	9	2.08 (1.53, 2.84)	18	8	2.39 (2.17, 2.64)	15	6	2.31 (1.94, 2.20)
Prior Infant Death									
Yes	22	9	6.37 (1.62, 25.10)	22	8	2.31 (1.82, 2.95)	25	5	3.94 (2.47, 6.89)
No	15	8	1.78 (1.52, 2.09)	14	6	2.15 (2.04, 2.27)	13	4	3.40 (3.11, 3.73)

*Parity was defined as high in third or higher numbered births to women under 25 years of age and fourth or higher numbered births to women 25 to 29 years of age. All other multiparous births were considered to represent low parity.[11]

society are complex and deep-rooted.[8,18] We suspect that the persistently high rate of low birthweight infants among Blacks reflects generations of poverty.[19] While the vital records-based studies can identify factors that affect perinatal outcome, there is a need for studies in greater depth. A limitation of the present study is that even with household income data for each birth we could not truly have "controlled" for Black-White economic differences since income cannot be equated with purchasing power.[20] Moreover, despite treating census tract income as an indicator of the environment within which people live, the lack of homogeneity among mothers of the same grouped income area limits the generalizability of the study.[21] A multidisciplinary approach is needed to better elucidate and remove causes of Black-White differences in neonatal outcome.

In summary, the extremes of residential environment show dramatic racial disparity in prevalence, yet traditional risk factors fail to explain why non-poor Blacks remain at high risk for poor pregnancy outcome. Given that a large percentage of Blacks in this country live at or below the poverty level we need to eliminate economic disparities between the races in addition to providing planned intervention if we hope to see the health gap closed. We suspect that it may require more than one generation of non-poverty and adequate services to see an impact upon infant birthweight.[19]

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The Dramatic Increase in the Rate of Low Birthweight in New York City: An Aggregate Time-Series Analysis

THEODORE JOYCE, PHD

Abstract: This paper uses monthly time-series data from 1968 through 1988 to test whether the observed increase in the incidence of low birthweight in New York City represents a reversal of the downward trend that existed prior to 1984. We find that the trend has shifted for both Whites and Blacks. We estimate that between July 1984 and December 1988 there were 3,110 additional low-birthweight births to Blacks and 1,385 additional low-birthweight births to Whites above what would have been expected had the rate of low birthweight not shifted upwards. The incremental costs of treating these infants in their first year of life is estimated at over $22,000,000 in 1986 dollars. (*Am J Public Health* 1990; 80:682–684.)

Introduction

The proportion of low birthweight births among Blacks in New York City has increased dramatically since 1984 and less dramatically among Whites (see Figure 1). This upturn represents a radical reversal of the downward trend in the low birthweight rate that had existed since 1968. Between 1968 and 1984 the average monthly percentage of low-birthweight births among Blacks in New York City fell from 13.9 to 10.1; by 1988, the average monthly rate had risen to 12.9. Given the present rate of increase, the rate of low birthweight among Blacks in 1990 will exceed the rate that existed 22 years earlier. This paper applies several statistical methods to the 21 years of monthly, race-specific data from New York City to measure the trend reversal in the rate of low birthweight since 1984. It also estimates the financial costs of such an increase in the incidence of low birthweight.

Methods

Data

The data on low birthweight are from micro-level vital statistics for New York City. Each year of data has been aggregated by month for Blacks and Whites separately from January 1968 through December 1988 (N=252). The data for Whites contain a large component which is Hispanic because ethnicity was not identified on New York City birth certificates until 1978. Moreover, the proportion of births to White women of Hispanic origin has increased over time. In 1978, for instance, 28 percent of all births in New York City were to women of Hispanic descent and 34 percent of all births were to White non-Hispanic women. By 1987, the Hispanic portion had risen to 30 percent while the White non-Hispanic portion had fallen to 29 percent. The implications of a shift in the ethnic distribution of births among Whites are discussed below.

New York City averaged approximately 100,000 resident live births per annum over the time span under study. The percentage of births to Whites and Blacks has varied between 69 percent White and 29 percent Black in 1970 to 57 percent White and 36 percent Black in 1987. Between 1968 and 1988 there were approximately 6,000 White births and 3,000 Black births per month.

Statistical Methods

Two approaches are used to illustrate the changes that occurred in the incidence of low birthweight in 1984. First, the race-specific rate of low birthweight is regressed on a constant, a linear trend term, a dummy variable that equals zero prior to July 1984 and one thereafter, and an interaction trend term. The interaction term is the product of the linear trend term and the dummy variable. An additional set of 11 dummies is included to control for seasonality. A negative coefficient on the trend term and a positive coefficient on the interaction term would be evidence that the downward trend in the rate of low birthweight prior to July 1984 had turned upwards after that point.[2] The sensitivity of the regression estimates are tested by re-estimating the model with January 1984 and January 1985 as the point at which the trend is assumed to have changed.

Another means of illustrating how the low birthweight series have changed involves the use of Box-Jenkins techniques.[3,4] Specifically, an autoregressive-integrated-moving-average model (ARIMA) is fit to each of the series from January 1968 through July 1984. The fitted model is used to project the rate of low birthweight that would have been observed if the underlying process which generated the observed data between 1968 and 1984 had not been altered. The projected rate of low birthweight is compared with the actual rate in order to approximate the additional number of

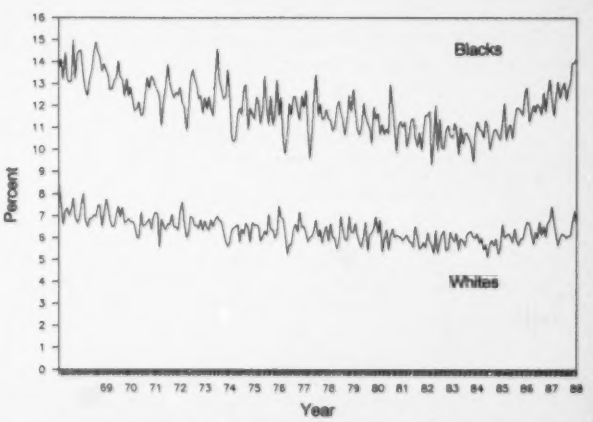

FIGURE 1—Percent of Low-Birthweight Births among Blacks and Whites, New York City, 1968–88

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low-birthweight births that have occurred as a result of the shift in the trend. The additional number of low-birthweight births are multiplied by national estimates on the short-term and long-term costs of low birthweight in order to estimate the financial burden of the increase in low birthweight.

Results

Estimates from the linear regression are presented in Table 1. For Whites and Blacks the downward trend in the rate of low birthweight became positive after July 1984 as evidenced by the negative coefficient on the trend term and the positive coefficient on the interaction trend term. The results are insensitive to the switching point of the interaction term from July 1984 to either January 1984 or January 1985 (additional regressions not shown).*

We fit ARIMA models to the low birthweight series based on data from January 1968 through June 1984. To eliminate seasonality, a twelfth-order difference was applied to each series; in the case of Blacks, a first-order difference transformation was also needed to achieve stationarity. Based on the autocorrelation and the partial autocorrelation functions, the low-birthweight series for Blacks can be characterized as a second-order moving average with a first-order seasonal moving average. The series for Whites can be characterized by a first-order seasonal moving average. Estimates of the ARIMA models are presented in Table 2. The Ljung-Box Q-statistic supports the adequacy of the model.

The estimated ARIMA models were used to predict the rate of low birthweight for each race from July 1984 through December 1988. A comparison of the actual rates and the projected rates is presented in Figure 2. What is particularly striking is the growing divergence between the actual and the projected low-birthweight rate among Blacks.

To underscore the magnitude of the change, we multiplied the projected rates of low birthweight from July 1984 through December 1988 by the actual number of live births over the same period. The product yields the number of low-birthweight births that would have been expected based on a 54-month forecast made in July 1984. The difference between the actual number of low-birthweight births and the projected number represents an estimate of the excess number of low-birthweight births above what would have been observed had the rate of low birthweight continued a realization consistent with the previous 16 years. For the entire 54-month period beginning in July 1984, we estimate that there have been 3,110 additional Black low-birthweight births and 1,385 White low-birthweight births.

To assess the financial burden of the additional low-birthweight births, we used cost estimates from the US Congress, Office of Technology Assessment.[5] The figures represent the incremental costs of a low-birthweight birth; that is, the costs above those associated with a normal-weight delivery. The costs are broken down into three categories: initial hospitalization, rehospitalization, and long-term treatments. To simplify matters, we grouped initial hospitalization and rehospitalization under the rubric of short-term costs. Long-term costs refer to expenses beyond the first year of life. The report found that, on average, the short-term costs of a low-birthweight birth ranged from a low of $5,040 to a high of $7,525 in 1986 dollars. If long-term costs were added, the total incremental costs varied from $14,040 to $30,525 per low-birthweight birth.

We focus on the short-term costs since these expenses have already been incurred. For Blacks, the total short-term costs associated with the excess number of low-birthweight births range from a low of $15,674,400 to a high of $23,402,750 in 1986 dollars. For Whites, the comparable costs are between $6,980,400 and $10,422,125.

Discussion

The increase in substance abuse is the most likely explanation for the increase in the incidence of low birthweight. New York City birth certificates reveal that between 1981 and 1987 the number of infants born to drug-abusing mothers rose from 6.7 per 1000 live births to 20.3 per 1000 live births, a tripling of the rate.** The extent of the underreporting is potentially serious, but the point is unlikely to change: the number of infants exposed to drugs has increased precipitously over the last four years. Moreover, there is a growing body of epidemiological evidence which links the use of cocaine during pregnancy to an elevated risk of low birthweight.[6-8] Finally, no other risk factors reported on birth certificates appear to have undergone sufficient change to account for the increased incidence of low birthweight. Annual data show little variation in the percentage of births to women with late or no prenatal care, the percentage of births born out-of-wedlock, and the percentage of births to women on Medicaid.[1]

The increase in the incidence of low birthweight has had relatively little impact on the neonatal mortality rate. Among Black non-Hispanics, for example, the neonatal mortality rate stood at 13.6 in 1983, 11.8 in 1984, 13.6 in 1985, 12.3 is 1986, and 13.4 in 1987.[1] The provisional figure for 1988 is 14.6.*** Among White non-Hispanics, Puerto Ricans, and other Hispanics, the neonatal mortality rate has either fallen slightly or remained essentially unchanged since 1983. The mortality rates imply that many of the additional low-

*A more detailed discussion of the regression results is available upon request to author.

TABLE 1—Estimated Piecewise Linear Regressions of the Rate of Low Birthweight on a Linear Trend Term: Blacks and Whites, 1968,1–1988,12[a]

	Coefficient	Standard Error	P-value
Blacks			
Constant	13.689	.157	.0001
Linear trend	−.016	.001	.0001
Dummy (1 if T > = July 1984)	−14.652	1.718	.0001
Trend × Dummy	.072	.008	.0001
Adjusted R^2	.70		
Whites			
Constant	7.165	.075	.0001
Linear trend	−.007	.001	.0001
Dummy (1 if T > = July 1984)	−3.965	.770	.0001
Trend × Dummy	.020	.003	.0001
Adjusted R^2	.52		

[a] The seasonal dummy variables are not shown. The estimates have been corrected for first-order autocorrelation by means of the Corchrane-Orcutt procedure.[2]

**Habel L, Lee J, Kaye K: Trends in Maternal Drug Abuse during Pregnancy in New York City 1978–87. Presented at the 116th Annual Meeting of the American Public Health Association, Boston, MA, Nov. 13–17, 1988.

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TABLE 2—Estimated ARIMA Equations for the Rate of Low Birthweight Blacks and Whites 1969,2–1984,8

Series	General Form (p,d,q) × (P,D,Q)	constant	Parameters ϕ_1	ϕ_2	Φ_{12}	Q-statistic (df = 39)
Blacks	(0,1,2) × (0,1,1)		−.708 (.072)	−.247 (.072)	−.688 (.059)	33.65
Whites	(0,0,0) × (0,1,1)	−.068 (.010)			−.745 (.052)	43.58

[a] Standard errors are in parentheses. The probability value for the Q-statistic is .71 for Blacks and .23 for Whites.

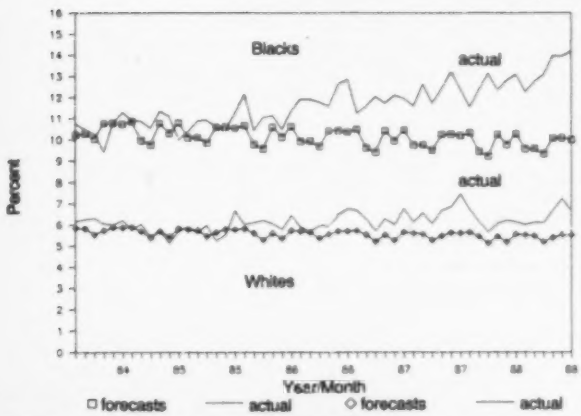

FIGURE 2—Percent of Low-Birthweight Births (forecast vs actual) among Blacks and Whites, New York City, July 1984–December 1988

birthweight infants survive the first month of life. However, the upturn among Blacks in 1988 suggests that the capability of the tertiary care system to manage the increase in low-birthweight births may be limited.

As mentioned above, the proportion of White births to women of Hispanic origin has risen over time. If the rate of low birthweight among Hispanics is higher than that of White non-Hispanic women, then the increase in the incidence of low birthweight among White women may simply reflect the change in the ethnic composition of White births. However, the increase in the proportion of White births to women of Hispanic descent has been dominated by an increase in non-Puerto Rican Hispanics. The incidence of low birthweight and neonatal mortality among this group of Hispanics is almost identical to that of White non-Hispanics.[1] Thus, the growth in the proportion of White births to women of Hispanic origin is not a good explanation for the increased incidence of low birthweight.

The lack of monthly data on substance abuse by pregnant women prevented a more direct test of whether the rise in the proportion of low-birthweight births is associated with the growth in substance abuse. Nevertheless, the increase in the aggregate rate of low birthweight in a city the size of New York is an important, yet troubling finding. At the very least it indicates a deterioration in the health of newborns in New York City. Moreover, anecdotal evidence from the daily media suggests that the use of cocaine, or its derivative crack, among pregnant women is a worsening problem in cities across the United States. If substance abuse is the causal factor behind the unprecedented rise in the incidence of low birthweight in New York City, then one can expect similar increases in low birthweight in other parts of the country. Given the costs of treating low-birthweight children, action taken now may be highly cost-effective.

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Secular Change in Birthweight among Southeast Asian Immigrants to the United States

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Abstract: We analyzed the Washington State births in 1980–1986 to Southeast Asian parents whose birth place was coded as being outside of the US. The mean birthweight increased from 3188 grams in 1980–81 to 3283 grams in 1986, an annual increment of about 18 grams (95% CI = 11, 25 grams). The prevalence of low birthweight (<2500 grams) decreased from 7.2 percent in 1980–81 to 5.4 percent in 1986, an annual reduction of 6.4 percent (95% CI = 1.3, 11.2 percent). In an analysis of information ascertained from the birth certificate, the change of paternal occupational status (from student to employed) was associated with 27 percent of the reduction in the prevalence of low birthweight, independent of maternal age, infant sex, and prior gravidity. A similar temporal change of birthweight during the same period of time was not observed among infants of US-born Asian mothers. Among the women who had two consecutive births from 1984 to 1986, the improvement of birthweight for the second birth compared to the first birth was much greater in infants born to the Southeast Asian mothers than that to White mothers. Our study suggests that living in the United States after immigration from their native countries has had a positive impact on the birthweight of infants born to Southeast Asian immigrants. (*Am J Public Health* 1990; 80:685–688.)

Introduction

Since the mid 1970s, approximately 800,000 Southeast Asian refugees have immigrated to the United States.[1] At the time of their arrival, the majority of these refugees were of lower socioeconomic status than the general population of the US, and had undergone severe hardships.[2] Health assessment by local and federal agencies revealed several serious problems among this population, including nutritional deprivation, anemia, and psychiatric disorders.[3–7] In addition, it has been reported that they lacked adequate prenatal care and were at high obstetric risk.[8] Since their arrival, however, several programs have been established to help these recent immigrants with financial support, education, and health care.[2] This study attempted to evaluate whether or not the risk of low birthweight has changed over time among Southeast Asian immigrants and, if so, to determine what factors may have accounted for any change observed.

Methods

Washington State contains one of the largest populations of Southeast Asian refugees[2]; the peak wave of the immigrants occurred in the late 1970s and early 1980s.* All births between 1980 and 1986 to Washington State residents (both mother and father) whose race was coded as "other Asian" (i.e., Asian but not Chinese, Japanese, or Filipino) and for whom place of birth was coded as "outside US" were selected from computerized files of Washington State Birth Certificates. We reviewed the original certificates of a random 5 percent sample of the selected births for each year to estimate the proportion of those births to parents who were truly born in Southeast Asia. About 70 percent were born to parents from Southeast Asian countries, primarily Vietnam, Laos, and Cambodia, while the majority of the remainder were born to parents from Korea, India, and the Pacific Islands. The following information on each birth available from the file of birth certificates was used in this study: birthweight, maternal age, marital status, gravidity, parity, sex of infant, the number of prenatal visits, the month of first prenatal visit during pregnancy, method of delivery, pregnancy and labor complications, malformation status, mother's smoking status during the pregnancy (ever vs never, available only for the years 1984–86), number of prior spontaneous and induced abortions, and mother's and father's occupations.

The infants born in 1980 and 1981 were taken as the reference cohort that presumably reflected the situation shortly after the immigrants initially arrived in the United States. Births in 1982 to 1986 among this population thus comprised successive comparison cohorts, assuming that the parents of these infants were those that had been in the United States for progressively greater lengths of time. To evaluate whether or not any changes of birthweight in the study population were part of a general trend among births to all Asian parents in Washington State, we also analyzed the 770 births available during the study period to Washington residents (both parents) whose race was indicated as being a specific Asian ethnic group (i.e., Chinese, Japanese, Filipino) or other Asian, and for whom the place of birth was coded as any one of the 50 US States or the District of Columbia.

Average annual changes in the proportion of low birthweight (<2500 grams) and high birthweight (≥3500) were estimated using logistic regression for dichotomous outcomes,[9] while corresponding changes in mean birthweight were estimated using multiple linear regression for continuous outcome.[10] The odds ratio was used as an approximation of the relative risk when dichotomous outcomes of birthweight were evaluated, i.e., the proportion of low birthweight and high birthweight. Although the proportion of the birthweight equal to or greater than 3500 grams is not a rare event (it occurred in about 20 to 30 percent of births in our study), the discrepancy between the odds ratio and relative risk for this outcome is practically negligible. To estimate the proportion of annual change in birthweight accounted for by specific demographic and obstetric factors, we determined the difference between the estimates of annual change

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*Department of Public Health, Refugee Health Service of Seattle-King County: Two-thirds of the Southeast Asian immigrants to Washington State reside in Seattle-King County.

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adjusted and unadjusted for the specific factors and divided this figure by the unadjusted estimates of annual change.

To examine more explicitly whether or not increased duration of residence in the US has influenced the distribution of birthweight among Southeast Asian immigrants, data were also analyzed on those Washington State parents who gave birth to two consecutive children during years 1984–87, and who met the same race and birthplace criteria as described above (data from 1980–83 were not available). Thus, the birthweight of the second child born during this period (not necessarily the second child ever born to each woman) was compared to that of preceding birth. In order to distinguish any secular change of birthweight among Southeast Asian immigrants from that among general population in the Washington State, we also analyzed White women who gave birth to two consecutive children during the period of 1984–87 (there were too few US-born Asian women for similar analyses of this type). The risks of low birthweight for Southeast Asian immigrants compared to American Whites were then calculated separately for each of the two births occurring during the years 1984–87. The change in low birthweight among Southeast Asian immigrants beyond the secular change in the general population in the State was computed by dividing the difference of odds ratio between two consecutive births by the odds ratio minus one for the first birth:

$$\frac{(OR_{\text{second birth}}) - (OR_{\text{first birth}})}{(OR_{\text{first birth}}) - 1}$$

Results

The prevalence of low birthweight (<2500 grams) decreased among Southeast Asian immigrants from 7.2 percent in 1980–81 to 5.4 percent in 1986, a reduction of almost 25 percent (RR = 0.75, 95% CI = 0.53, 1.03) (Table 1). During this same time period, the proportion of birthweight equal to or greater than 3500 grams increased from about 24 percent in 1980–81 to 30 percent in 1986, a 26 percent increment (RR = 1.26, 95% CI = 1.09, 1.45). The mean birthweight increased steadily from 3188 grams in 1980–81 to 3283 grams in 1986 with an average increase of 95 grams (95% CI = 62, 120 grams) during the six-year period (Table 1). A similar secular change of birthweight distribution was not observed among US-born Asian population (data available on request to authors).

The analyses of the data from those subjects with two consecutive births during 1984–87 revealed that the prevalence of low birthweight among Southeast Asian immigrants declined 55 percent (OR = 0.45, 95% CI = 0.23, 0.86) in the second birth compared to the preceding birth. Compared to Whites, there was a 92 percent [(1.05-1.61)/(1.61-1)] reduction, after adjustment for maternal age and parity, in the risk of low birthweight for the second birth compared to the preceding birth among Southeast Asian immigrants beyond the secular change among general population (Table 2).

Based on data available from the birth certificate (see Methods), we compared the temporal changes in factors considered *a priori* to be associated with birthweight to the changes of birthweight during the same period of time (Table 3). There were large increases during the study period in the proportion of women giving birth after age 30 and in the primary cesarean section rate. There was also a 60 percent decrease in the proportion of fathers who listed "student" as their occupation. Aspects of reproductive history, maternal marital status, level of prenatal care, and prevalence of cigarette smoking during pregnancy showed little or no variation between 1980–81 and 1986 although information on smoking during pregnancy was only available for the last three years.

From 1980–81 to 1986, there was a 6.4 percent annual reduction in the prevalence of low birthweight, a 5.8 percent annual increase in the proportion of high birthweight (≥3500 grams), and a 17.7 gram annual increment of mean birthweight among immigrants from Southeast Asia (Table 4). Temporal variation of maternal age, infant sex ratio, and prior gravidity explained between 17 and 28 percent of the secular change in birthweight (depending upon birthweight measure) and, beyond the explanatory power of these trends, the change in paternal student status accounted for an additional 16 to 27 percent of the change in birthweight. Together, maternal age, infant sex, gravidity, and paternal occupational status accounted for between 42 and 55 percent of the change in birthweight among births to immigrants from Southeast Asia, and inclusion of other aspects of obstetrical history, prenatal care, method of delivery, infant malformation status, and demographic characteristics in the models did not confound the above results or explain the remaining unexplained portion of the change in birthweight parameters.

Discussion

There are several limitations in our analyses. Most important, we did not review the more than 7,000 birth certificates to distinguish births to Southeast Asian immigrants from births to parents of other ethnic groups also categorized by Washington State as "other Asian." Our review of a random sample of these certificates indicates that if such a distinction were possible, about 70 percent of the study population would be classified as Southeast Asian in origin. However, our review of the birth certificates also

TABLE 1—Distribution of Birthweight (BW) among Immigrants from Southeast Asia, by Time Period, Washington State, 1980–86

Time Period (N)	BW < 2500 gms % (n)	BW ≥ 3500 gms % (n)	Mean BW grams (SE)
1980–81 (2094)	7.2 (150)	23.8 (498)	3188 (11)
1982 (1210)	7.6 (92)	25.8 (312)	3190 (15)
1983 (976)	5.8 (57)	29.2 (285)	3238 (16)
1984 (1008)	5.8 (58)	27.1 (273)	3241 (16)
1985 (1062)	5.9 (63)	28.0 (297)	3243 (15)
1986 (994)	5.4 (54)	30.0 (298)	3283 (17)

TABLE 2—Comparison of Low Birthweight (LBW) between two Consecutive Births among Southeast Asian Immigrants and American Whites, Washington State, 1984–87

	Proportion of LBW[a] (N)			
Birth Sequence	SE Asian % (N)	White % (N)	OR[b]	95% CI
First[c]	6.6 (423)	4.3 (21562)	1.61	1.1, 2.4
Second[d]	3.1 (425)	2.9 (21570)	1.05	0.6, 1.8

[a] <2500 grams.
[b] Adjusted for maternal age and parity.
[c] Three Southeast Asians and 69 Whites with missing data.
[d] One Southeast Asian and 61 Whites with missing data.

TABLE 3—Distribution (%) of Characteristics among Immigrants from Southeast Asia, by Time Period, Washington State, 1980–86

Factors	1980–81 (N = 2105)[a,b]	1982 (N = 1214)	1983 (N = 976)	1984 (N = 1015)	1985 (N = 1064)	1986 (N = 998)
Maternal age (years)						
<20	6.6 (139)	6.1 (74)	5.7 (56)	3.4 (35)	4.0 (43)	2.8 (28)
20–24	27.8 (584)	31.9 (386)	24.2 (236)	23.5 (239)	19.7 (210)	19.8 (197)
25–29	36.4 (764)	35.3 (428)	38.4 (375)	37.5 (381)	39.7 (422)	37.0 (369)
≥30	29.2 (614)	26.7 (323)	31.7 (309)	35.5 (360)	36.6 (389)	40.4 (403)
Infant sex						
Male	50.7 (1067)	50.5 (613)	54.2 (529)	49.1 (498)	52.5 (559)	53.4 (553)
Female	49.3 (1038)	49.5 (601)	45.8 (447)	50.9 (517)	47.5 (505)	46.6 (465)
Prior gravidity						
0	28.6 (589)	28.5 (342)	27.5 (263)	30.1 (295)	27.5 (282)	26.8 (259)
1–2	46.0 (948)	49.0 (589)	46.9 (448)	46.2 (452)	47.5 (488)	49.2 (476)
≥3	25.4 (524)	22.5 (270)	25.6 (245)	23.7 (232)	25.0 (257)	24.0 (232)
Primary cesarean section						
Yes	6.0 (127)	6.2 (75)	8.1 (79)	7.3 (74)	8.0 (85)	10.9 (109)
No	94.0 (1978)	93.8 (1139)	91.9 (897)	92.7 (941)	92.0 (979)	89.1 (889)
Paternal occupation						
Student	27.7 (563)	24.2 (290)	15.1 (144)	11.4 (114)	10.0 (103)	10.1 (100)
Other	72.7 (1471)	75.8 (910)	84.9 (811)	88.6 (882)	90.0 (922)	89.9 (887)

[a] Numbers in parentheses are number of live births.
[b] The number of subjects for an individual factor may not match the total number of subjects due to missing data.

TABLE 4—Annual Change in Birthweight among Immigrants from Southeast Asian, Washington State, 1980–86

Birthweight Measures	Crude	Adjustment 1[c]	Adjustment 2[d]
Percent change in the prevalence of low birthweight (<2500 grams)[a]	−6.4	−4.6 (28.1)[e]	−2.9 (54.7)
(95% CI)	(−1.3, −11.2)	(0, −9.6)	(+2.6, −8.1)
Percent change in proportion of high birthweight (≥3500 grams)[a]	5.8	4.8 (17.2)	3.4 (41.4)
(95% CI)	(2.8, 8.8)	(1.8, 8.0)	(0.3, 6.5)
Change in mean birthweight (grams)[b]	17.7	13.1 (26.0)	10.3 (41.8)
(95% CI)	(10.8, 24.6)	(6.2, 20.0)	(3.4, 17.2)

[a] Estimated from logistic regression.
[b] Estimated from multiple linear regression.
[c] Estimates following adjustment for maternal age, infant sex, and prior gravidity categorized as shown in Table 3.
[d] Estimates following adjustment for paternal student status in addition to adjustment for maternal age, infant sex, and prior gravidity.
[e] The numbers in parentheses are the proportion of crude estimates that can be explained by the adjusted factors.

indicates that the proportion of parents who were born in Southeast Asia did not change appreciably over the study period. Since there is little reason to expect that Asians not born in Southeast Asia who were included in our study population would have experienced any noticeable change in their birthweight distribution, the inclusion of such misclassified births (with respect to race) would only lead to an attenuation of the true trends among births to Southeast Asian immigrants.

Another possible source of misclassification that may have arisen in our study is that calendar year (the independent variable in our analyses) is not a perfect correlate of length of time of residence in the US, since some immigrants from Southeast Asia continued to arrive in Washington State from abroad each year, and some immigrated to the US before 1980. Thus, a proportion of the births to Southeast Asian immigrant parents in later years of the study period might have been to recent immigrants, while a proportion of the births in the earlier years of the study period might have been to parents who had been in the US since the mid-1970s. If the health status of more recent immigrants is better than that of past immigrants, which might be reflected in an improved birthweight outcome of their offsprings, the attribution of change of distribution of birthweight that we observed to the length of residence in the US may be spurious. However, our analysis of consecutive births to the same parents during the latter half of the study period showed a decline in low birthweight among immigrants from Southeast Asia which can not be attributable to misclassification of recency of migration. In addition, there is evidence that the health status of recent Southeast Asian immigrants has not improved (at least compared to earlier immigrants) as measured by the prevalence of hepatitis B surface antigen carriers and TB infection.** We therefore believe that this source of misclassification would also lead to observed trends which are, if anything, diminished in magnitude compared to the actual secular changes.

Information on a major risk factor for low birthweight, maternal cigarette smoking during pregnancy, was only available for the three most recent years of the study period. Nevertheless, the proportion of births to mothers who smoked during pregnancy was very low in these years, and was relatively stable, ranging from 3 percent in 1984 to 2.4 percent in 1986. Thus, we feel that our inability to account for changes in cigarette smoking status over the entire study period is unlikely to have explained our results.

The majority of immigrants from Southeast Asia during this period came from underdeveloped parts of their native countries.[1–3] Some of them had experienced nutritional deprivation, starvation, disease infection, and horrible tortures prior to their arrival in the United States.[2–5] Immigration to the United States and the subsequent improvement of their economic and health status might be expected to have

**Cochran J: Director of Refugee Health Program, Department of Public Health, Massachusetts. (personal communication)

some impact on pregnancy outcomes in this population, particularly birthweight. Our study, conducted over a seven-year period, provides evidence that living in the United States has improved the birthweight distribution of infants born to immigrants from Southeast Asia since the time of initial immigration. Of the information available on the birth certificate, secular changes in maternal age, male sex, prior gravidity, and paternal occupation were all correlated with the increase in birthweight. Of these factors, the change in father's occupational status (from "student" to "employed") is the one which is most likely to reflect the change of the Southeast Asian immigrants' socioeconomic environment, which may include improved nutrition, family security, and better adaptation to a new culture. Further research is needed to identify specific factor(s) that might have contributed to the change of birthweight distribution among this population.

ACKNOWLEDGMENTS

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NIH Consensus Development Conference on Early Stage Breast Cancer

Treatment of early stage breast cancer is the subject of an upcoming Consensus Development Conference sponsored by the National Institutes of Health (NIH). The conference, scheduled for June 18–21, 1990 in Masur Auditorium of the NIH Clinical Center, is open to the public.

The purpose of the conference is to reach agreement on the treatment of early stage breast cancer. Key issues to be addressed are:

- The roles of mastectomy versus breast conservation,
- Optimal techniques for breast conservation,
- Adjuvant therapy for patients with node negative breast cancer,
- Use of prognostic factors in managing node negative breast cancer,
- Directions for future research.

The conference will bring together specialists in oncology, surgery, clinical trials, and other relevant fields to present current scientific thinking about the diagnosis, management, and prevention of breast cancer. William C. Wood, MD, Chief of Surgical Oncology, Massachusetts General Hospital, will chair the panel, which will produce the draft report and invite comments from the conference participants. To register for the conference or to obtain further information, contact:

Conference Registrar
Prospect Associates
Suite 500
1801 Rockville Pike
Rockville, MD 20852
Tel: 301/468-meet

Potential Exposure and Health Risks of Infants following Indoor Residential Pesticide Applications

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Abstract: Air and surface chlorpyrifos residues were measured for 24 hours following a 0.5 percent Dursban® broadcast application for fleas inside a residence. Two of the three treated rooms were ventilated following application. Maximum air concentrations were measured 3–7 hours post-application. Peak concentrations in the infant breathing zone were 94 $\mu g/m^3$ in the nonventilated room and 61 $\mu g/m^3$ in the ventilated room, and were substantially higher than concentrations in the sitting adult breathing zone. Concentrations of approximately 30 $\mu g/m^3$ were detected in the infant breathing zone 24 hours post-application. Surface residues available through wipe sampling were 0.7–1.6 $\mu g/cm^2$ of carpet on the day of application and 0.3–0.5 $\mu g/cm^2$ 24 hours post-application. Estimated total absorbed doses for infants were 0.08–0.16 mg/kg on the day of application and 0.04–0.06 mg/kg the day following application, with dermal absorption representing approximately 68 percent of the totals. These doses are 1.2–5.2 times the human No Observable Effect Level (NOEL). Exposures to cholinesterase inhibiting compounds following properly conducted broadcast applications could result in doses at or above the threshold of toxicological response in infants, and should be minimized through appropriate regulatory policy and public education. (*Am J Public Health* 1990; 80:689–693.)

Introduction

The health risks associated with residual pesticides in air and on surfaces in residences have only recently been examined from a public health perspective. The Committee on Urban Pest Management established by the US National Academy of Sciences noted that 5,000 health-related incidents involving pesticides were reported as occurring in homes in the US from 1966 to 1979.[1] Home pesticide applications may pose health risks for occupants, particularly infants and children. A study of 20 adult residents exposed to dichlorvos following crack-and-crevice treatments found a slight decrease in group mean serum cholinesterase, with three residents reporting a feeling of illness 24 hours post-application.[2] Six of 21 children admitted to Arkansas Children's Hospital for organophosphate intoxication were judged to have been exposed following insecticide spraying inside the home.[3] A review of 37 hospitalized pesticide poisonings among infants and children at the Children's Medical Center in Dallas, Texas revealed that five cases were due to pesticide absorption following spraying or fogging inside residences.[4] California State agencies reviewed 44 illness reports in 1984 associated with residential pesticide use,[5] and subsequently conducted a hazard assessment for acute intoxication potential among infants following indoor pesticide treatments.[6] Concern also exists regarding the chronic health effects of children's exposure to pesticides.[7]

Residential monitoring studies have focused primarily on ambient air concentrations of pesticides following several types of applications, including termiticide applications,[8–13] DDVP resin strips hung in households,[14–16] and crack-and-crevice or baseboard treatments.[17–22] Recently the US Environmental Protection Agency initiated a major study of pesticide air concentrations in homes.[23] Limited studies of two additional application methods have been conducted. Broadcast applications,[24,25] involve the hand spraying of an aqueous mixture directly onto target surfaces (e.g., rugs, floors, furniture), while total aerosol release applications (foggers)[26–28] involve activation of a pressurized canister in an unoccupied room, with the pesticide aerosol covering all surfaces (walls, floor, ceiling). Both of these methods produce residues on surfaces to be contacted by occupants. Flea treatment by broadcast application was selected for study here because of the potential for both high air concentrations and substantial dermal exposure. A preliminary risk assessment has been conducted for crawling infants (9–10 months of age).

Methods

Sampling was conducted according to guidelines developed by the Canadian government's Department of Health and Welfare.[29] The study was conducted in three rooms of an unoccupied apartment in New Jersey in June 1987, and extended for 24 hours following application. All rooms were carpeted wall-to-wall with 1.2 cm thick nylon pile. Total surface area treated was 45.27 m^2. No heating or air conditioning systems were in operation during the study. Windows or doors were open in two of the three rooms, while no ventilation occurred in the third room.

Chlorpyrifos application for flea treatment was conducted by a licensed pest control applicator. The formulated product, Dursban® L.O. (EPA Registration No. 464-571), contains 41.5 percent chlorpyrifos (O,O-diethyl O-(3,5,6-trichloro-2-pyridyl) phosphorothioate). The formulation was applied approximately 40 cm above the carpet as a 0.50 percent aqueous spray (40 ml/3.785 L water) with a hand-held fan broadcast nozzle attached to a CO_2 pressurized tank. Applications in the three rooms were identical, covering the entire floor and lasting 5–7 minutes per room.

Air samples were collected in duplicate in each room prior to application and at the following time intervals post-application: 0–0.5 hours, 0.5–1, 1–1.5, 1.5–3, 3–5, 5–7. An additional two-hour sample was collected 24 hours post-application. The samplers were placed in the center of each room mounted 100 cm and 25 cm above the treated surface, approximately 1.5 m apart. Battery-powered porta-

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ble air sampling pumps with flow rates of 2 L/min were calibrated with the sampling train in line prior to and following the sampling period. Chlorpyrifos vapors were collected with ORBO-44 tubes (Supelpak 20). Tubes were capped and stored in an ice chest for transport to the laboratory.

Aluminum foil squares (10 cm × 10 cm) were placed in the rooms before treatment to measure residues deposited on the carpet surface. Five squares were collected from each room immediately following application (5 minutes). The wipe procedure was based on the standard method of the US Occupational Safety and Health Administration.[30] Sets of three wipes were collected in each room at eight time intervals post-application: 20, 40, 80, 100, 170, 260, 380 minutes, and 24 hours. All areas to be wiped were marked by disposable cardboard frames with 10 cm × 10 cm cut-out interiors. A new plastic glove was worn for each wipe sample to avoid cross-contamination of samples. The wipe consisted of three strokes across the surface with a 7.5 × 7.5 cm surgical gauze pad sprayed lightly with distilled water. This procedure was repeated with a second gauze pad, changing the wipe orientation 90°. Pads were placed in glass storage jars in an ice chest for transport to the laboratory.

All samples were stored at −20°C until analysis. Granular contents of the Orbo-44 tubes were transferred into serum vials filled with 10.0 ml of pesticide-grade toluene. A subset of the samples were analyzed for breakthrough into the secondary section of the tubes, but such breakthrough was negligible. Capped vials were shaken at high speed (100 cpm) for 5 minutes on a mechanical shaking table, allowed to sit for 30 minutes and the extracts transferred to GC vials for analysis. Both the foil and the pads were extracted in 30 ml of toluene on the shaking table at high speed for 30 minutes and the extracts transferred into GC vials.

Samples were analyzed on a Hewlett Packard 5890A gas chromatograph equipped with an electron capture detector. Heptachlor epoxide was added to all samples as an internal standard. The limit of detection for 1-μl injections was 1 picogram/μl. The limits of detection for the sampling media were as follows: air samples, 83 ng/m^3; foil and gauze samples, 0.6 ng/cm^2. Solvent blanks run with each set of samples had no detectable chlorpyrifos. Fortification/recovery studies were conducted for all sampling media. The mean and standard deviation from these studies were as follows: air samples, 85.3% ±6.0; aluminum foil squares, 98.6% ±2.5; gauze wipes, 84.0% ±10.1. Data have been adjusted on the basis of these recovery values. Two air samples, three foil and three wipe samples were collected in each room prior to application. No background chlorpyrifos levels were detected.

Results

Chlorpyrifos levels on aluminum foil samples collected immediately (5 minutes) following application averaged 13.6 μg/cm^2 (±6.1) and levels in the three rooms were not significantly different. Measured deposition corresponded well with the 13 μg/cm^2 value calculated from label recommendations.

Chlorpyrifos residues available by wipe sampling in the ventilated rooms dropped by more than one-half within 30 minutes, averaging 0.7 μg/cm^2 throughout the remainder of Day 1 and 0.3 μg/cm^2 24 hour post-application (Table 1). Surface residues in the nonventilated room followed the same pattern, but were significantly higher at all time intervals.

Chlorpyrifos vapors measured in the sitting adult breathing zone (100 cm above the carpet) increased over time (Figure 1). While concentrations in the ventilated rooms never exceeded 20 μg/m^3, levels >60 μg/m^3 were measured in the nonventilated room 3–5 hours post-application. The effect of ventilation was further reflected in samples 24 hour post-application: >40 μg/m^3 with no ventilation compared to <10 μg/m^3 with ventilation.

Substantially higher chlorpyrifos concentrations were measured in the infant breathing zone (25 cm above carpet), with ventilation having far less impact (Figure 2). Concentrations reached 94 μg/m^3 5–7 hours post-application in the nonventilated room and exceeded 60 μg/m^3 3–5 hours post-application in the ventilated rooms. After 24 hours an equilibrium concentration in all rooms of approximately 30 μg/m^3 was reached. It thus appears that a vertical concentration gradient developed following the application, with the treated carpet serving as a source of volatilized chlorpyrifos. Open windows provided mixing and dilution of air one meter above the carpet, but had much less effect on concentrations near the floor.

Time-weighted averages for the 24 hour post-application period in the infant breathing zone were 41.2 and 66.8 μg/m^3 for ventilated and nonventilated rooms, respectively, substantially higher than the corresponding values of 12.3 and 45.7 μg/m^3 for the adult breathing zone. All of these concentrations exceeded the National Academy of Sciences (NAS) interim guideline of 10 μg/m^3 for chlorpyrifos in indoor air following termiticide treatments.[31]

A risk assessment was conducted based on the scenario of a 9–10 month old infant playing in a carpeted room beginning at least one hour after a 0.5 percent chlorpyrifos

TABLE 1—Chlorpyrifos Residues Available by Wipe Sampling: Effects of Time and Ventilation

	Ventilated Rooms			Nonventilated Room		
Sample Time (hr)	N	Mean Residue[a] (μg/cm^2)	Percent Initial Deposit†	N	Mean Residue[b] (μg/cm^2)	Percent Initial Deposit
<0.5 Immediate	6	1.69	12.4	3	3.90	28.6
0.5–6.5 Day 1	35*	0.69	5.0	18	1.56	11.4
24 Day 2	6	0.28	2.0	3	0.48	3.5

a,b: all ventilated room values significantly lower than nonventilated room values (ANOVA: Student-Neuman-Keuls Test; $p < .01$)
†deposition following application = 13.6 μg/cm^2
*25.2 μg/cm^2 value excluded as outlier

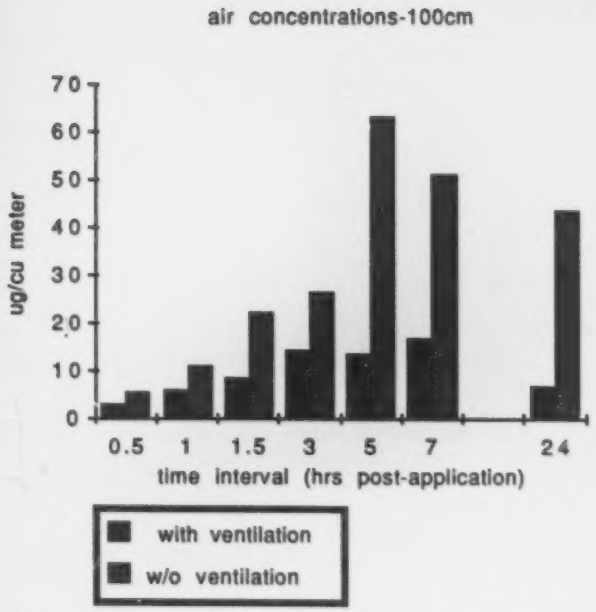

FIGURE 1—Chlorpyrifos Air Concentrations in the Sitting Adult Breathing Zone (100 cm)

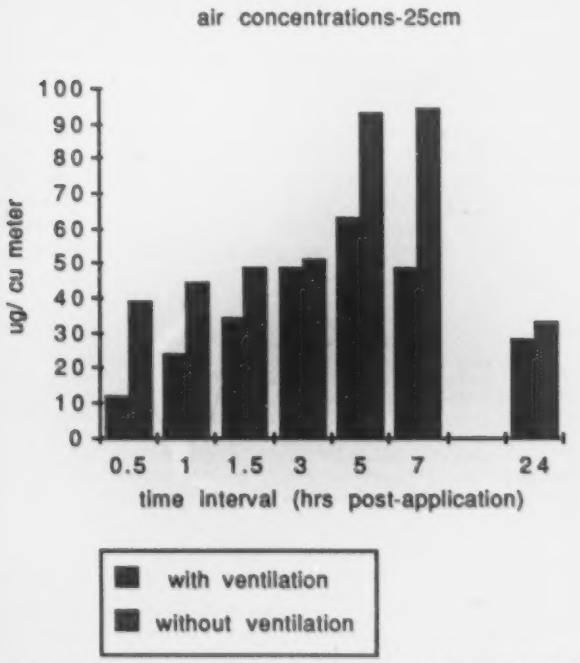

FIGURE 2—Chlorpyrifos Air Concentrations in the Infant Breathing Zone (25 cm)

broadcast application. The following assumptions were employed:

1. Infant weight = 9.0 kg[32]
2. Infant respiratory volume = 2.1 L/min resting (16 hours/day); 6.3 L/min active (8 hours/day)[33]
3. 100% respiratory absorption of chlorpyrifos vapors
4. Total carpet contacted in one day = 2.3 m^2 (25 ft^2)
5. 100% transfer of available residues (wipe samples) to skin
6. 3% dermal absorption of chlorpyrifos[6,34]

Estimated absorbed dermal doses for infants (Table 2) are the product of surface area contacted, available residues (Day 1 and Day 2 in Table 1) and percent absorbed, divided by body weight. The estimated dose in the nonventilated room is 0.120 mg/kg on Day 1, 3.2 times the dose for Day 2. Ventilation reduced the dose estimates approximately two-fold. Estimated absorbed respiratory doses are the product of time-weighted air concentrations, respiratory volume and percent absorbed, divided by body weight. Estimated Day 1 dose with no ventilation is 0.038 mg/kg, more than twice the Day 2 dose. Ventilation reduced the dose on Day 1 by approximately one-third, but had little effect on Day 2 estimates. Total absorbed dose estimates for the day of application are 0.075 and 0.158 mg/kg for ventilated and nonventilated rooms, respectively. Day 2 estimates are approximately 2–3 times lower. Dermal exposure represents on average 68 percent of the total absorbed dose.

Discussion

Chlorpyrifos residues following broadcast application were substantially higher than those reported for other types of residential applications. Total release aerosol applications have produced deposition levels of approximately 2 $\mu g/cm^2$ and maximum air concentrations following ventilation of 14 $\mu g/m^3$.[28] Crack-and-crevice or baseboard applications have resulted in deposition levels of from 1–3 $\mu g/cm^2$,[18,22] and air concentrations of 1–2 $\mu g/m^3$.[20,21] Termiticide applications have produced air concentrations of <1–8.5 $\mu g/m^3$.[11–13] Broadcast applications thus appear to produce deposition levels 5–10 times higher and peak air concentrations approximately one to two orders of magnitude higher than other application procedures. Of further concern is the finding that vapor concentrations increase over the first few hours, peaking 3–7 hours post-application.

The extent to which indoor pesticide applications might produce intoxications among residents is largely unknown. A recent risk assessment calculation of a 2.68 mg/kg absorbed dose for infants in chlorpyrifos-treated residences was based on several conservative assumptions, including complete (100 percent) absorption of chlorpyrifos through the skin.[6] The risk assessment presented here avoids such worst-case assumptions, but includes major uncertainties in the dermal exposure estimates. The rate of transfer of pesticides from treated surfaces to skin and the amount of surface contacted over time are not known. Research has been initiated to adapt fluorescent tracer and video imaging techniques used in occupational exposure studies to address these issues.[35,36] Several factors not included in the dose calculations suggest that chlorpyrifos absorption could be substantially higher. First, a 3 percent dermal absorption estimate may be low. The conventional topical dose for pesticide absorption studies is approximately 40 $\mu g/cm^2$,[37] whereas the average dose applied to the skin in the chlorpyrifos absorption study was >4000 $\mu g/cm^2$.[34] High topical doses result in relatively low percent absorbed values.[38,39] An estimate of the rate of chlorpyrifos penetration through the skin is needed to more accurately estimate absorbed dermal dose. Second, infant skin may be more permeable than the adult skin used to estimate chlorpyrifos absorption. Although the stratum cor-

TABLE 2—Total Estimated Absorbed Chlorpyrifos Dose for Infants: Day of Application (Day 1) and Day following Application (Day 2)

Room Condition	Dermal Dose (mg/kg)	% Total	Respiratory Dose (mg/kg)	% Total	Total Dose (mg/kg)	Percent of NOEL*
Day 1						
Ventilated	0.052	69	0.023	31	0.075	250
Nonventilated	0.120	76	0.038	24	0.158	527
Day 2						
Ventilated	0.022	58	0.016	42	0.038	127
Nonventilated	0.037	67	0.018	33	0.055	183

*Human No Observable Effect Level = 0.03 mg/kg/day

neum of the infant is considered to be fully developed, the permeability of infant skin in humans has not been measured.[39] Studies in infant rats have demonstrated increased percutaneous absorption of chlorpyrifos relative to adults.[40] Third, ingestion from deposits on hands would increase the estimates, since chlorpyrifos is absorbed by the oral route more efficiently than by the dermal route.[34] Finally, skin conditions to which infants may be particularly susceptible (e.g., dryness and rashes) are known to reduce the barrier properties of the skin.[39]

The no observable effect level (NOEL) for chlorpyrifos is based on measurable changes in plasma acetylcholinesterase. The NOEL in humans is 0.03 mg/kg/day.[41] All of the absorbed dose estimates in Table 2 exceed the NOEL, with doses 2.5–5 times the NOEL on Day 1, declining to 1.2–1.8 times on Day 2. The US Environmental Protection Agency has derived a "human reference dose" of 0.003 mg/kg/day for chlorpyrifos by applying a 10-fold safety factor to the human NOEL.[42] The dose estimates in this study exceed the human reference dose by 10–50 times.

NOEL values are commonly employed to determine standards for human safety, but are based on an assumption of continuous exposure (e.g., Acceptable Daily Intakes of pesticide residues in foods). A similar assumption underlies the NAS guideline for indoor air concentrations.[30] Since the potential exposures calculated here would be intermittent, their toxicological significance is difficult to interpret. As yet no short-term toxicological measure analogous to the NOEL is available.

Despite uncertainties in exposure/absorption estimates and toxicological interpretation, the dose values derived in this study raise a public health concern. Broadcast applications and possibly total release aerosol/fogging applications of acutely toxic insecticides may result in dermal and respiratory exposures sufficient to cause measurable toxicological responses in infants. While such exposures are not likely to produce recognizable symptoms of cholinesterase inhibition, the possibility of cases with subclinical effects cannot be ruled out. Confirmation of such exposures would require biological monitoring (e.g., detection of 3,5,6-trichloro-2-pyridinal in the urine for chlorpyrifos).

The potential for exposures greater than those reported here should also be noted. The training of many urban pesticide applicators is minimal and surveillance of application performance is nonexistent in most states. It is not unreasonable to assume that accidents and cases of inadvertent over-application will occur under these circumstances, albeit with low frequency. Furthermore, repeated applications for fleas and other household pests are not uncommon.

Current federal regulatory requirements for indoor pesticide applications are limited to label instructions. The label for broadcast applications of the compound under study here is typical (K. Lunchick, USEPA; personal communication); i.e., the premises should be vacated during application, and humans or pets should not contact treated surfaces until the spray has dried. These instructions are normally communicated by the applicator to occupants as a 1–2 hour reentry period. The finding of elevated air concentrations 3–7 hours post-application and the potential for dermal contact indicate that this reentry period is inappropriate for infants and small children.

Several strategies are available to reduce the risks associated with indoor pesticide applications. First, notification requirements could be established to inform occupants of potential health risks and means for minimizing exposure. Recommendations for broadcast applications of acutely toxic compounds such as chlorpyrifos might include thorough ventilation prior to reentry regardless of seasonal conditions, and well-defined reentry periods beyond 1–2 hours with special warnings regarding contact with treated surfaces by infants and small children. Second, more rigorous training and licensing procedures for applicators and application surveillance programs could be developed. Finally, product registrations could be modified or withdrawn for specific applications if an acceptable level of risk cannot be demonstrated.

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'Late Breaker' Session on Injury Control Invites Abstracts

The Injury Control and Emergency Health Services special primary interest group of the American Public Health Association has announced it will again feature a "late breaker" session during the APHA upcoming 118th annual meeting in New York City. The session will be held on Tuesday, October 2, 8:30 am–10:00 am, and will feature work completed within the last few months—after the deadline for consideration in the regular symposia of the APHA annual meeting.

Abstracts of 250 words or less will be accepted by the Injury Control SPIG until August 15, 1990. Please send the abstract, title of the paper, authors' name, address and telephone number to: Richard Waxweiler, Division of Injury Control, Centers for Disease Control, Mail Stop F-36, Atlanta, GA 30333. Tel: 404/488-4695.

Breastfeeding Patterns in Puerto Rico

Jose E. Becerra, MD, MPH, and Jack C. Smith, MS

Abstract: In the 1950s, Puerto Rico began an active industrialization program. We used data from the 1982 Puerto Rico Fertility and Family Planning Assessment to describe the trend in the incidence of breastfeeding in Puerto Rico over time and to ascertain some of its determinants. From 1946 through 1982, 5,884 infants were born among this statistically representative sample of reproductive-aged women. The proportion of infants who had ever been breastfed was 59 percent for births before 1960 (mean duration = 7.8 months), dropped to 25 percent for infants born from 1970 to 1974 (mean duration = 4.9), and rose to 38 percent for births delivered from 1980 to 1982 (mean duration = 3.4). Prior breastfeeding experience was an important determinant of breastfeeding a newborn. Infants of mothers who had breastfed a previous baby were 7.3 times more likely to be breastfed (95% confidence interval = 6.6, 8.0) compared with infants of mothers who had not previously breastfed. The 38 percent of infants who were breastfed in Puerto Rico in the early 1980s is below the 74 percent to 97 percent reported in Latin America and below the 54 percent reported in the United States for the same period. This study provides baseline data for any future intervention strategies. (*Am J Public Health* 1990; 80:694–697.)

Introduction

A resurgence in the practice of breastfeeding has occurred throughout the world.[1] In the United States the downward trend in breastfeeding practice has been reversed since the late 1970s.[2] Breastfeeding trends vary in less developed countries, and three basic phases of progression have been described.[1]

The traditional phase is characterized by a high proportion of breastfed infants and a long duration of breastfeeding. In the transformation phase, both the proportion of breastfed infants and the duration of breastfeeding show a decrease. The third, or resurgent phase, is characterized by an increase in both the proportion of breastfed infants and the duration of breastfeeding. The patterns of these trends are not uniform in any country; they vary by geography and by the socioeconomic status of different groups.

The relation between the economic development initiated in Puerto Rico in the 1950s and breastfeeding practices among Puerto Ricans has not been studied before. The present study does so, using data from a 1982 survey.

Methods

In 1982, a multipurpose island-wide social, demographic, and maternal/child health study of women ages 15–49 years old living in Puerto Rico was undertaken as a collaborative project involving the University of Puerto Rico School of Public Health, the Puerto Rico Department of Health, and the Centers for Disease Control. The sampling methods have been described in detail elsewhere.[3] The completion rate of the household questionnaire was 91 percent.

A total of 3,175 women were interviewed and, of this number, 2,012 had had at least one live birth. For each live birth reported, mothers were asked: 1) "When your baby was an infant, did you breastfeed him/her?"; 2) "How many months old was s/he when you stopped breastfeeding him/her?"; and 3) "At what age was s/he first given any infant formula, cow's milk, solid foods other than breastmilk on a regular basis?" Of the 2,012 mothers queried, only one mother could not provide a response to any of these questions.

The total number of live births among the women in the sample was 5,884, all delivered between 1946 through 1982. For our analysis, we divided the study period into six birth delivery periods: 1946–59 (N = 709); 1960–64 (N = 924); 1965–69 (N = 1,135); 1970–74 (N = 1,239); 1975–79 (N = 1,207); and 1980–82 (N = 640). We were unable to determine the year of birth for 30 (0.5 percent) infants. Given the blurred distinction between rural and urban areas in Puerto Rico throughout the study period, the place where the delivery occurred (home, private hospital, and public hospital) was used instead of geographical (urban vs rural) correlates.

We used SESSUDAN,[4] a standard errors program, to compute standardized rates from sample survey data and to produce weighed estimates with 95% confidence intervals. We then calculated the proportion of infants who were ever breastfed and the proportion of infants who were exclusively breastfed for at least the first month of life for each birth delivery period stratified by place of delivery (home, private hospital, and private hospital) and by years of maternal education (<12, 12–15, >15) reported at the time of the interview (1982).

We also used the life table analysis program in BMDP[5] to calculate the mean duration of breastfeeding for each birth delivery period and for the three categories of maternal education for births occurring between 1975 and 1982. Infants of mothers still breastfeeding at the time of the interview and infants who had died while still breastfeeding were considered (statistically) censored observations.

We also used SAS[6] to calculate the breastfeeding percent ratio of multiparous mothers with and without previous breastfeeding experience, adjusted for years of maternal education and birth delivery period. In addition, we used the logistic regression program in BMDP[7] to simultaneously determine the strength of association (measured by the odds ratio [OR]) between the infant's breastfeeding status (ever breastfed or not) and maternal age in years (<20, ≥20), place where the mother was raised (continental United States, Puerto Rico), and years of maternal education (<12, 12–15, >15) among primiparas delivering from 1975 to 1982.

Results

In Puerto Rico, the proportion of infants who were ever breastfed was 37.3 percent (95% CI = 34.6, 39.9) for the entire period of 1946 to 1982 (Table 1). This proportion was

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TABLE 1—Trends for Infants Ever Breastfed in Puerto Rico (aggregated by parity), 1946–82

	Period of Birth						
	1946–59	1960–64	1965–69	1970–74	1975–79	1980–82	Total*
	Percent (sample size)						
Primiparous Mothers							
Ever breastfed	57.7 (312)	45.1 (313)	32.7 (393)	20.3 (405)	26.0 (391)	43.4 (193)	35.7 (2012)
Education (years)							
<12	62.8 (213)	48.8 (163)	37.3 (170)	17.9 (168)	20.7 (113)	32.0 (75)	39.1 (906)
12–15	50.5 (78)	39.4 (122)	27.5 (183)	21.4 (187)	26.0 (222)	44.8 (93)	31.8 (886)
>15	32.2 (21)	48.0 (28)	35.6 (40)	24.4 (50)	29.5 (56)	72.7 (25)	36.9 (220)
Type of facility							
Public hospital	62.4 (165)	48.0 (174)	36.7 (207)	18.4 (223)	21.7 (223)	42.6 (117)	36.0 (1112)
Private hospital	44.2 (94)	39.4 (127)	27.2 (179)	22.4 (179)	32.2 (167)	45.2 (75)	32.6 (821)
Home	68.8 (51)	— (9)	— (5)	— (2)	— (0)	— (0)	67.9 (69)
Multiparous mothers							
Ever breastfed	60.7 (397)	50.1 (611)	39.6 (742)	27.5 (834)	28.9 (814)	35.9 (446)	38.0 (3869)
Education (years)							
<12	62.0 (307)	55.5 (416)	44.2 (442)	30.2 (450)	28.8 (394)	30.8 (194)	41.7 (2224)
12–15	60.1 (74)	38.2 (156)	35.8 (241)	23.6 (314)	28.2 (345)	40.3 (197)	33.3 (1331)
>15	38.9 (16)	38.5 (39)	20.7 (59)	26.9 (70)	32.5 (75)	39.5 (55)	31.2 (314)
Type of facility							
Public hospital	58.2 (186)	51.2 (329)	43.7 (412)	30.6 (449)	30.0 (459)	33.8 (243)	38.9 (2087)
Private hospital	43.8 (89)	32.7 (186)	29.8 (282)	23.5 (364)	28.2 (338)	38.9 (199)	30.4 (1460)
Home	78.8 (116)	81.7 (86)	59.3 (44)	30.5 (17)	24.6 (11)	— (2)	68.9 (290)
Total*	59.4 (709)	48.4 (924)	37.3 (1135)	25.1 (1239)	28.1 (1207)	38.3 (640)	37.3 (5684)

(*)the difference between the sample size for total and the summation of cells corresponds to the missing values in each category.

59.4 percent (95% CI = 53.8, 65.0) for births occurring before 1960, dropped to 25.1 percent (95% CI = 21.2, 29.1) for infants born from 1970 to 1974, and rose to 38.3 percent (95% CI = 34.1, 42.4) for births delivered from 1980 to 1982 (Figure 1). The proportion of infants who were exclusively breastfed for at least the first month decreased from 27.4 percent (95% CI = 23.2, 31.3) before 1960 to 4.1 percent (95% CI = 0.0, 7.9) in 1982 (Figure 1).

The mean duration of breastfeeding for all infants throughout the study was 5.6 months (95% CI = 5.2, 5.9). The mean duration of breastfeeding declined consistently from 7.8 months (95% CI = 6.9, 8.6) for births occurring before 1960 to 3.4 months (95% CI = 2.8, 4.0) for births delivered from 1980 to 1982.

Breastfeeding status and place of delivery were related (Figure 2). Before 1975, infants delivered at home or in public hospitals were more likely to be breastfed than were infants

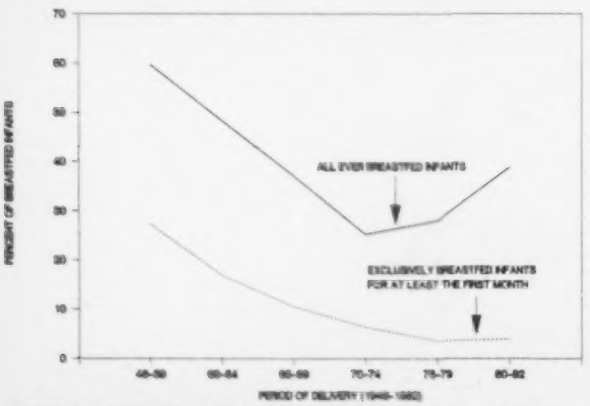

FIGURE 1—Breastfeeding Trends in Puerto Rico, 1946–82: Percent of Infants Breastfed by Birth Delivery Period

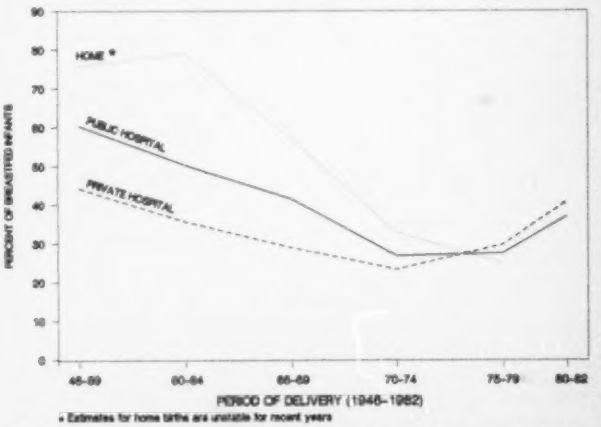

FIGURE 2—Infants Ever Breastfed by Place of Delivery, Puerto Rico, 1946–82

delivered at private hospitals. However, this pattern is reversed after 1975. From 1980 to 1982, 40.6 percent (95% CI = 33.8, 47.5) of infants delivered in private hospitals were ever breastfed, compared with 36.8 percent (95% CI = 30.8, 42.8) of infants delivered in the public sector.

A similarly reversed pattern appears in the practice of breastfeeding after 1975 in relation to maternal education (Figure 3). From 1980 to 1982, 50.0 percent (95% CI = 37.9, 61.6) of infants of mothers with more than 15 years of formal education were ever breastfed, compared with 41.7 percent (95% CI = 36.4, 47.1) for infants of mothers with 12–15 years of education, and 31.4 percent (95% CI = 25.1, 37.8) for infants of mothers with fewer than 12 years of education. However, multiparous mothers with fewer than 12 years of education did not show the same pattern as their primiparous counterparts in recent years (Table 1). From 1980–82, primiparous mothers showed the strongest correlation between

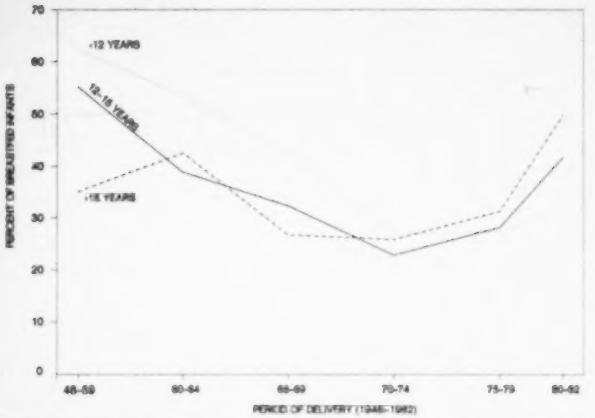

FIGURE 3—Infants Ever Breastfed and Maternal Education at Interview, Puerto Rico, 1946–82

education and breastfeeding practice and the greatest rise in the proportion of infants ever breastfed (Table 1).

From 1975 to 1982, the mean duration of breastfeeding for infants of mothers with more than 15 years of formal education and delivered after 1975 as 3.2 months (95% CI = 2.4, 4.1); the duration was 3.4 months (95% CI = 2.8, 3.9) for mothers with 12–15 years of education and 4.1 months (95% CI = 3.2, 4.9) for mothers with fewer than 12 years.

On the average, infants of multiparous mothers who had breastfed a previous baby were 7.3 times more likely to be breastfed (95% CI = 6.6, 8.0) than were infants of mothers who had not previously breastfed. This measure of association was adjusted for maternal education and birth delivery period.

The place where the mother had been raised, maternal education, and maternal age were examined among primiparas. From 1975 to 1982, maternal education was the strongest independent correlate of breastfeeding among first live-birth infants. Mothers with 12–15 years of education had OR of 1.8 (95% CI = 1.1, 2.8), and mothers with more than 15 years of formal education had OR of 2.8 (95% CI = 1.5, 5.1) compared with mothers with fewer than 12 years of formal education. These estimates were adjusted for maternal age and place where the mother was raised (this latter as a proxy for cultural identification).

Discussion

In the 1950s, Puerto Rico began an active industrialization program that reduced infant mortality from 114.1 infant deaths per 1,000 live births in 1940 to 44.4 in 1960 and 19.2 in 1980.[8] The life expectancy increased from 46.0 years in 1940 to 69.4 in 1960 and 74.9 in 1980.[8]

Our results document that the traditional phase of high rates of breastfeeding among women who delivered at home or in public hospitals and who were less educated declined in Puerto Rico with the advent of industrialization in the 1950s. By 1980, Puerto Rico was between the transformation and resurgent phases of breastfeeding practice. That is, the upward trend in the proportion of infants who were ever breastfed since 1975 was accompanied by a very low and decreasing proportion of infants who were exclusively breastfed during the first month of life. This trend shows that an increasing proportion of mothers who breastfed are probably using formula supplementation during the first month. However, very few medical indications justify introducing food other than breastmilk to a full-term baby during its first four months of life.[9]

Evidence shows that breastfeeding practice in Puerto Rico lags behind the United States and many Latin American countries. For 1980, 54 percent of infants in the United States had ever been breastfed[10] and, for the same period, 74 percent to 97 percent of all infants in selected Latin American countries had ever been breastfed.[11] In comparison, approximately 40 percent of infants born in Puerto Rico in 1980 had ever been breastfed. For Puerto Ricans living in the northeastern region of the United States, the proportion of infants who were ever breastfed is even lower: 26 percent for the 1980–82 period.*

The average number of months that an infant is breastfed in Puerto Rico is consistently decreasing. As previous research has shown in other populations,[12,13] the lack of adequate familial, social, and professional support, and the insensitive employment environment facing modern, highly educated women discourage mothers from breastfeeding. The downward trend in number of months of breastfeeding parallels the decreasing proportion of infants exclusively breastfed for at least the first month of life.

The association between maternal education and breastfeeding is reversed during the upward trend of breastfeeding after 1975. This reversal is consistent with previously published studies.[2] After 1975, mothers with higher education were more likely to initiate breastfeeding than were mothers with less education, whereas the opposite was true before 1960. This pattern is similar to the association between place of delivery and breastfeeding. It is important to realize that institution of delivery is associated with maternal education and socioeconomic status in Puerto Rico.[3]

Although recall bias could be invoked to explain some of these findings, we find no plausible and consistent hypothesis to support such contention. In addition, the general pattern of our results follows the expected interaction between economic development and breastfeeding practices.

Since 1975, maternal education, probably measuring (in addition to socioeconomic status) health behavior awareness and increased capacity to change health belief systems, is strongly correlated with initiation of breastfeeding among primiparas. This trend is encouraging and should guide policy-makers to carry out interventions that will improve breastfeeding practices on the island. However, the decreasing duration of breastfeeding in recent years as maternal education increases provides grounds for concern. This finding differs with previously published studies[14] on the relationship between maternal education and duration of breastfeeding in recent years.

Previous breastfeeding experience was a very strong predictor of breastfeeding a subsequent infant in our study. This fact underscores the importance of role models for young mothers and the self-sustaining nature of a change in breastfeeding behavior. This finding held true even after we adjusted for maternal education and birth delivery period.

In addition to the well known psychological and biological benefits of breastfeeding for the infant,[15] intervention strategies should also emphasize the maternal benefits of

*Carter O, Ezzati T: Breastfeeding practices among Mexican Americans, Cuban Americans, and Puerto Ricans: HHANES, 1982–84 (Abstract). American Public Health Association 115th Annual Meeting, 1987, New Orleans, LA.

breastfeeding: breastfeeding helps postpartum weight loss,[16] breastfeeding prolongs postpartum amenorrhea,[17] and breastfeeding may exert a protective effect against premenopausal breast cancer.[18,19]

In summary, breastfeeding practice in Puerto Rico in 1982 fell between the transitional and resurgent phases of progression. Although we have documented an upward trend in the proportion of infants who were ever breastfed since 1975, more sensitive indicators of breastfeeding practice, such as the proportion of infants who were exclusively breastfed in the first month and duration of breastfeeding, show disturbing decreasing trends in Puerto Rico.

ACKNOWLEDGMENT

The authors wish to acknowledge the original contributions and thoughtful review of the manuscript made by Dr. Rosa B. Licha-de-Becerra. Part of this work was presented at the American Public Health Association, 115th Annual Meeting, October 19, 1987, New Orleans, Louisiana.

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Nominations Invited for 1990 McAlpin Award in Mental Health Research

The National Mental Health Association (NMHA) is seeking nominations for its 1990 McAlpin Mental Health Research Achievement Award to be presented to a researcher in the field of mental illness. The award honors the memory of William R. McAlpin, Jr., who was devoted to the cause of mental health and the advancement of mental illness research.

Nominations will be considered by a panel of judges for researchers whose work offers significant promise in increasing scientific knowledge in the understanding of one or more of the mental illnesses and meets the highest research standards. Previous distinguished recipients are Donald F. Klein, MD, Columbia University; Floyd Elliott Bloom, MD, Salk Institute Center for Behavioral Neurobiology; Paul Greengard, PhD, Rockefeller University; and William T. Carpenter, Jr., MD Maryland Psychiatric Research Center.

The McAlpin Award will be presented during the Seventh Annual National Scientific Symposium on Mental Health and Mental Illnesses, to be held in Houston, Texas, September 14–15. The recipient will be expected to attend the symposium to accept the award.

Nominations from sponsors (individuals and organizations) must be received no later than July 31, 1990. For complete details on nomination procedures contact the NMHA Executive Office, 1021 Prince Street, Alexandria, VA 22314-2971, (703) 684-7722 for a brochure on the 1990 McAlpin Award.

Dietary Intake of Americans Reporting Adherence to a Low Cholesterol Diet (NHANES II)

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Abstract: We studied the dietary intake of persons age 18 and over participating in the 1976–80 National Health and Nutrition Examination Survey (NHANES II). We compared the diets of those on a "low cholesterol" diet (dieters, n = 296) with the diets of individuals not following a special diet (non-dieters, n = 10,052). Dietary intakes were assessed by 24-hour recall and food frequency reports and were adjusted for differences in age, sex, race, education, smoking, and socioeconomic status by multivariate statistical techniques. Dieters consumed 16 percent (99% confidence interval- −24, −9) fewer calories than non-dieters, and had decreased intake of saturated fat by 25 percent (−34, −15) and cholesterol by 21 percent (−32, −10). However, their intake of vitamin A, thiamine, riboflavin, niacin, calcium, and iron were not significantly different from non-dieters and their ascorbic acid intake was 18 percent (1, 36) higher. The nutrient density (nutrient/1000 kcal) among dieters was increased over non-dieters by 35 percent (−3, 72) for vitamin A, 35 percent (1, 70) for thiamine, 30 percent (−4, 65) for riboflavin, 27 percent (9, 44) for niacin, 60 percent (40, 80) for vitamin C, 14 percent for calcium (−5, 32), and 15 percent for iron (6, 25). Compared with non-dieters, dieters consumed 27 percent (9, 45) more poultry and 39 percent (12, 66) more fish, but consumed 30 percent (−44, −16) less eggs and 15 percent (−24, −6) less meat. (*Am J Public Health* 1990; 80:698–703.)

Introduction

Recent evidence has linked reduction of blood cholesterol with reduced incidence of coronary heart disease.[1,2] Dietary intervention effectively lowers blood cholesterol levels[3,4] and therefore the cholesterol lowering diet is currently emphasized as an important public health measure. Recent guidelines established by the National Cholesterol Education Program have defined blood cholesterol values above 200 mg/dl as undesirable due to their association with increased coronary heart disease risk[5]; more than half of all Americans fall into this category.[6] Community programs to educate the public concerning hypercholesterolemia and the importance of a proper diet have recently been established,[7–9] and many individuals now report dietary changes in response to these initiatives.[8] Because efforts to enhance knowledge concerning hypercholesterolemia and diet among the public are still in progress, one may anticipate that more Americans will attempt dietary modification in the near future.

Despite this willingness to change dietary habits, the ability of Americans who adopt a "low cholesterol diet" to make significant dietary modifications is unknown. Compared with the typical American diet, the cholesterol lowering diet recommended by the National Cholesterol Education Program is lower in total fat, saturated fat, and cholesterol. In addition, caloric intake is limited, particularly in overweight individuals.[5] To achieve these goals, foods must be substituted into the diet to replace those which are rich in saturated fat and cholesterol. To modify food intake correctly, the dieter must be aware of the basic principles of the cholesterol lowering diet as well as the nutritional adequacy of potential food substitutions. Otherwise, the modification of food choices may have minimal effect on saturated fat and/or cholesterol intake, or may be made at the expense of the intake of other important nutrients. Subjects recruited for research studies designed to evaluate the efficacy of a short period of dietary intervention have generally shown significant decreases in serum cholesterol levels when the diet was instituted, suggesting that appropriate dietary changes were implemented during the study period.[1–4,10,11] However, individuals participating in these studies were highly selected and were provided with intensive nutrition counseling; therefore, they may not be representative of most Americans who wish to adopt a cholesterol lowering diet.

The purpose of this study was to determine if Americans who adopt a cholesterol lowering diet make dietary modifications that: 1) are in accordance with accepted dietary guidelines, and 2) allow for an adequate supply of other nutrients.

Methods

Data were analyzed from the second National Health and Nutrition Examination Survey (NHANES II), a comprehensive population survey which included a detailed dietary questionnaire from which nutrient intakes were calculated in a sample of over 10,000 adults. The NHANES II sample was selected so that population subgroups of special interest for nutritional assessment were oversampled. Among adults, the oversampled groups included persons over 60 years of age and persons below the poverty level. Sample weights were then computed for each participant so that the total sample closely estimated the noninstitutionalized civilian population. This sample weighting reflects the individual selection probability, adjustments for non-response, and post-stratification adjustments. A complete description of the survey methods has been reported.[12]

Data from NHANES II were acquired from sample persons over several visits. At the initial visit, demographic data (i.e., age, education and financial status, household information, etc.) and some of the medical history were obtained. At the second visit, the remainder of the medical history, dietary recall and food frequency information, and blood for laboratory testing were acquired. Adults ages 18 to 74 who attended this second visit were included in this study. Because not all persons who were willing to undergo the initial visit agreed to participate at the second visit, the potential for bias was introduced if these persons differed significantly from other sample persons. The sample weighting in NHANES II was therefore adjusted to correct for this

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problem.[13] This sample weight (the "examined final weight") has been employed for all analyses in this paper.

Nutrient intakes for vitamin A, thiamin, riboflavin, niacin, vitamin A, iron, and calcium were estimated from 24-hour recall interviews. These nutrient values were obtained from the US Department of Agriculture reference handbook[14] or directly from the food manufacturer, and were recorded directly on the NHANES II data tapes following completion of the survey. Vitamin and mineral supplements were not included in the calculation of nutrient intake. If two major meals could not be recalled, or if the respondent refused to cooperate or was uncooperative due to attitude, then the recall data were judged to be unsatisfactory by the interviewer. These unsatisfactory interviews were excluded from the analysis; imputed dietary intakes were not substituted for these unsatisfactory recalls. Three-month food frequency reports provided estimates of consumption of specific food groups (such as skim and whole milk, eggs, meat, fish, poultry, etc.). Responses from food frequency questions were converted into the number of weekly servings; respondents reporting occasional intake of less than one serving weekly were arbitrarily assigned a weekly intake of 1/2 serving. Dietary recall data are presented in Tables 2 through 5; data from the food frequency reports are presented in Table 6.

All participants attending the second visit were asked the question "Are you following a special diet?" If they responded affirmatively, they were then asked a series of questions to define the diet, such as "Is this diet to lose weight?"; "Is this a low salt diet?"; or "Is this a vegetarian diet?" Additional questions asked included whether the diet was prescribed by a health professional and the length of time that the diet had been followed. The complete list of questions asked to describe the diet are provided in the Appendix. Participants who were told by a physician that they had a previous heart attack or "hardening of the arteries" were considered to have a history of coronary artery disease. The poverty index, provided in NHANES II, was used to assess socioeconomic status. This is a measure of financial income adjusted to geographic poverty level for family unit; values of less than 1.0 are below the poverty level.

Because the association of dietary cholesterol with blood cholesterol and coronary heart disease was widely recognized during the period when NHANES II was conducted,[15–23] this study evaluated the dietary patterns of individuals (n = 296) who responded affirmatively to the question "Are you following a low cholesterol diet?" We assumed that individuals following this diet were attempting dietary modification to reduce their blood cholesterol levels and hence their risk of coronary heart disease. Multiple responses to define the diet were permitted in the survey. For example, among subjects stating adherence to a "low cholesterol diet," 55 percent also reported following a "low calorie diet," 68 percent followed a low fat diet, 12 percent followed a "diabetic diet," and 54 percent followed a "low salt diet." Respondents who reported following other diets (i.e., high blood pressure, diabetes) in addition to the "low cholesterol" diet were included in this study.

TABLE 1—Characteristics of Study Participants

	Dieters[a]	Non-dieters[b]
N	296	10052
Age (SD)	50.3(14)	39.9(16)
Sex (% male)	49.7	44.7
Race (% White)	91.2	89.8
BMI[c] (SD)	26.6 (4.8)	24.9 (4.8)
History of CAD (%)	25	3
Smoking (%)	25	38
Poverty Index[d]	2.9 (1.5)	2.7 (1.5)
Grade Level[e]	12.2 (2.8)	12.0 (3.1)

[a]Dieters-Study participants who reported adherence to a low cholesterol diet.
[b]Non-dieters-Study participants who did not report adherence to a specific diet.
[c]Kg/m2.
[d]Poverty Index-financial income adjusted to geographic poverty level for family unit; values of less than 1.0 are below the poverty level.
[e]Grade Level-Highest grade completed.

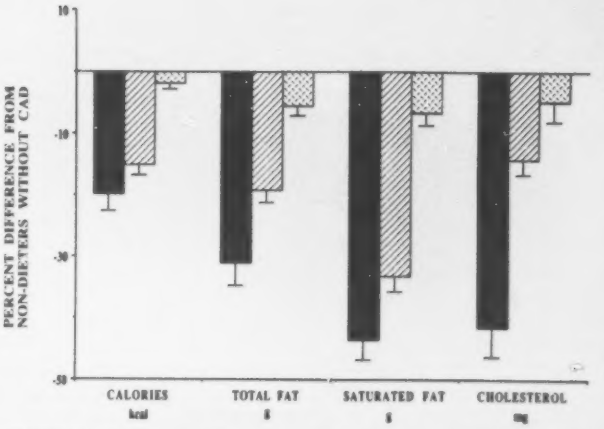

FIGURE 1—The Percent Difference in Caloric, Total Fat, Saturated Fat, and Cholesterol Intake of Dieters with CAD (black bars; N = 72), Dieters without CAD (hatched bars; N = 217), and Non-dieters with CAD (speckled bars; N = 323) compared to Non-dieters without CAD (N = 9681)

A total of 12,501 adults attended the second visit. However, respondents who were following special diets other than a low cholesterol diet (i.e., diabetic, hypertensive, low sodium, low calorie, n = 1,608), who did not have sufficient financial information recorded to compute a poverty index (n = 390), who did not record their level of education (n = 100), or who were judged by the interviewer to have unsatisfactory 24-hour recalls (n = 55) were excluded from the analysis. After these exclusions, 10,348 respondents remained eligible for the study. Those who attended the second visit but were subsequently excluded (n = 2,153) were more likely than included subjects to be male (49.2 vs 38.3 percent), but were otherwise similar to included subjects in percent who were White (89.0 vs 86.4 percent), not following a low cholesterol diet (97.4 vs 96.7 percent), and in their mean age (50.9 vs 45.2 years).

Individuals who were not adhering to any special diet (n = 10,052) were then compared with participants who indicated that they were following the low cholesterol diet (n = 296). In this paper, respondents who reported adherence to the low cholesterol diet are referred to as "dieters"; those subjects not following a diet are referred to as "non-dieters."

Statistical Analysis

Variance estimates were corrected for the complex sample design using the SUPERCARP program.[24] Multiple regression[25] was used to determine the effect of diet status (i.e., dieter compared with non-dieter) on the dietary intake of each nutrient or food group, after adjusting for age, gender, educational level, poverty index, race, and number of cigarettes smoked daily. The body mass index (BMI) was not used

TABLE 2—Intake of Protein, Carbohydrate and Fat among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Calories (kcal)	2072	1735	−16.3 (−23.7, −8.9)
Protein (g)	80.2	73.7	−8.0 (−15.8, −0.3)
Carbohydrate (g)	225.6	196.5	−12.9 (−21.1, −4.7)
Fat (g)	85.4	66.4	−22.1 (−30.8, −13.3)
Saturated (g)	30.7	23.2	−24.5 (−33.7, −15.4)
Monounsaturated (g)	31.6	24.2	−23.3 (−33.7, −12.8)
Polyunsaturated (g)	11.9	9.7	−18.6 (−30.1, −7.2)
Cholesterol (mg)	363	287	−20.8 (−31.8, −9.7)

TABLE 3—Percent of Total Caloric Intake for Protein, Carbohydrate and Fat among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

	Percent of Total Caloric Intake		
Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Protein	15.6	18.3	16.8 (6.8, 26.8)
Carbohydrate	44.6	46.4	4.1 (0.3, 7.8)
Total Fat	36.5	32.8	−9.9 (−13.5, −6.3)
Saturated Fat	13.0	11.1	−14.4 (−19.7, −9.1)
Monounsaturated Fat	14.9	12.9	−13.6 (−19.0, −8.2)
Polyunsaturated Fat	5.8	5.5	−3.8 (−15.3, 7.8)

TABLE 4—Mean Intake of Vitamins and Minerals among Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrients	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Vitamin A (IU)	5249	5284	+0.7 (−25.6, 27.0)
Thiamin (mg)	1.33	1.38	+4.3 (−15.0, 23.7)
Riboflavin (mg)	1.84	1.82	−1.2 (−17.9, 15.4)
Niacin (mg)	21.1	20.4	−3.1 (−11.4, 5.2)
Ascorbic acid (mg)	100.0	118.3	+18.3 (0.4, 36.2)
Calcium (mg)	778	688	−11.5 (−23.4, 0.4)
Iron (mg)	13.7	12.5	−9.0 (−18.4, 0.4)

TABLE 5—Mean Vitamin and Mineral Nutrient Density (nutrient/1000 kcal) of Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Nutrient Density (nutrient/1000 kcal)	Non-Dieters Intake	Dieters Intake	Percent Difference Dieters vs Non-Dieters (99% CI)
Vitamin A (IU/1000 kcal)	2618	3790	+34.5 (−3.4, 72.4)
Thiamin (mg/1000 kcal)	0.66	0.89	+35.0 (0.7, 69.7)
Riboflavin (mg/1000 kcal)	0.91	1.18	+30.3 (−4.2, 64.9)
Niacin (mg/1000 kcal)	10.59	13.42	+26.7 (9.1, 44.4)
Ascorbic acid (mg/1000 kcal)	52.7	85.9	+60.1 (39.8, 80.4)
Calcium (mg/1000 kcal)	381	433	+13.8 (−4.9, 32.4)
Iron (mg/1000 kcal)	6.89	7.95	+15.4 (6.2, 24.6)

as a covariate in the analyses presented in this paper because of the potential effect that diet status may have on body weight. However, separate analyses were also performed in which the BMI was included as an additional covariate. All covariates were forced into the model. Dependent variables not approximating a normal distribution were transformed logarithmically; these included saturated fat, milk, poultry, organ meats, vitamins A and C, and the nutrient density of

vitamins A, B_1, B_2, and iron. However, the results of the analysis were not affected by this transformation and, for purposes of clarity and consistency, the data are presented untransformed. The Cook's distance was determined for residuals and found to be less than 0.2, indicating that outliers did not significantly bias the results of the analyses.[26] In Tables 2, 3, 4, and 6, the adjusted mean values for both non-dieters and dieters are given, as well as the percent difference between the dieters and non-dieters. Because of the large number of analyses performed, the 99% CI, rather than the 95% CI, was employed in this paper and is provided in the tables; negative values indicate that the mean intake for dieters is lower than the intake for non-dieters.

Results

Characteristics of dieters are compared with non-dieters in Table 1. Dieters were older, more likely to be female, and have slightly lower BMI than non-dieters, but were similar in poverty index, education level, and race. Dieters were also more likely to have coronary artery disease and less likely to be smokers. The mean duration that the diet had been followed was 147 weeks. A diet prescription was received from a health professional (dietitian, nurse or doctor) by 69 percent of dieters.

Total caloric intake was 16 percent lower (99% CI = −24, −9) in dieters compared with non-dieters, representing a decrease of 340 kcal (Table 2). Although part of this decrease was due to reduced protein and carbohydrate intake among dieters, most of the reduction in total calories was due to a 22 percent (99% CI = −31, −13) decrease in total fat intake. This reduction in total fat intake resulted from lower saturated, monounsaturated, and polyunsaturated fat consumption. Mean cholesterol intake was also reduced in dieters compared with non-dieters (21 percent decrease; 99% CI = −32, −10).

The composition of the diet among dieters also differed compared to non-dieters (Table 3). Dieters had a higher percent of total calories obtained from protein and carbohydrate and a lower proportion of total calories derived from total fat, saturated fat, and monounsaturated fat. The percent of total calories derived from polyunsaturated fat was similar between dieters and non-dieters. Dieters were more likely to meet goal intake levels for the cholesterol lowering diet by restricting total fat to 30 percent of the diet, saturated fat to 10 percent of the diet, and cholesterol intake to 300 mg daily. For dieters, 35.2 percent met this goal for total fat compared with 22.3 percent for non-dieters, while 41.7 percent of dieters and only 23.2 percent of non-dieters met this criteria for saturated fat. For dietary cholesterol, 66.5 percent of dieters, compared with 54.3 percent of non-dieters, achieved the goal level for daily intake.

Because the presence of coronary artery disease (CAD) may also motivate individuals to consume more healthful foods without consciously following a cholesterol lowering diet, the influence of previous CAD on dietary intake was evaluated. Compared to non-dieters without CAD, dieters both with and without a previous history of CAD had decreased caloric, total fat, saturated fat, and cholesterol intake (Figure 1). On the other hand, non-dieters with a history of CAD had caloric, total fat, saturated fat, and cholesterol intake similar to non-dieters without a CAD history.

Despite the decreased caloric intake, dieter vitamin and mineral intake was not significantly reduced compared with non-dieters (Table 4). This finding occurred because dieters selected foods with increased nutrient density (nutrient/1000 kcal). Marked increases in nutrient density were observed in dieters compared with non-dieters for vitamin A, thiamin, riboflavin, ascorbic acid, and niacin (Table 5). Smaller increases in nutrient density for iron and calcium were observed, and the total intake of these minerals in dieters was slightly decreased.

Dieters selected food that conforms to dietary guidelines for a cholesterol lowering diet (Table 6). They consumed 45 percent less whole milk products and 60 percent more skim milk products than non-dieters. Dieters ate 30 percent fewer eggs than non-dieters, and consumed 15 percent less meat. Consumption of poultry and fish were significantly increased compared with non-dieters.

Subsequent analyses were performed using the BMI as an additional covariate. All differences between dieters and non-dieters persisted; inclusion of this variable as a covariate had very little effect on the results for any nutrient, suggesting that the observed differences between groups was not attributable to body mass.

Discussion

Americans reporting adherence to a cholesterol lowering diet had dietary patterns which differed markedly from study participants who were not following any diet. Dieters reduced their intake of calories, total fat, and saturated fat. They consumed less red meat and whole milk and consumed more poultry, fish, and skim milk. Despite these significant differences in diet between dieters and non-dieters, dieters maintained their intake of essential vitamins and minerals by selecting food with increased nutrient density. These dietary trends are in accordance with dietary guidelines designed to reduced blood cholesterol levels. The findings suggest that dieters had sufficient nutritional understanding to make

TABLE 6—Mean Servings from Specific Food Groups Consumed Weekly by Americans Reporting Adherence to a Low Cholesterol Diet (Dieters) Compared with Americans not Following a Specific Diet (Non-dieters)

Food Groups	Non-Dieters Servings/week	Dieters Servings/week	Percent Difference Dieters vs Non-Dieters (99% CI)
Whole milk products	4.55	2.52	−44.7 (−58.6, −30.8)
Skim milk products	2.92	4.66	+59.5 (22.9, 96.1)
Meat	6.53	5.58	−14.6 (−23.3, −5.9)
Organ meats (i.e. liver)	0.38	0.31	−19.2 (−34.6, −3.8)
Poultry	1.34	1.71	+27.0 (8.8, 45.2)
Fish	1.02	1.42	+39.1 (12.4, 65.7)
Eggs	2.68	1.88	−29.7 (−43.5, −15.9)

specific food choices to reduce dietary cholesterol, total fat, and saturated fat without compromising the nutrient intake of their overall diet.

Since NHANES II was conducted from 1976–80, this study reflects the prevailing dietary recommendations during that period. Many advances in the current understanding of the cholesterol lowering effect of specific nutrients have occurred within the past decade.[27] However, the dietary guidelines to lower blood cholesterol levels were formulated in the 1970s and included the recommendations for the reduction of total fat, saturated fat, and dietary cholesterol,[15,16,21–23] similar to those recommended currently.[5] Although the reported dietary intake of dieters was consistent with these recommendations, the extent that knowledge of these guidelines actually influenced and/or motivated dieters to improve their diets cannot be determined from a cross-sectional study of this nature.

It is of interest that 31 percent of the dieters did not receive a diet prescription from a health professional (dietitian, nurse or physician) and decided on their own to follow a cholesterol lowering diet. In fact, they adhered to the cholesterol lowering diet as well as individuals who had received a diet prescription (data not shown). This is important because individuals with desirable (<200 mg/dl) or borderline high blood cholesterol levels (200–239 mg/dl) may be less likely to be referred by a health professional for dietary counseling compared with persons with higher values.[28] Since even individuals with relatively desirable cholesterol levels may benefit from a cholesterol lowering diet, it is reassuring that many Americans can successfully follow this diet even without formal consultation with a dietitian.

This study is limited by the methodological problems associated with estimating dietary intake in cross-sectional studies of free-living populations. Dietary recall of the preceding 24-hour period may not provide an accurate or representative assessment of current nutritional intake for an individual. However, this limitation is lessened when assessing the average intake of a group, since the group mean intake is less sensitive to day-to-day variability.[29] Further error in the data can be introduced during the reporting and recording of food intake, as well as in the analysis of the nutrient content of food.[30] During NHANES II, efforts were made to reduce these sources of error by employing trained interviewers and using a uniform approach to nutrient analysis during the entire survey. Because data from both dieters and non-dieters were acquired using the same methodology, error introduced from these sources would not be likely to bias the results of this study. An additional concern is that questions concerning adherence to a special diet may have biased dietary recall data. For example, individuals who reported adherence to the low cholesterol diet may have underreported ingestion of foods high in cholesterol and saturated fat. However, the questions concerning adherence to specific diets were among hundreds asked of each respondent and were obtained by interviewers who were unaware of the goals of the current study. Therefore, it is unlikely that questions describing the diet biased the reporting of nutrient intake data.

The results of a cross-sectional study alone cannot define the presence of a causal relationship. For example, individuals who naturally prefer diets particularly low in cholesterol and fat may report that they are following a low cholesterol diet. These individuals may not have modified their food intake due to the adoption of a diet. However, this hypothesis is unlikely because dieters, compared with non-dieters, had a much higher prevalence of coronary heart disease, suggesting that they modified their diet in response to this illness. Further, 69 percent of dieters had received the diet prescription from a health professional. Therefore, a more likely explanation for our results is that dietary intake was modified as a consequence of the adoption of the diet.

Diet modification can effectively reduce blood cholesterol levels. Studies specifically designed to evaluate the efficacy of cholesterol lowering diets report cholesterol reductions in the range of 10–15 percent.[11,31–33] Further, the diet is well tolerated.[34] However, perfunctory and routine dietary instruction in patients without proper motivation may have no impact on dietary compliance, weight reduction, or blood cholesterol levels.[35,36] In our study, only subjects who reported that they were following a cholesterol lowering diet were included as dieters. Therefore, individuals prescribed a cholesterol lowering diet but who were unwilling or unable to comply would probably not report adherence to the low cholesterol diet at the time of the survey, and would be excluded from the analysis. Thus, this report is likely to include only respondents who were sufficiently motivated to adopt this diet, and who felt that they were, at least in part, successful. Only 3 percent of the total sample, including only 15 percent of those with coronary heart disease, reporting following the low cholesterol diet. The willingness of the American public to accept this diet will be one important factor determining general compliance and efficacy.

Results from this study indicate that adherents to the cholesterol lowering diet report consumption of foods and nutrients consistent with recommended cholesterol lowering diet guidelines. In addition, the intake of essential vitamins and minerals is not compromised by adherence to this diet. Since the NHANES II survey utilizes a population-based sample, subjects included in the analysis represent a cross-section of the American public. Therefore, this analysis suggests that favorable dietary modifications can be achieved by Americans who choose to follow this diet. Efforts to increase the American public's awareness of the importance of dietary intervention to reduce cholesterol levels and thereby decrease coronary heart disease risk should be emphasized. Once Americans are prepared to adopt the cholesterol lowering diet, these results suggest that healthful dietary modification in accordance with accepted guidelines to reduce blood cholesterol levels is an achievable goal.

APPENDIX

NHANES II Questions Asked to Describe Diet

	Response
Are you on a special diet?	Yes/No
If yes, is this diet to lose weight?	"
Gain weight?	"
For diabetes?	"
For kidney failure?	"
For ulcers?	"
For diverticulitis?	"
For allergies?	"
For heart trouble?	"
For high blood pressure?	"
For pregnancy? (females only)	"
For any other reason?	"
What kind of diet is it?	
	Yes/No
Low fat?	"
Low protein?	"
High protein?	"

APPENDIX, cont.

	Response Yes/No
Low salt?	"
Low carbohydrate?	"
Low sugar?	"
Low caloric?	"
Low cholesterol?	"
High caloric?	"
Vegetarian with animal by-products? (egg, dairy, etc.)	"
Vegetarian without animal by-products?	"
A bland diet?	"
Some other type?	"
Was this diet prescribed by a doctor, dietitian or nurse?	"
Length of diet (in weeks)	

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Spanish Version of the Nottingham Health Profile: Translation and Preliminary Validity

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Abstract: We report the transfer into Spanish of a multidimensional measure of perceived health originally developed in Great Britain, the Nottingham Health Profile (NHP), and an assessment of the preliminary validity of the version is presented.

Translation of the questionnaire was obtained from experts and from a Spanish monolingual lay group. Construct validity of the version was assessed in two studies: testing relationship of NHP scores to other self-reported measures of health in a general population survey; and comparing NHP scores for a group of frequent users and for a group of non-users of primary health services.

Mean scores of NHP dimensions were higher for people with poorer self-reported health and higher for the frequent health services users than for the non-users.

Findings suggest that the Spanish version of the NHP is culturally equivalent to the original questionnaire, and has a similar level of construct validity. Nevertheless, further research on reliability and on the weighting system is required to establish the equivalence of the Spanish version definitively. (*Am J Public Health* 1990; 80:704–708.)

Introduction

The impetus to produce reliable and valid measures of health has grown dramatically as the need for better evaluation of health services has increased. In the last two decades a number of questionnaires have been designed which measure the physical, mental, and social aspects of health.[1] Although most of these measures have been developed and tested in English-speaking cultures, mainly in the United States, they are widely used in different countries and in non-English-speaking cultures.

The content of health status measures as well as the wording and the form of administration are very dependent on cultural values.[2,3] Transferring a validated instrument to a different culture is an efficient strategy, since developing a new measure is a complex and time-consuming task, requiring much expertise. However, this process should follow a methodology that ensures a similar level of validity, reliability, and cultural meaning for the new version.[4,5] This methodology includes several steps: translation (ensuring conceptual rather than linguistic equivalence), and testing for feasibility, validity, and reliability. Furthermore, measures containing scaled items usually include specific weights for each item, thus making it necessary to validate the weights for the adapted version as well, in order to assure that scores obtained in the original and adapted version have similar meaning.[6,7]

In past work[8] we identified the need for a measure to adequately monitor health status of the population and equity in health services utilization. We have focused our work on adapting a validated measure rather than on developing a new one. We selected the Nottingham Health Profile (NHP), a validated measure of perceived distress developed in Great Britain,[9–12] and previously adapted in Sweden[7] and France.[13]

The NHP is a self-administered questionnaire containing a first part with 38 statements belonging to six dimensions of health (Energy, Pain, Emotional Reactions, Sleep, Social Isolation, and Physical Mobility). The respondent answers "yes" if the statement adequately reflects his/her current status or feeling, or "no," otherwise.[9] British researchers weighted each item in a dimension using Thurstone's paired comparisons method.[14] Part II contains questions on seven areas of daily life.

For transferring the NHP into Spanish, the following strategy was designed: first, a conceptually equivalent translation; second, a preliminary assessment of the validity of the version obtained; and finally develop Spanish weights, contingent upon the establishment of the validity of the unweighted version.

Methods

Translation Process

Ideally, a panel of bilingual people should be used to obtain translations and blind back-translations.[15] Because it was difficult to find a satisfactorily representative panel of bilinguals (same fluency in Spanish as in English, middle/lower socioeconomic class, and experiencing chronic illnesses), a specific methodology for adapting the NHP into Spanish was designed by one of the NHP authors (S. Hunt).[16] After complete explanations by Dr. Hunt, translations of each item were obtained by two of us (JMA and JA). These translations were back-translated into English and discussed again. A list containing translated items and alternative translations was made. Close agreement was reached in most items and they were graded as A "satisfactory agreement" (32 out of 45, 71 percent), B "almost satisfactory but one or two words uncertain" (nine items, 20 percent), and C "doubtful translation" (four items, 9 percent).

In order to overcome these disagreements, a group of 10 Spanish unskilled workers listened to anecdotes that reflected all the items on the questionnaire and described them freely in their own words. These words were added to the list of alternative translations. This list was later presented to the same group in order to point out the expressions they found odd and to select those that would conform to their language usage. The final version included the most popular expressions for items B and C (see Appendix). A pre-test of this version was carried out with a group of cardiology and rheumatology hospital outpatients who were asked to complete the questionnaire and to identify the statements difficult to understand, those that were acceptable, and those easily understood.

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Having obtained a Spanish version of the NHP, a translation into Catalan (language of romanic origin similar to Spanish, spoken by some eight million people mainly in the northeastern part of Spain) was also made. Seven Catalan-Spanish bilinguals, who were easy to convene, were used to obtain translations and back-translations and also to decide the exact final wording. The close agreement between both Catalan and Spanish versions led us to include both versions in further studies of the Spanish NHP.

Validation Studies

Achieving conceptual and linguistic equivalence when transferring a questionnaire from one culture to another does not entirely guarantee that the validity of the original version stands in the new culture. Although the method used to translate the NHP into Spanish followed prescribed procedures, the results of this translation were further tested to determine whether it performed in a manner similar to the British NHP.

In order to make an assessment of the validity of the Spanish version of the NHP, construct validity was tested in two studies. First, the relationship of NHP scores to other self-reported measures of health was tested; second, NHP scores for a group of frequent users and for a group of non-users of primary health services were compared.

In the first study, 1,220 participants ages 41 years and over completed the NHP (75 percent Spanish version and 25 percent Catalan version) as part of a random sample health survey of the city of Barcelona.[17] They also provided information about self-rated health (SRH), chronic conditions selected from a list of 34 conditions, chronic limitation of activity due to health problems, and restricted activity in the two weeks before the interview. These variables had been used in previous studies in Spain.[18,19] Social class was assigned to the respondents based on the occupation of the head of the household, following an adaptation[20] of the British Registrar's Social Classification.[21]

In the second study, clinical records of individuals ages 20 and over were sampled systematically from the registry of the Health Center of Barañain (Navarra). Following the criteria of a previous validity study of the British NHP,[11] people were classified as "non-users" if no medical visits were registered in the last six months, and as "frequent users" if three or more visits were made by individuals less than 50 years old, or five or more visits for individuals age 50 and over. As illness was the construct of interest, routine medical examinations were not included. In total, 396 people were considered eligible for the study (195 "non-users" and 201 "frequent users"). They were asked to complete the Spanish version of the NHP and to give information on self-rated health and chronic conditions. Complete responses were obtained from 338 individuals (85.3 percent of the eligible). Following the previous British study,[11] influence of age and sex in the analysis was eliminated by randomly selecting from the respondents a subsample of 276 people containing equal numbers of "non-users" and "frequent users" in each age and sex group. Four age ranges were categorized by 10-year intervals (20–29, 30–39, 40–49, and 50 and over).

We calculated scores for each NHP dimension in these studies by adding all the answers to the items in the dimension, giving the value of 0 for answers of "no" and 1 for answers of "yes". To obtain a final score in each dimension, and to overcome the fact of having a different number of items in some of the dimensions, each sum was multiplied by 100 and divided by the number of items in the dimension. Possible scores ranged from 0 (all answers of "no" in the dimension, denoting absence of distress) to 100 (all answers of "yes", denoting maximal distress). Statistical significance was assessed using nonparametric tests (one-tailed Wilcoxon-Mann-Whitney, and Kruskal Wallis tests at a 5 percent level of significance[22]) because NHP responses do not follow a normal distribution.[9]

Mean scores of the NHP for the general population ages 41 and over of the city of Barcelona are presented in Table 1. Highest mean scores corresponded to Sleep and to Emotional Reactions (23.2 and 20.8, respectively), while the lowest was that of Social Isolation (7.2). Women scored considerably higher than men in all the dimensions. Mean scores were higher for social classes IV–V (manual workers) than for social classes I–II (directors, professionals and others).

People rating their health as "very good" scored 2.9 in Energy, 3.7 in Pain, and 4.9 in Social Isolation, as compared with those with "poor" or "very poor" health, scoring 64.3, 58.0, and 21.4, respectively (Table 2). People with at least one of the 34 chronic conditions listed scored significantly higher than those not reporting any condition, the score being higher as the number of conditions increased. Similar differences in

TABLE 1—Mean Scores for each Dimension of the Spanish Version of the NHP in the General Population Ages 41 and over of Barcelona.

	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Total	14.1	15.6	20.8	23.2	7.2	14.9 (1,220)
Sex						
Male	8.5	9.1	16.1	17.6	5.7	10.6 (578)
Female	19.0	21.5	25.0	28.2	8.6	18.8 (642)
Age group						
41–49	8.9	10.7	19.8	16.7	4.4	9.0 (320)
50–59	12.6	14.4	20.9	20.7	7.0	11.5 (405)
60–69	15.4	18.4	18.7	27.1	7.8	17.5 (292)
70+	23.3	21.8	25.0	32.8	11.5	27.3 (203)
Social Class (age-adjusted scores)*						
I–II	10.3	8.0	15.7	16.2	4.3	11.2 (256)
III	10.5	11.5	15.1	18.0	5.5	12.9 (299)
IV–V	17.0	20.8	25.5	27.1	8.9	17.4 (606)

*59 individuals not classified.

TABLE 2—Mean Scores for Each Dimension of the Spanish Version of the NHP by Level of Self-reported Health Variables (convergent validity variables), General Population Ages 41 and over of Barcelona

Characteristics	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Self-Rated Health*						
Very good	2.9	3.7	12.1	12.0	4.9	7.0 (119)
Good	6.1	7.8	14.8	17.2	4.9	9.0 (692)
Fair	25.5	28.1	30.6	34.2	10.6	23.8 (350)
Poor or very poor	64.3	58.0	47.4	56.1	21.4	49.3 (59)
Chronic Conditions**						
No	3.4	3.5	11.4	10.6	2.6	4.3 (245)
Yes	16.8	16.7	23.2	26.4	8.4	17.7 (975)
Number of Chronic Conditions*						
0	3.4	3.5	11.4	10.6	2.6	4.3 (245)
1	5.5	7.4	16.8	17.4	5.7	9.3 (278)
2	12.3	13.9	19.6	24.0	7.5	13.9 (237)
3	18.1	21.6	24.3	23.3	8.3	18.9 (161)
4	19.0	24.0	26.9	31.6	9.2	21.5 (129)
5+	38.5	37.2	35.0	43.3	14.3	32.1 (170)
Chronic Limitation of Activity**						
No	10.7	12.5	19.0	21.0	6.6	12.5 (1,086)
Yes	42.0	41.0	35.0	42.1	12.6	35.1 (134)
Restricted Activity Days (in the past two weeks)**						
No	11.9	13.8	19.7	22.1	6.8	13.4 (1,133)
Yes	41.8	39.6	33.5	37.3	12.3	33.4 (87)

*Kruskal-Wallis test: $p < 0.0001$, for all dimensions.
**Wilcoxon-Mann-Whitney one-tailed test: $p < 0.0001$, for all dimensions.

size and direction were found for chronic limitation of activity and for restricted activity days (Table 2). Logistic regression analysis showed that NHP scores remained significantly associated with the construct validity variables, when age, sex, and social class were taken into account.

As shown in Table 3, the mean scores in each of the NHP dimensions were higher for the "frequent users" than for the "non-users." The most important relative differences corresponded to Pain and Energy dimensions (50 percent or more increase). Also, people reporting better health scored lower than those reporting worse health. NHP scores showed an ascending gradient related to reported number of chronic conditions (Table 3).

Discussion

Accurate translation is a first requirement in the process of transferring a health measure from the culture of origin to another culture.[2,5,15,23] However, it is a common practice that researchers themselves directly translate health questionnaires using technical and/or uncommon expressions which can affect validity, reliability, and comparability of results.

The translation methodology used for transferring the NHP into Spanish ensured a conceptually equivalent version.[24] The high level of agreement achieved in most back-translated items supports this equivalence. The participation of the monolingual lay panel in the translation ensured that statements of the new version were based on lay

TABLE 3—Mean Scores for each Dimension of the Spanish Version of the NHP by "Non-users" and "Frequent Users" and by other Convergent Validity Variables: Study of the Health Center of Barañain (Pamplona) (n = 276)

Characteristics	Energy	Pain	Emotional Reactions	Sleep	Social Isolation	Mobility (n)
Use of Health Services**						
Non-users	8.7	8.3	16.9	16.7	6.4	10.2 (138)
Frequent users	13.0	13.8	23.9	23.8	8.8	13.8 (138)
Self-Rated Health*						
Very good	0.0	2.1	7.7	7.8	2.1	3.6 (38)
Good	4.5	6.6	15.4	14.1	6.2	7.1 (158)
Fair	25.1	21.9	35.2	39.1	10.3	25.0 (69)
Poor or very poor	57.6	37.5	50.6	24.0	30.9	33.7 (11)
Number of Chronic Conditions*						
0	5.3	1.0	10.2	8.4	3.1	3.9 (38
1	7.1	5.8	12.8	13.8	3.1	8.2 (66
2	10.8	11.2	20.8	17.4	8.2	11.5 (78)
3	12.6	11.3	27.3	21.4	13.6	15.6 (45)
4	14.1	25.0	28.5	36.4	11.5	20.6 (26)
5+	24.4	27.2	35.5	52.4	10.0	25.6 (23)

*Kruskal-Wallis test: $p < 0.01$, for all dimensions.
**Wilcoxon-Mann-Whitney one-tailed test: $p < 0.05$, for all dimensions.

expressions of health perceptions, as the original instrument. The fact that similar expressions had been obtained for other European versions of the NHP, further suggests that NHP items address aspects of health perceptions that have conceptual equivalence for these cultures.[7,13]

As mentioned above, the strategy for transferring the NHP into Spanish included the assessment of validity of the version before obtaining the weights of the items. As there is no gold standard against which validity of complex health status measures could be evaluated,[1,25,26] construct validity rather than criterion validity of the Spanish version has been assessed.

The results of the studies presented preliminarily suggest that the unweighted Spanish version of the NHP has construct validity. Mean scores in every NHP dimension are clearly different for individuals with diverse degrees of perceived health as assessed by a variety of other construct validity variables. All differences were in the expected direction: the lower the level of the perceived health indicator the higher the NHP score. The similarity of the results from both studies, with different designs and populations, further supports the validity of the questionnaire.

A similar distribution of NHP scores across different self-rated health (SRH) categories has been observed in several studies in Britain.[9,27,28] SRH has been shown to be a good predictor of health outcomes, even after adjusting for a number of variables including "objective" health status.[29,30] The consistency and the strength of the relationship between NHP scores and SRH is evidence of the validity of the questionnaire. In both studies presented, an increase was observed in mean NHP scores as the number of chronic conditions increased. Even though the severity of the conditions included was not considered, and the fact that conditions reported may reflect both medically diagnosed and patient perceived health problems,[31–33] it was expected that, on the average, people reporting more health conditions would have a worse perception of their health.

The approach of the first study for assessing construct validity, i.e., to compare scores for the new measure for different levels of self-reported health variables, has been previously utilized.[26,34,35] Most of these studies have reported high correlations among the health measures and the construct validity variables, similar to the results presented for the Spanish NHP. However, the relationship found between NHP responses and construct validity variables may be overestimated as responses to both were obtained directly by the respondent.

Conversely, in the second study, the number of visits was obtained from clinical records, independently of the measure of the responses to NHP. Thus, mean score differences between non-users and frequent users provide further evidence of the validity of the Spanish version of the NHP. However, these differences, although statistically significant, are not very large. This seems to be due to the fact that the scores of the "frequent users" are not so high as might be expected. The most likely explanation of this could be that people in the poorest health were misclassified due to a reduced likelihood of home visits by the doctor being recorded. Another explanation can be found in the fact that many of the "non-users" are non-symptom-free. A comparison of the distribution of health problems suggests that highest differences between these two groups were for conditions which have low potential impact on NHP scores (e.g., hypertension and hearing problems).

Nevertheless, to completely assess the equivalence of the questionnaire, similarity of the weights of the items should be established, as it has been done for other NHP European versions[7,13] and for the British version of the Sickness Impact Profile.[6] As mentioned above, the British authors of the NHP developed weights for the items in each dimension, using Thurstone's method of paired comparison;[14] weights being calculated by considering the proportion of times each statement was judged to be more severe than each of the other statements in the dimension by a representative group of lay people.

Developing the weights for the Spanish version would provide an important basis for definitively assessing validity of the questionnaire and for ensuring cross-cultural comparability.

APPENDIX

English and Spanish Versions of Selected Items of the Nottingham Health Profile (and graded agreement of back-translation)

British Item	Spanish Item	(Agreement)
Energy		
The days seem to drag	Los días se me hacen interminables	(A)
I'm tired all the time	Siempre estoy fatigado	(C)
Pain		
I have pain at night	Tengo dolor por las noches	(A)
Emotional Reactions		
Worry is keeping me awake at night	Las preocupaciones me desvelan por la noche	(A)
I lose my temper easily these days	Ultimamente me enfado con facilidad	(B)
Things are getting me down	Las cosas me deprimen	(C)
Sleep		
I take tablets to help me sleep	Tomo pastillas para dormir	(A)
I'm waking in the early hours of the morning	Me despierto antes de hora	(C)
Social Isolation		
I'm finding it hard to get on with people	Me cuesta llevarme bien con la gente	(A)
I'm finding it hard to make contact with people	Ultimamente me resulta difícil contactar con la gente	(C)
Physical Mobility		
I can only walk about indoors	Sólo puedo andar por dentro de casa	(A)
I find it hard to reach for things	Me cuesta coger las cosas	(C)

(A) Satisfactory translation
(B) One or two words uncertain
(C) Doubtful translation

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Public Health Briefs

Evaluating Community-based Nutrition Programs: Assessing the Reliability of a Survey of Grocery Store Product Displays

Allen Cheadle, PhD, Bruce Psaty, MD, PhD, Edward Wagner, MD, MPH, Paula Diehr, PhD, Thomas Koepsell, MD, MPH, Susan Curry, PhD, and Michael Von Korff, PhD

Abstract: A pilot test of a survey of grocery store product displays was conducted to measure the amount of health-education information provided and the proportion of the display devoted to "healthier" products. Inter-rater reliability ranged between 0.73 and 0.78 for the healthiness indices and between 0.30 and 0.67 for the health education measures. Test-retest reliability ranged from 0.44 to 1.0. (*Am J Public Health* 1990; 80:709–711.)

Introduction

There have been a number of recent efforts directed at entire communities to modify dietary behavior in order to reduce the risk of cardiovascular disease and cancer.[1–4] Evaluation of these interventions requires "community-level indicators" of program effectiveness—measures of the community environment that are not merely aggregates of individual behavior.[5] We measured the reproducibility of a survey of grocery store product displays to be used as part of the evaluation of community-based nutrition programs. Such surveys are particularly useful when grocery stores and supermarkets play a role in the intervention, as in the Giant Foods project[1] and in the Henry J. Kaiser Family Foundation Community Health Promotion Grants Program (CHPGP).*

Methods

We surveyed four general product areas within each grocery store: fresh produce, meat, milk, and bread. For each major product area, two types of information were recorded: presence or absence of health-promotion items, and the physical dimensions of product displays. The protocol took 30–45 minutes for an experienced rater to complete.

- *Health-promotion items* were defined as health education activities (usually printed material) on or near the displays, which provided nutritional information or increased awareness of healthy food choices, and which were independent of product packaging. Examples of health-promotion items included: shelf labeling, posters, recipes, and store health-information centers.
- *The measurements of the displays* were designed to create an index of the relative amount of space occupied by "healthy" products in each product area, calculated as the proportion of shelf space devoted to such products (e.g., those low in fat) relative to the overall size of the display of similar foods.

The sampling frame consisted of full-service supermarkets, defined as grocery stores with two or more check-out stands, carrying fresh produce and fresh meat, that were listed in the Yellow Pages telephone directory. The sample for the Seattle pilot test of inter-rater reliability included 13 independent stores and 24 chain stores. For this part of the field test, each store was surveyed by two raters who visited the four product areas in a different sequence. Test-retest reliability was evaluated on an additional random sample of 61 stores in five communities that are part of the overall Kaiser Foundation evaluation. These test-retests were carried out by the same rater on some occasions and by different raters on others, with a variety of time intervals separating the visits.

Three indices of display "healthfulness" are presented here: the proportion of poultry and fish in the meat display, the proportion of reduced-fat milk in the milk display, and the proportion of 100% whole wheat bread in the bread display. Health promotion items were coded simply as present or absent for each of the major product areas. To assess reliability, we used the intra-class correlation coefficient[6] for the continuous variables and weighted kappas[7] for the discrete variables.

Results

As Table 1 shows, chain stores (particularly national or regional chains) more often offered health promotion items than did independent stores. By contrast, there was little apparent difference in the three display variables by store type. Table 2, column (1) gives inter-rater reliability coefficients computed for the first visits to the 37 stores in the pilot sample. Reliability for the display variables was fairly high, in the range of .7 to .8, but was lower for the health promotion items. The low figures for the milk and meat health promotion items were partly due to an open-ended format for recording these items in the pilot survey; a closed format used in the later surveys produced better results. The figures in column

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*Wagner EH, Kehrer B: The Henry J. Kaiser Family Foundation Community Health Promotion Program: Evaluation design considerations. Presented at the annual meeting of the International Epidemiological Association, Helsinki, Finland, August 6–13, 1987.

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TABLE 1—Means of Grocery Store Survey Variables, by Store Ownership Category, Seattle Pilot Test Stores

Variables	Independent (n = 13)	Chains		
		Local (n = 13)	National/Regional (n = 12)	All (n = 37)
Health promotion items[a]				
Produce display	46.1	50.0	100.0	64.9
Meat display	69.2	100.0	100.0	89.2
Milk display	30.8	41.7	50.0	40.5
Display variables[b]				
% Poultry + fish	44.4	50.6	43.8	46.2
	(4.9)	(6.9)	(5.8)	(6.5)
% Reduced-fat milk	67.9	66.6	62.5	65.7
	(8.9)	(6.4)	(9.8)	(8.6)
% Whole-wheat bread	5.9	5.8	4.4	5.4
	(2.7)	(2.1)	(1.2)	(2.1)

NOTES: Standard deviation in parentheses.
[a]Percent of stores with health education materials in the indicated section. Store was counted as having health promotion items if either of the two raters recorded them.
[b]Percent of the display with indicated products. Measurements for each store are the average of the two raters. Definitions for numerator and denominator were as follows:
Meat: Shelf space occupied by poultry and fish/Total
Total = Fresh meat only; excludes frozen, canned, smoked or otherwise processed meat.
Milk: Shelf space occupied by 2% fat and skim milk/Total
Total = 2% fat, skim and whole milk only (half-gallon cartons)
Bread: Shelf space occupied by 100% whole wheat bread/Total
Total = Sliced, packaged loaves; excludes specialty bakery section, desert pastry, french bread, baguettes.

TABLE 2—Reliability Coefficients for Display and Health Promotion Item Measurements[a]

Variables	Different raters, same occasion[a]	Different raters, different occasions[b]	Same rater, different occasions[c]	Same rater, same occasion[d]
	(n = 37)	(n = 15)	(n = 27)	(n = 19)
Health promotion items				
Produce display	.67	1.00	.92	—
Meat display	.36	.63	.91	—
Milk display	.30	.46	1.00	—
Display Variables				
% Poultry + fish	.78	.86	.44	.98
% Reduced-fat milk	.75	.68	.58	.91
% Whole-wheat bread	.73	.47	.65	.93

[a]Statistical methods:
Display Variables: Intra-class correlation coefficients
Health Promotion Items: Weighted kappas
[a]Site: Seattle, WA (n = 37 stores)
[b]Site: Solano County, CA (n = 15)
[c]Sites: San Francisco, CA (n = 15), Orange County, CA (n = 12)
[d]Sites: Santa Cruz, CA (n = 15), Fresno, CA (n = 4)

(2), Table 2, show the correlations between two raters over a one-week time interval. This should approximate contemporaneous inter-rater reliability measures since few changes in health education materials were likely to have been made in this brief period. The revised format raised inter-rater reliability for the meat and milk displays—to 0.63 to 0.46, respectively.

The intra-class correlation coefficients presented in column (4), Table 2, all above 0.9, indicate high reliability in the same rater measuring the same display twice. Thus, most of the variation between occasions by the same rater was due to changes made by stores. The relatively high coefficients for health promotion items in column (3) suggest that these changed infrequently for the time intervals represented here. Display items showed lower intra-class correlation coefficients over time, particularly the meat display.

Discussion

Although sales data would reflect consumption most accurately, these are often unavailable to researchers. In the alternative measures which we used, measurements were made unobtrusively, and all of the stores approached for the pilot test agreed to participate; the response rate for the main study was 88 percent. Inter-rater reliability in the pilot was relatively high, particularly for the display variables. The lower inter-rater reliability figures for the health promotion items may have been due to the one-week interval between rater visits although, as noted earlier, significant changes were unlikely to have been made in that brief period. Test-retest reliability results indicated that both health promotion and display measures were relatively stable over periods ranging from one week to two months. Validity of the approach is currently being assessed, first by comparing the grocery store measures to the socioeconomic characteristics of people in the surrounding area, and second by correlating the survey measures with responses to dietary surveys of randomly sampled individuals living near the stores.

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Poison Control Center Follow-up of Occupational Disease

EDDY A. BRESNITZ, MD, MS

Abstract: We followed up 73 of 372 calls to a Regional Poison Control Center (RPCC) that involved workplace disease/exposure(s); most other calls were not made by the workers. An average of 12 additional people per workplace were potentially exposed. Six of the 73 contacted a government agency for investigation of the hazard/illness. Twenty-five percent of callers were still exposed an average of seven months after the original call. The results indicate that poison control centers should develop a public health component to calls about possible workplace poisonings. (*Am J Public Health* 1990; 80:711–712.)

Introduction

An accurate estimate of the incidence of occupational disease in the United States is hampered by the lack of an active surveillance system.[1–4] Investigators have proposed using existing data systems including regional poison control centers[5,6] for passive surveillance of occupational disease. A call may represent a group of affected/exposed workers and serve as a sentinel health event that identifies hazardous worksites.[7]

We conducted a follow-up survey of individuals with occupational exposure/disease who contacted the Delaware Valley Regional Poison Control Center (DVRPCC), over a 12-month period.

Methods

Approximately 7,760 individuals over the age of 17 contacted the Delaware Center between July 1, 1986 and June 30, 1987. Information was recorded on the American Association of Poison Control Centers' precoded report form. Referral guidelines listing the telephone numbers of outside agencies to which occupationally related callers may be referred are available to staff. The guidelines indicate that calls or complaints about known Occupational Safety and Health Administration (OSHA) regulated substances should be referred to OSHA and complaints about substances not covered or enforced by OSHA should be referred to the National Institute for Occupational Safety and Health (NIOSH).

A workplace exposure was recorded on the intake form of 372 calls (4.8 percent). Trained personnel, unrelated to the poison control center, abstracted data from these intake forms and called the telephone number obtained during the initial encounter. These follow-up calls were made between July 1 and November 1, 1987, an average of seven months after the initial call.

The interviewer explained the purpose of the study to the affected worker and obtained informed consent. To proceed, the interview was then either administered immediately or within one week at a prearranged time. In three cases, the interviewee was the sentinel case's employer who answered the questions from his/her perspective. Interviewers used a pre-tested, pre-coded form containing predominantly closed-ended questions. The analysis included 95% confidence intervals for differences in proportions.[8]

The study protocol was reviewed and approved by the Committee for the Protection of Human Subjects of the Medical College of Pennsylvania and the Medical Advisory Panel of the DVRPCC.

Results

The interviewers successfully interviewed 73 of the 372 work-related calls. Table 1 compares the characteristics of the 73 workers interviewed and the 299 workers not interviewed from information on the Center's intake form. Of those not interviewed, 209 (69.9 percent) calls originating from medical personnel or hospitals were excluded, because we did not believe that our questions would be answered without the patient's consent. Forty-nine callers (16.4 percent) either left no telephone number or gave a disconnected or false number. Of the remainder, 16 (5.4 percent) had been terminated from their jobs, 15 (5 percent) refused to answer questions and one (.3% percent) was an informational call.

The interviewed workers (responders) had acute exposures to more than 60 different chemicals at 73 different worksites; 19 individuals were exposed to two or more substances. All but four of the affected workers were symptomatic at the time of the initial call.

The responders had worked an average of 5.5 years at the worksite where the exposure occurred at the time of the follow-up call. Forty-nine (67 percent) of the 73 responders indicated that other workers (mean of 12) had been exposed

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TABLE 1—Comparison of Interviewed and Non-Interviewed Workers Based on Information from Poison Control Center (PCC) Intake Form

Characteristics	Exposed Workers Interviewed (N = 73)	Exposed Workers Not Interviewed (N)	95% CI of Difference
Mean Age (years)	30.8	30.5 (229)	
Sex (%)			
Male	66	78 (277)	(−23.9, 0.1)
Female	34	22	
Site of Caller (%)			
Residence	63	13	(38, 62)
Workplace	34	15 (289)	(7, 31)
Health Care Facility	3	72	(−75, −63)
Hospitalized (%)	0	6 (186)	(−12, −4)
PCC called back within 24 hours (%)			
Yes	69	59	(−2, 22)
No	31	37 (288)	
Tried and Failed	0	3	
Referral to Other Non-Medical Agency (%)			
Yes	6	15 (246)	(−16, −2)
No	94	85	
Route of Exposure			
Inhalation	67	42	(13, 37)
Ocular	11	32 (289)	(−30, −12)
Dermatologic	21	20	(−9, 11)
Ingestion	1	6	(−9, 11)
Parenteral	0	0	

at their worksite. Eight (16 percent) of these 49 responders indicated that one or more co-workers had been out on sick leave for the same exposure.

Overall, 48 responders reported they were advised to call their physician; 75 percent of these complied with this advice. Poison center staff recorded referrals to outside agencies in only four of the 73 calls (5 percent). However, 10 (14 percent) responders stated they were advised to call either OSHA, NIOSH, or the local health department. Five of seven reported that they called OSHA. One receiving a recommendation to call the health department or NIOSH did so.

Twenty-five of the 47 responders who approached their employer reported the institution of one or more primary preventive measures. Seven responders indicated that they were either fired or harassed or transferred.

Table 2 describes other issues assessed in the study. Only 37 percent of the responders had heard of either municipal, state, or federal right-to-know legislation, most frequently in the workplace or in the media. A responder was more likely to have heard about right-to-know laws if he/she had been hired less than one year prior to their initial call (62.5 vs 30 percent, 95% CI of difference = 5, 60).

TABLE 2—Other Public Health Issues Assessed in Responding Workers at Follow-Up

Health Issues (N)	Yes % (N)	95% CI
Worker still exposed (73)	25 (18)	16, 36
Existence of workplace union (70)	23 (16)	14, 34
Health and safety person present in workplace at time of exposure (e.g., employer, manager) (69)	49 (35)	37, 61
Worker still having health problems (72)	4 (3)	1, 12
Suffered disability or work loss (72)	32 (23)	21, 41
Sick leave covered by Workers' Compensation (23)	22 (5)	8, 44
Currently seeing physician (71)	4 (3)	1, 12
Caller heard about right-to-know law (73)	37 (27)	26, 49
Caller read material safety data sheet (69)	17 (12)	9, 28

Discussion

The data reveal that poison center staff infrequently referred occupationally related calls to agencies concerned with occupational health despite existing guidelines and may not have routinely documented such information. Referred callers tended to follow poison center staff advice when given.

Blanc and Olson[5] determined that many more occupationally related cases were enumerated through a state-mandated physician-initiated active reporting system than through a passive system such as a poison control center. Our study supports this finding.

Although we studied a selected sample of workers, the survey most likely underestimates the potential magnitude of the problem. The majority of those not reached were workers evaluated in a health care facility or admitted to hospitals. A greater percentage of those not reached were hospitalized, indicating more severe medical problems and, perhaps, more serious workplace hazards in this group.

Poison control centers reported that 6.8 percent of approximately 312,000 calls from adults in 1987 were related to exposures in the workplace.[9] Although this data base appears to be a source of supplementary surveillance data for occupational illness, if our experience represents that of other centers, the staff may need to modify their protocols and improve their public health approach to callers with occupationally related illness/exposures. Centers could refer all workplace-related calls to a separate state or federal agency responsible for hazard evaluation and follow-up. Failure to investigate these calls may result in continued exposure and illness in all workers connected to an index case.

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Illegal Abortion in Mexico: Client Perceptions

Susan Pick de Weiss, PhD, and Henry P. David, PhD

Abstract: An exploratory study of the perceptions of 156 abortion clients in Mexico suggests that perceived quality of service was the main reason for choosing physicians while cost and anonymity were the major reasons for choosing nonphysicians. "Too young" was the most often cited reason for pregnancy termination, followed by economic situation and having too many children already. Cost was, on average, equivalent to three to four weeks minimum wage; physicians' charges were about three times higher than those of nonphysicians. (*Am J Public Health* 1990; 80:715–716.)

Introduction

In Latin America, abortion is legally restricted everywhere except in Cuba.[1] In Mexico, abortion is legally permitted only for reasons of health, rape, or incest.[2] Contraceptive services are widely available, especially in the cities.

The public health, economic, social, and psychological costs associated with the illegal termination of unwanted pregnancies have been repeatedly documented[3–6]. Complications of clandestine abortions presently constitute the fifth largest cause of maternal mortality and the third highest reason for admission to gynecological services in the metropolitan hospitals of the Mexican Social Security System; no reliable data are available on women admitted to other services.[7]

To obtain information on the perceptions of women seeking clandestine abortions in Mexico, an exploratory study was conducted in Mexico City, Acapulco, and Oaxaca. Another objective was to test the feasibility of conducting behavioral research in an area of public health that throughout much of the region is culturally taboo and severely restricted by law.

Methods

Twelve experienced female graduate and undergraduate psychology students, sensitive in conducting individual interviews, some of whom had had abortions, were trained by the senior author (SPdW) to seek out personal acquaintances who had terminated unwanted pregnancies illegally in the recent past. On completion of interviews the subjects were invited to suggest other possible study participants. Of 159 women known to have had at least one induced abortion in the recent past, 156 agreed to individual interviews. The questionnaire was composed of mostly open-ended items that had been field-tested. Interviewees were assured of anonymity and confidentiality of replies.

Results

Of the 156 women, 119 had their abortion performed by physicians, 32 by untrained service providers, and five could not recall their providers' qualifications. As shown in Table 1, of the women attended by a physician most were not married. Ages ranged from 12 to 56 years; clients attended by physicians were significantly older and better educated than those seen by others. More professional women were seen by physicians. Over one-third of the women had at least one child.

A lower proportion of clients attended by physicians had experienced two or more abortions compared to those seen by nonmedically trained persons. Most of the procedures performed by physicians occurred in a private clinic or hospital (91.6 percent), while other service providers generally operated in the client's home. Quality of service was the most frequent reason for going to physicians (30.8 percent), followed by security, while cost (56.2 percent) was the main reason for seeking other providers, followed by security and anonymity. As also shown in Table 1, when asked in an open-ended question without probe, the most often cited reason for having an abortion among all women was becoming pregnant at too young an age. Another major reason among physician-attended women was the partner's rejection of the pregnancy. Among women attended by untrained providers, the second and third reasons were economic and having too many children already.

No differences between the two groups were noted among cited referral sources which included friends, sexual partners, family members, physicians, and psychologists. About half the physician-attended women considered the location of the service far away from their home compared to 25 percent of those attended by untrained providers. The cost of a physician-induced procedure was, on average, the equivalent of three to four weeks legal minimum wage in Mexico. A physician-provided service was generally two to three times more expensive than that provided by others ($88 vs $33 in 1987/88). Among women attended by physicians 43 percent reported receiving contraceptive counseling in connection with their abortion, compared to only 3 percent of those who went to other providers.

As further shown in Table 1, for the women attended by physicians, the most often reported procedures were curettage; over one-fifth did not know what procedure had been

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TABLE 1—Characteristics of Two Groups of Woman Who Had Induced Abortions, Mexico

Characteristics	Physician Attended (n = 119)	Other Attended (n = 32)	Difference	(95% CI)
Mean Age	26.2	24.5	1.7	(−.85, 4.25)
Mean No. of Children	1.97	2.06	−0.09	(−.9, .72)
Average Cost in Dollars	88	33	55	(45.2, 64.8)
	Percentages			
Single	67	59	8	(−9,25)
Attended College	39	25	14	(−9,37)
Professional Occupation	19	4	15	(8,22)
Two or More Abortions	13	22	−9	(−36, 18)
Received Contraceptive Information	43	3	40	(15, 65)
Post-abortion Problems	22	47	−25	(−53, 3)
	Procedure Used			
Curettage	50	13	37	(15, 59)
Catheters	3	19	−16	(−46, 14)
Injections	3	25	−22	(−51, 7)
Suction	20	–	–	(–)
Wire, Needles, Herbs	–	29	–	(–)
Unknown to Woman	24	13	11	(−17, 39)
	Reason for Abortion			
Too Young	31	34	−3	(−27, 21)
Partner Pressure	39	13	26	(1, 51)
Too Many Children	8	16	−8	(−38, 22)
Economic	15	19	−4	(−32, 24)
Other Reasons	7	18	−11	(−41, 19)

used. Among the women attended by untrained service providers, the most often cited procedures were injections, catheters, herbs, curettage, and wires or needles; few did not know what procedure was used.

Before going to a service provider, almost two-thirds of all the women had purchased pseudoabortifacients without adequate knowledge of possible risks. About one-third of all the women had tried inducing an abortion themselves, resorting to herbs, injections, or a combination of both, or of either with pills, hard exercise, or the introduction of objects into the cervix. Post-abortion complications were noted by less than one-fifth of the physician-attended women (mostly cramps) compared to nearly half of those attended by untrained providers who noted more hemorrhages (22 percent vs 47 percent). When asked if they would recommend their provider to other women, three-fourth of the physician-attended women replied affirmatively compared to one-fourth of those attended by other service providers.

Discussion

It is recognized that the findings from this exploratory study are limited by the nonrepresentativeness of the sample. However, the responses obtained confirm informal impressions that nonmedically trained persons provide abortion services primarily to women of lower socioeconomic and educational levels, and that such women are at greater risk for experiencing post-abortion complications and for future unwanted pregnancies since contraceptive counseling is seldom provided.

The method of open-ended interviewing developed for this exploratory study appears suitable for further adaptation for behavioral research on the sensitive topic of abortion with more representative populations in those countries of the region where the procedure remains a taboo topic. In concert with more epidemiological research, such studies may be expected to contribute to a reexamination of the public health risks of legally restricted abortion and to motivate improved access to effective contraception that will reduce the future incidence of unwanted pregnancies.

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A Survey of State Medicaid Policies for Coverage of Abortion and Prenatal Diagnostic Procedures

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Abstract: In the summer of 1988, we surveyed all states to evaluate access to Medicaid funding for abortion after the diagnosis of an anomalous fetus. All state Medicaid programs covered amniocentesis, and most had expanded coverage to include newer prenatal diagnostic procedures for eligible women. In 29 states, however, abortion coverage was limited to instances when continuation of abortion threatens the life of the mother. Only 13 states paid for a woman on Medicaid to obtain an abortion after diagnosis of an anomalous fetus. (*Am J Public Health* 1990; 80:717–720.)

Introduction

Medicaid is a joint state- and federally-funded program, enacted in 1965, to provide health care coverage for many indigent and disabled citizens. Within broad federal guidelines, each state has latitude in deciding which medical and ancillary services are to be covered by its Medicaid program.

In 1973, the US Supreme Court decision in *Roe v. Wade* prohibited states from interfering with a woman's right to abortion except to protect maternal health or a viable (third trimester) fetus. By 1974, three-fourths of the states covered most abortions under their Medicaid programs.[1]

In 1977, Congress passed the Hyde Amendment, which limited federal reimbursement of abortions to situations that threatened the life or physical health of the mother, and to victims of rape and/or incest. Current federal policy reimburses states only for abortions performed to save the life of the mother. Abortions for other reasons, which constitute over 99 percent of all abortions, must be paid for by non-federal or, in most cases, state funding.[2]

At present, increasingly sophisticated techniques such as amniocentesis, chorionic villus sampling (CVS), ultrasound, and biochemical assays are permitting earlier and broader detection of fetal abnormalities. Malpractice fears impel physicians to identify patients at risk for producing children with prenatally-diagnosable defects and offer them appropriate testing.[3] At least one state requires that maternal serum alpha-fetoprotein (MSAFP) screening be offered to pregnant women.[4] Ethical practice dictates that when tests show a fetal anomaly exists, the woman be offered the option of pregnancy termination, within the guidelines of each state's law regulating abortion.[5] Information has been unavailable nationally on coverage of prenatal diagnostic services by Medicaid programs and on utilization of these procedures by pregnant Medicaid recipients.

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The survey reported here delineates state-by-state Medicaid guidelines for coverage of prenatal diagnostic procedures and abortion.

Methods

We developed a questionnaire that asked about Medicaid coverage of seven prenatal services: amniocentesis, ultrasound to rule out fetal anomalies, CVS, MSAFP testing, fetal DNA analysis, chromosome analysis on cultured amniotic fluid cells, and genetic counseling. We included the standard Current Procedural Terminology Codes for each procedure (when available). Respondents were asked to check either "covered", "not covered", "covered on a case-by-case-basis", or "unknown" for each procedure. We asked about Medicaid coverage of abortion for seven indications, broad enough to encompass the variation in state policies, using the legal terminology of indications for abortion as reported in the Medicare and Medicaid guide.[6]

We mailed the questionnaire to the director of the Medicaid program in each state and the District of Columbia. After follow-up, including telephone calls, the final response rate was 100 percent. We did not attempt to verify how completely these guidelines were followed in each state. The responses reflect Medicaid regulations as of summer 1988.

Results

Each state's coverage of prenatal diagnostic procedures is shown in Table 1. The majority of states cover all procedures listed, and almost all have developed guidelines for coverage, as indicated by the low numbers of "case-by-case" or "unknown" responses. All states and the District of Columbia cover amniocentesis, and all but one cover MSAFP testing. Two-thirds cover CVS and three-fourths cover DNA analysis, both of which are newer technologies for the detection of fetal abnormalities.

Table 2 lists each state's coverage for abortion. At the time of the survey, seven states and the District of Columbia paid for abortion on demand. All states cover abortions that save the life of the mother, the only ones for which federal reimbursement is available. In 29 states, this is the *only* indication for which Medicaid will cover an abortion. Eleven states will pay for abortion when the indication is a known or suspected fetal anomaly, and two other states evaluate coverage on a case-by-case basis. Just over one-fourth of the states will pay for "medically necessary" abortions, usually as certified by an attending physician. Likewise, victims of rape and/or incest are covered for abortion in one-fourth of the states. This indication has been hotly debated on a federal level, as President George Bush recently vetoed a congressional package that would have reinstated federal reimbursement for abortion in cases of rape and incest.

Discussion

A combination of Medicaid regulations has led to the incongruous policy in many states of covering prenatal diagnostic services but not the selective abortion in cases of a diagnosed fetal abnormality. We believe that these policies

TABLE 1—Medicaid Coverage of Prenatal Diagnostic Procedures by States and District of Columbia as of September 1988

States	Amnio-centesis	Fetal Ultrasound	CVS	MSAFP	DNA Analysis	Karyo-typing	Genetic Counseling
Alabama	X	X	X	X	X	X	X
Alaska	X	X	X	X	X	X	X
Arizona	X	X	X	X	C	X	X
Arkansas	X	X	C	X	X	X	X
California	X	X	0	X	0	X	X
Colorado	X	X	0	X	?	X	X
Connecticut	X	X	?	X	X	0	?
Delaware	X	X	X	X	X	X	X
District of Columbia	X	X	C	X	0	X	X
Florida	X	0	X	X	X	X	?
Georgia	X	X	X	X	X	X	?
Hawaii	X	0	C	X	..	X	X
Idaho	X	X	X	X	X	X	0
Illinois	X	X	X	X	X	X	X
Indiana	X	X	X	X	X	X	X
Iowa	X	0	X	X	X	C	C
Kansas	X	0	0	X	X	0	0
Kentucky	X	X	X	X	X	X	X
Louisiana	X	X	0	X	X	X	X
Maine	X	?	X	X	X	X	X
Maryland	X	X	X	X	C	X	X
Massachusetts	X	X	C	X	X	X	X
Michigan	X	X	X	X	X	X	X
Minnesota	X	X	X	X	?	X	X
Missouri	X	0	0	X	0	X	X
Mississippi	X	X	C	X	C	X	X
Montana	X	X	X	X	X	X	X
Nebraska	X	X	X	X	X	X	X
Nevada	X	X	X	X	X	X	X
New Hampshire	X	X	X	X	X	X	X
New Jersey	X	X	C	X	C	X	X
New Mexico	X	X	X	X	X	C	X
New York	X	X	X	X	0	X	X
North Carolina	X	X	0	X	X	0	0
North Dakota	X	X	X	X	C	X	X
Ohio	X	X	X	X	X	X	X
Oklahoma	X	C	0	0	0	0	0
Oregon	X	X	X	X	X	X	0
Pennsylvania	X	0	0	X	X	X	X
Rhode Island	X	X	C	X	C	C	X
South Carolina	X	X	X	X	X	X	X
South Dakota	X	X	X	X	X	X	X
Tennessee	X	X	X	X	X	X	X
Texas	X	X	0	X	X	X	X
Utah	X	X	X	X	X	X	X
Vermont	X	X	X	X	X	X	X
Virginia	X	X	X	X	X	X	X
Washington	X	X	X	X	X	X	0
West Virginia	X	X	X	X	X	X	X
Wisconsin	X	X	X	X	X	X	X
Wyoming	X	X	X	X	X	X	X
Totals							
Covered	51	43	34	50	38	44	42
Not Covered	0	6	9	1	5	4	6
Case-by-case	0	1	7	0	6	3	1
Unknown	0	1	1	0	2	0	3

Symbols
X = covered
0 = not covered
C = case-by-case
? = unknown

discriminate against poor women and interfere with ethical medical practice. By covering prenatal diagnostic services in Medicaid programs, each state in this country acknowledges the medical necessity of these services and its commitment to providing access to them. Since abortion is a recognized option for women carrying an anomalous fetus, states that refuse to pay for abortion interfere with women's rights. Once a state has agreed to pay for diagnostic procedures, we do not believe it should influence subsequent medical decisions.

In the future, Medicaid guidelines will affect more and more women, as Congress encourages states to expand eligibility criteria for pregnant women. By July 1990, federal Medicaid guidelines mandate that all states cover pregnant women at poverty level, and will give states the option of covering women with incomes up to 185 percent of poverty

TABLE 2—Medicaid Coverage of Abortion Services by States and the District of Columbia as of September 1986

State	On Demand	Life-threatening	Physical Health Damage	Mental Health Damage	Fetal Anomaly	Medical Necessity	Rape/ Incest
Alabama	0	X	0	0	0	0	0
Alaska	X	X	X	X	X	X	X
Arizona	0	X	0	0	0	0	0
Arkansas	0	X	0	0	0	0	0
California	X	X	X	X	X	X	X
Colorado*	0	X	C	C	C	C	0
Connecticut	0	X	C	C	C	X	C
Delaware	0	X	0	0	0	0	0
District of Columbia*	X	X	X	X	X	X	X
Florida	0	X	0	0	0	0	0
Georgia	0	X	0	0	0	0	0
Hawaii	X	X	X	X	X	X	X
Idaho	0	X	C	C	0	C	0
Illinois	0	X	0	0	0	0	0
Indiana	0	X	0	0	0	0	0
Iowa	0	X	0	0	X	0	X
Kansas	0	X	0	0	0	0	0
Kentucky	0	X	0	0	0	0	0
Louisiana	0	X	0	0	0	0	0
Maine	0	X	C	C	0	C	0
Maryland	0	X	X	X	X	0	X
Massachusetts	X	X	X	X	X	X	X
Michigan*	X	X	X	X	X	X	X
Minnesota	0	X	0	0	0	X	X
Missouri	0	X	0	0	0	0	0
Mississippi	0	X	0	0	0	0	0
Montana	0	X	0	0	0	0	0
Nebraska	0	X	0	0	0	0	0
Nevada	0	X	0	0	0	0	0
New Hampshire	0	X	0	0	0	0	0
New Jersey	X	X	X	X	X	X	X
New Mexico	0	X	0	0	0	0	0
New York	0	X	X	X	0	X	0
North Carolina	0	X	0	C	0	0	0
North Dakota	0	X	0	0	0	0	0
Ohio	0	X	0	0	0	0	0
Oklahoma	0	X	0	0	0	0	0
Oregon	0	X	X	X	0	X	0
Pennsylvania	0	X	0	0	0	0	X
Rhode Island	0	X	0	0	0	0	0
South Carolina	0	X	0	0	0	0	0
South Dakota	0	X	0	0	0	0	0
Tennessee	0	X	0	0	0	0	0
Texas	0	X	0	0	0	0	0
Utah	0	X	0	0	0	0	0
Vermont	0	X	0	0	0	0	0
Virginia	0	X	X	X	0	X	0
Washington	X	X	X	X	X	X	X
West Virginia	0	X	X	X	X	X	X
Wisconsin	0	X	X	0	0	0	X
Wyoming	0	X	0	0	0	0	0
Totals							
Covered	8	51	14	13	11	14	14
Not Covered	43	0	33	33	38	34	36
Case-by-case	0	0	4	5	2	3	1

Symbols
X = covered
0 = not covered
C = case-by-case
*Since the fall of 1986, Michigan and Colorado, by voter referenda, and the District of Columbia, by legislative action, altered their regulations to pay only for life-threatening abortions.

level.[7] Therefore, the public health implications of Medicaid policies on abortion will become even greater.

As budgetary pressures force state and federal agencies to cut costs, the economic burden of restrictive abortion funding will loom larger. The financial cost of the Hyde Amendment was estimated conservatively to be $95 million in 1986. For every dollar spent on a desired abortion through Medicaid, four dollars are saved in medical and social welfare costs in just two years.[8] The medical and emotional costs of bearing children that are either not wanted or have major anomalies may be far greater.

Health care costs of infants and children with genetic disorders and chronic illnesses are at least three times higher than those of other children.[9] Those born to low-income families pose an even greater challenge to an already overburdened household. Children with disabilities require addi-

tional health care services including occupational and physical therapy, psychosocial support, home health care, respite and day care.

On a state level, Medicaid policies on abortion have been heavily influenced by political pressure from right-to-life groups. Since our survey, two states (Colorado and Michigan) have altered their regulations because of referenda placed on the ballot in the November 1988 elections, and the District of Columbia has withdrawn funding entirely. Regardless of one's views on abortion, we believe the Medicaid program should not be used to force the poorest women among us to bear children with severe congenital anomalies.

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Obstetrical Pulmonary Embolism Mortality, United States, 1970–85

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Abstract: To investigate pulmonary embolism as a cause of obstetrical death, vital records data from 1970 through 1985 were analyzed. Results showed that the number of obstetrical pulmonary embolism deaths per 100,000 live births declined by 50 percent for both Whites and Blacks. However, Black women maintained more than a 2.5-fold higher risk, and women over age 40 had a ten-fold higher risk of embolism mortality. Thus, although the risk of obstetrical pulmonary embolism death has declined, some subgroups of women remain at higher risk. (*Am J Public Health* 1990; 80:720–721.)

Introduction

Several studies of maternal mortality have found pulmonary embolism to be the leading cause of maternal death in recent years.[1–5] It has been suggested that pulmonary embolism deaths may be becoming more important as a cause of maternal death because deaths due to other causes are declining more rapidly, or because medicolegal concerns favor the diagnosis of causes of death perceived to be nonpreventable.[1,4] Little is known nationally about trends in maternal mortality due to pulmonary embolism.

The purpose of the current analysis was to determine the absolute and relative importance of pulmonary embolism as a cause of obstetrical death over time on a national basis, and to describe the demographic risk factors for obstetrical pulmonary embolism (OPE) death.

Methods

The total number and characteristics of obstetrical deaths were obtained from multiple cause of death mortality tapes made available by the National Center for Health Statistics (NCHS). Up to 20 diagnoses are recorded from each death certificate and coded according to the International Classification of Diseases (ICD).[6,7] Obstetrical deaths were identified as those with any nonabortive pregnancy-related diagnosis (ectopic pregnancies, spontaneous abortions, induced abortions, and molar pregnancies were excluded). For the years 1970 through 1978 OPE deaths included ICDA-8 codes 634 and 673, while for 1979–85 they included ICD-9-CM code 673. These data do not allow one to separately identify amniotic fluid, thrombotic or air pulmonary embolism; therefore all causes of pulmonary embolism are grouped together.

The OPE mortality ratio is defined as the number of obstetrical deaths associated with pulmonary embolism per 100,000 live births during the same time period. The numbers of live births for each year were obtained from national natality computer tapes made available by NCHS. The OPE mortality ratios for both Blacks and Whites over time were standardized to the age distribution of White live births 1970–85.

The percent of obstetrical deaths due to embolism is the number of pulmonary embolism-associated obstetrical deaths per 100 obstetrical deaths during the same time period. The analysis is restricted to women over the age of 14 years and younger than 45 years who were classified as either White or Black.

Results

From 1970 through 1985 there were a total of 1,417 OPE deaths and 55,096,188 live births, for an overall OPE mortality ratio of 2.6 OPE deaths per 100,000 live births. Over the 16-year interval OPE mortality ratios, standardized by age, declined more than 50 percent for both Whites (from 3.3 to 1.3 per 100,000 live births) and for Blacks (from 9.0 to 4.2 per 100,000 live births) (Figure 1). However, Black women maintained more than a 2.5-fold higher risk of OPE mortality than White women throughout the 16-year interval. This racial disparity did not vary systematically over time or

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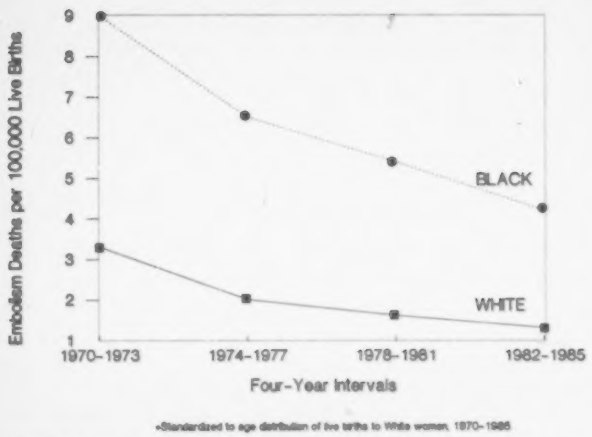

FIGURE 1—Obstetrical Pulmonary Embolism Deaths per 100,000 Live Births,* United States, 1970–85

between age groups. The percentage of all obstetrical deaths which were associated with pulmonary embolism did not differ between White and Black women, and remained relatively constant over time at approximately 25 percent.

Aggregating all 16 years under analysis, the absolute risk of OPE death increased with age for both race groups and was at least 2.5 times higher for Black women than for White women at all ages (Figure 2). For both racial groups, the risk for women 40 years of age or older was nearly 10 times greater than for women less than age 25.

Discussion

Although recent studies have found pulmonary embolism to be the leading cause of maternal death, vital records data show that the absolute risk of OPE death has been dropping and the proportion of obstetrical deaths associated with pulmonary embolism has not been increasing. Thus the belief that maternal pulmonary embolism deaths are a growing problem in the US is not supported by vital records data.

The limitations of vital records data in the surveillance of maternal death have been previously described.[8] Investigations designed specifically to identify and evaluate maternal deaths uncover more deaths, and often have more available information for assigning a cause of death. Indeed, vital records data do not allow clinical differentiation of immediate cause of death from underlying conditions which may contribute to death. We reviewed concurrent diagnoses from the death certificates of the OPE deaths under analysis. The most common concurrent diagnoses were cardiovascular disease, arrhythmia, shock or cardiac arrest (40 percent), hemorrhage or coagulopathy (17 percent), venous disease (9 percent), or "other ill-defined causes of morbidity or mortality" (9 percent). As these conditions could either precede or follow an OPE, inferences about which condition was the principal cause of death versus which conditions may have contributed to causing death cannot be made based on these data. However, we found no change over time in the distribution of concurrent diagnoses. In addition, we repeated our analysis using the single underlying cause of death as determined by NCHS. Results revealed trends over time and by race and age which were identical to those reported in this paper.

In this study we were unable to evaluate type of pulmonary embolism, although previous studies of maternal death have found half or more of maternal pulmonary embolism deaths to be thrombotic in nature.[1,3–5,9] Among causes of embolism, thrombotic events may be the most amenable to prevention through changes in medical/surgical practices.[10,11] Further research is necessary to characterize the temporal relationship between delivery and thrombotic events, as well as to identify potential risk factors for thrombotic death, such as method of delivery (vaginal vs cesarean section), personal characteristics (e.g., obesity), or underlying medical, surgical, or obstetrical conditions. With this information, interventions such as prophylactic use of pressure gradient stockings or anticoagulation[3] may be implemented and evaluated for those at highest risk. Unfortunately, vital records data alone cannot provide the increased clinical detail necessary to guide risk reduction strategies for this leading cause of obstetrical death in the United States; therefore much depends on current efforts to gather supplemental information about maternal deaths.[12]

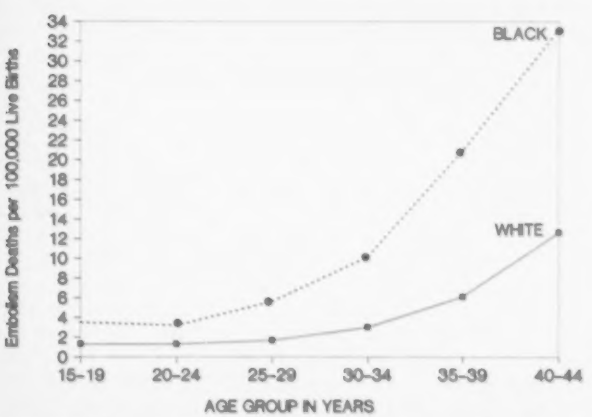

FIGURE 2—Obstetrical Pulmonary Embolism Deaths by Age and Race, United States, 1970–85

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Cervix and Breast Cancer Incidence in Immigrant Caribbean Women

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Abstract: Cervix and breast cancer incidence in 1978–82 was computed for immigrant and United States-born Black women in Brooklyn, New York. Compared to the national SEER (Surveillance, Epidemiology and End Results) rates, US-born and Haitian women had high rates of invasive cervical cancer, while English-speaking Caribbean immigrants had an average rate. However, while US-born women had an average rate of carcinoma in situ of the cervix, both immigrant groups had low rates. Both immigrant groups had low rates of breast cancer, whereas US-born Black women had an average rate. (*Am J Public Health* 1990; 80:722–724.)

Introduction

Immigrant populations challenge the United States systems for preventive health care because they may differ from the US-born in their patterns of disease and in their knowledge and utilization of preventive health services. Where health statistics are recorded only by race, differences within populations may be obscured and information inadequate to plan effective programs.

In Brooklyn, New York City, about 20 percent of the Black community of about 700,000 are immigrants, primarily from the Caribbean basin. Haitian immigrants with invasive cancer of the cervix are diagnosed at a significantly later stage than US-born Black women,[1] and Haitians are less likely than US-born or English-speaking Caribbean women to have had a Pap test or to practice breast self-examination.[2] We determined the incidence of breast and cervix cancer among ethnic subgroups of the Black community in Brooklyn in order to help plan effective cancer prevention activities.

Methods

Population data from the 1980 US census were obtained from a 5 percent Public Usage Microdata Sample for Brooklyn. Birthplace was categorized as: 1) USA; 2) English-speaking Caribbean islands; 3) Haiti; and 4) Other. "Other" birthplaces included Africa, Europe, Canada, central and south America, and Spanish-speaking Caribbean islands. Birthplace was available for 92 percent of Black foreign-born women. For each five-year age group, foreign-born women of unspecified birthplace were allocated to above categories 2–4 in proportion to women of known birthplace. Cancer cases in Brooklyn residents from 1978–82 were obtained from the New York State Cancer Registry.[3] Cases were histologically confirmed in 93–99 percent depending on diagnosis.[3] Birthplace was not available for 8–14 percent of cases and was allocated as described for population data.

Although carcinoma in situ may be diagnosed in ambulatory facilities and therefore has potential for underregistration, low-income populations are usually dependent on hospital-based facilities and there is no reason to assume differential registration for immigrant and non-immigrant women.

For each group of Brooklyn women, we computed the age-standardized incidence ratio (SIR—observed:expected cases) comparing each group to the total Black US rates. We used the "indirect method" by applying the five-year age-specific rates for Black women from the 1978–81 data of the Surveillance, Epidemiology and End Results (SEER) program to the Brooklyn populations to obtain the expected numbers of cases.[4] The 95% confidence intervals (CI) of the SIR were computed from a Poisson distribution.[5] The indirect method was used because Haitian and "Other" groups had less than 50 cancer cases per site giving unstable age-specific rates.

The 1980 census undercounted low-income minority populations especially in areas with many immigrants.[6] Incidence ratios based on census data are therefore maximum ratios.[7] Since undocumented immigrants were probably recent arrivals, we assumed one uncounted immigrant of the same age, sex and birthplace for every two counted immigrants who entered the USA after 1969. This increased the English-speaking Caribbean adult female population by 23 percent, the Haitian population by 30 percent, and the "Other" population by 18 percent. We used this adjusted population to estimate *minimum* cancer incidence ratios, an arbitrary though "reasonable" maneuver that was necessary to assess the importance of unusually high ratios that might have programmatic implications.

Results

Incidence ratios for invasive cervical cancer were high for Haitian-born women, yet for carcinoma in situ, ratios were low for all three immigrant groups (Table 1). US-born women had a high incidence ratio for invasive cervical cancer but an average ratio for carcinoma in situ. For breast cancer all immigrant groups had low incidence ratios, whereas the ratio in US-born women indicated an incidence similar to the SEER rate. The SIRs cannot be compared to one another because they were not standardized to the same age distribution. When the population was adjusted for uncounted immigrants, all the SIRs were reduced, but relationships were essentially unchanged.

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TABLE 1—Standardized Incidence Ratios for Cancer of the Cervix and Breast by Birthplace among Black Women in Brooklyn, 1978–82

Cancer Site		Birthplace			
		USA	ESC*	Haiti	Other
A. Census Population					
Cervix (invasive)	N	296	78	36	26
	SIR	1.28	1.06	2.06	0.80
	95% CI	1.14, 1.43	0.84, 1.32	1.46, 2.85	0.52, 1.18
Cervix (in situ)	N	700	141	33	38
	SIR	0.96	0.64	0.48	0.38
	95% CI	0.89, 1.03	0.54, 0.75	0.33, 0.68	0.27, 0.52
Breast	N	754	206	45	40
	SIR	0.97	0.82	0.81	0.39
	95% CI	0.90, 1.04	0.71, 0.94	0.59, 1.08	0.28, 0.52
B. Adjusted Population					
Cervix (invasive)	SIR		0.90	1.62	0.72
	95% CI		0.72, 1.11	1.12, 2.25	0.47, 1.05
Cervix (in situ)	SIR		0.50	0.37	0.33
	95% CI		0.42, 0.60	0.26, 0.51	0.23, 0.43
Breast	SIR		0.71	0.64	0.33
	95% CI		0.62, 0.82	0.47, 0.86	0.23, 0.45

N = cases in five years (1978–82)
*English-speaking Caribbean

Selected characteristics of the four groups (excluding "other") are shown in Table 2. Immigrants had higher rates of high school completion and labor force participation and lower rates of poverty and fertility than US-born women in Brooklyn and Black women nationally.

Discussion

The incidence ratios reflect both the underlying prevalence of cervical neoplasia and the level of Pap smear screening. Factors consistently associated with a high prevalence of cervical neoplasia include low socioeconomic status (SES) and early sexual activity. These factors may account for the high incidence of invasive cervical cancer in US-born Black women in Brooklyn, also noted in Detroit, the major low-income, northern city in SEER.[8]

In the immigrant groups, the average to high rates of invasive cervical cancer combined with low rates of carcinoma in situ suggest low screening rates among all Black immigrants. Community and hospital studies of selected populations have consistently reported lower screening rates in immigrants than in US-born women.[1,2] A relatively low prevalence of cervical neoplasia in the Brooklyn immigrants may also be a possibility given their relatively high socioeconomic status and low early fertility (a proxy for sexual activity) reported in the census.

Cervical neoplasia among Haitian immigrants in Brooklyn has several unique features including: an unusually high incidence of invasive cancer (even when adjusted for a census undercount) with a high proportion in advanced stages,[1] and consistently lower screening rates than other Black women in community[2] and hospital populations.[1] Furthermore, two unpublished studies* suggest that Haitians have lower hysterectomy rates than other Black women in Brooklyn. First, in a community-site screening program, only 3 percent of 561 Haitian women had a hysterectomy compared to 17 percent of 378 English-speaking Caribbean immigrants and 14 percent of 319 US-born women. Second, among hospital inpatients with nongynecologic diagnoses undergoing routine cytology, none of 28 women with French names had a hysterectomy compared to 16 percent of 257 other Black women. In each study these differences held for all age groups, and the hysterectomy rates in the US-born and English-speaking Caribbean women were similar to rates in women in upstate New York.[9] Although conclusive evidence is lacking, we suggest that the uniquely high rate of invasive cervical cancer in Haitians may reflect a greater proportion of women with uteri at risk, low screening rates among Haitian immigrants in Brooklyn, and a severe lack of screening in deeply impoverished Haiti, where clinical and laboratory facilities are limited. A high rate of cervical neoplasia in Haitians has also been reported from Miami.[10]

TABLE 2—Selected Characteristics of Black Women in Brooklyn by Birthplace

Characteristics	Brooklyn Women by Birthplace			National data
	USA	ESC	Haiti	
Fertility in women ages 15–24—Children per 1000 women	502	313	295	540
% Women over age 25 with education ≥ 12 grade	58.4	84.8	67.2	51.5
% Women ages 16 and over in labor force	45.8	67.0	63.2	53.3
% Persons with income below poverty	39.2	18.3	26.0	29.9

National data for Black women: US Census 1980.[12] Brooklyn data from Public Usage Microdata Sample for Brooklyn.

The finding of an average incidence of breast cancer among US-born Black women in Brooklyn underlines the value of examining ethnic differences, since the overall rate in Black women would otherwise appear encouragingly low. The below-average incidence of breast cancer in both Haitian and English-speaking Caribbean immigrants is consistent with other studies which show persistently low rates of breast cancer in migrants from countries with low incidence.[11] However, limited exposure to clinical breast examination and mammography among immigrants might also contribute to low rates of detection of occult breast lesions. The demographic characteristics indicate that other risk factors associated with high rates of breast cancer—high SES and delayed pregnancy—cannot explain the low rates in immigrants.

These findings indicate the importance of evaluating cancer rates in different ethnic groups in heterogeneous communities, and have particular implications for cancer prevention activities in cities with Caribbean immigrant populations. The low rates of carcinoma in situ suggest that more basic education and cytologic screening are needed. The uniquely high rate of invasive cervical cancer in Haitians, combined with linguistic and other barriers to care, warrant special programs for Haitian women. Finally, the most intensive efforts to promote breast examination and mammography should probably be focused on US-born women.

ACKNOWLEDGMENTS

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*Fruchter RG: Unpublished data.

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Ten-Year Changes in Birthweight Distributions of Black and White Infants, South Carolina

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Abstract: The birthweight distributions of Black and White infants in South Carolina were compared for the paired-year periods 1975–76 and 1985–86. No discernible changes in birthweight distributions between the two time periods were observed especially among Black infants. The distributions among White infants reflected an overall improvement in birthweight most noticeably above 2500g. (*Am J Public Health* 1990; 80:724–726.)

Introduction

Birthweight is considered the most important predictor of infant mortality particularly for deaths occurring within the first 28 days of life.[1,2] While significant declines have occurred in infant mortality rates in the United States only slight declines in the incidence of low birthweight (<2500 grams) have been noted.[3,4] When noted, the declines were not observed for all segments of the population. Considerable racial differences persist; Black infants are twice as likely to be low weight at birth as White infants.[4]

South Carolina continues to have one of the highest infant mortality rates in the nation. Nevertheless, infant mortality rates and particularly neonatal mortality rates have declined substantially in the last decade. To determine whether improvement in birth weight has occurred in South Carolina, 1975–76 birthweight distributions of Black and White infants were compared with those of 1985–86.

Methods

Reported birthweights of infants born to mothers residing in South Carolina at the time of birth were obtained from public tapes of matched births and deaths provided by the Office of Vital Records and Public Health Statistics, South Carolina Department of Health and Environmental Control. Sex-specific birthweight distributions for Black infants and White infants were compared for the paired-year periods 1975–76 and 1985–86. Five-ounce weight classes were chosen to reflect the original measurements (ounces) and to adjust for propensity to report in quarter-pound units for infants weighing near average. Cumulative frequency distribution curves for each paired-year period and for each race-sex group were generated. Nonparametric procedures were used for statistical comparison.[5]

Results

There were no discernible changes in the birthweight distributions especially among Black infants and no statistically significant differences in birthweight distributions between the two time periods for any race-sex comparison (Figures 1 and 2). The shift toward larger infants was more obvious for White infants than for Black infants when comparing categorical values (Table 1). There was no change

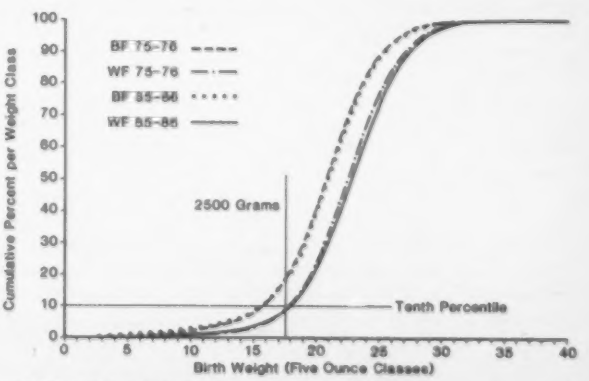

FIGURE 1—Cumulative Birthweight for Black and White Female Infants, South Carolina, 1975–76 vs 1985–86

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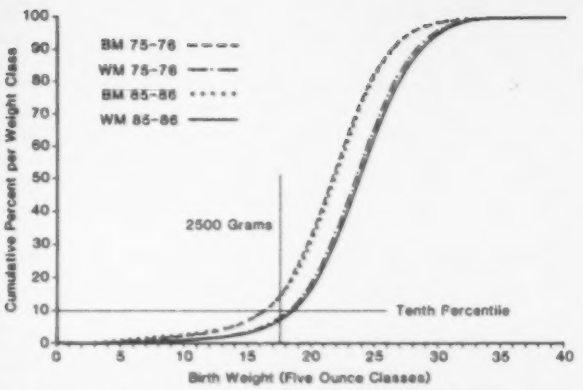

FIGURE 2—Cumulative Birthweight for Black and White Male Infants, South Carolina, 1975–76 vs 1985–86

in the proportion of Black or White infants weighing less than 2500g. Among White female and male infants, increases in birthweight were observed at the 10th, 50th, and 90th percentiles. Increases were observed among Black female infants only at the 90th percentile and among Black male infants only at the 50th percentile.

Changes were observed for certain maternal characteristics related to birthweight and/or infant mortality from 1975 to 1986 (Table 2). The proportion of teenagers giving birth declined from 32 percent to 23 percent among Blacks and from 19 percent to 12 percent among Whites accompanied by an increase in the proportion of women 30 years of age or older giving birth. A 16 percent difference (from 46 percent to 30 percent) in the proportion of mothers with less than a high school education was observed among Blacks with a 10 percent difference observed among Whites (from 31 percent to 21 percent). The proportion of unmarried mothers increased from 43 percent to 53 percent among Black women. There was little change in the proportion of expecting mothers seeking early prenatal care over the 10-year period.

TABLE 1—Summary Statistics, Birthweight Distributions of Black and White Infants for 1975–76 and 1985–86 Paired Years

Sex/Year	Number	Mean(g)	Standard Deviation(g)	LBW %	Percentiles(g) 10%	50%	90%
Females							
BF '75–76	18230	3029	600	14.4	2353	3090	3685
BF '85–86	19512	3036	618	14.2	2353	3090	3714
Difference		+7	+18	−0.2	0	0	+29
WR '75–76	27414	3300	564	6.7	2665	3317	3969
WF '85–86	30599	3335	579	6.7	2676	3374	3997
Difference		+35	+15	0.0	+11	+57	+28
Males							
BM '75–76	18684	3151	624	11.7	2438	3203	3855
BM '85–86	20008	3157	648	11.5	2438	3232	3856
Difference		+6	+24	−0.2	0	+29	+1
WM '75–76	29255	3424	602	6.0	2722	3458	4111
WM '85–86	32590	3465	605	5.4	2778	3487	4167
Difference		+41	+3	−0.6	+56	+29	+56

Discussion

It is clear that there has been no real improvement in the proportion of infants weighing less than 2500g between 1975 and 1986. Improvements in birthweight in this period are attributable to the segment of population producing less-than-average weight infants, but not so small as to qualify as low birthweight, i.e., <2500 g. The observations presented here are consistent with other reports showing only overall marginal and selective improvement in birthweights.[3]

Neonatal mortality rates in South Carolina declined by 32 percent among Blacks (from 17.2/1,000 live births to 11.7/1,000 live births) and by 39 percent among Whites (11.4/1,000 live births to 6.9/1,000 live births) from 1975 to

TABLE 2—Change in Selected Maternal Characteristics, 1975–76 to 1985–86

	Black			White		
	1975–76 (N = 37,189) %	1985–86 (N = 39,672) %	% difference	1975–76 (N = 57,548) %	1985–86 (N = 64,017) %	% difference
Age (years)						
<18	16	10	−6	7	4	−3
18–19	16	13	−3	12	8	−4
20–29	56	58	+2	67	64	−3
≥30	12	17	+5	14	23	+9
Education (years)						
<12	46	30	−16	31	21	−10
12	43	48	+5	42	39	−3
13–15	6	15	+9	15	20	+5
≥16	5	7	+2	12	19	+7
Marital Status						
Married	57	47	−10	95	92	−3
Unmarried	43	53	+10	5	8	+3
Prenatal Care Began (months)						
0	2	3	+1	1	1	0
1–3	52	53	+1	76	77	+1
4–6	37	34	−3	19	18	−1
7–9	8	10	+2	4	4	0

Totals may differ from 100% (or zero) because of rounding.

1986. (Office of Vital Records and Public Health Statistics, South Carolina Department of Health and Environmental Control, Columbia, SC.) Clearly the changes in neonatal mortality rates are not explained by changes in birthweight distributions. Low birthweight is an underlying contributor to neonatal mortality intricately related to a host of other biological, medical, sociodemographic, and behavioral factors. While many of these factors are vague, non-specific and non-operational, they are, in turn, related to specific, intervenable factors of prenatal care, nutritional intake, and smoking and alcohol consumption during pregnancy.[6] The explanation for the apparent paradox between significant declines in neonatal mortality rates and very marginal improvement in birthweight is not likely to be found in an examination of medical records and routinely collected vital statistics.

Finally, the absence of improvement in the proportion of low birthweight (LBW) infants over the time period 1975–86 should raise questions about either a flawed strategy of national health policy and/or inadequate application or utilization of support programs such as WIC (Women, Infants and Children supplemental feeding program), AFDC (Aid for Families with Dependent Children), and food stamps. Focusing on infant mortality as both a guide to operations and as a criterion for success in maternal and infant health programs is a suspect strategy. The effect of reducing the proportion of infants in the low birthweight tail of the distribution would certainly result in improvements in infant mortality rates, but not vice-versa. If the evidence of improvement among those whose infants weighing above the 2500g critical value is correctly interpreted, then there is more than a hint that the national strategy as it is applied in South Carolina should look beyond infant mortality. One approach would be to renew attention to conditions of life which are clearly known to influence maternal/familial well-being: hygiene, sanitation, crowding, nutrition, injury hazards, toxic substances, and unsafe conventions of behavior in the home and neighborhood.

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Unlicensed Midwifery Practice in Washington State

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Abstract: We examined the role of unlicensed midwives in Washington State by questioning mothers of infants born out-of-hospital with an unlicensed person in attendance. Only a small proportion of the state's births (0.11 percent) were attended by unlicensed midwives. Unlicensed midwives attended 7 percent of home births, licensed midwives and certified nurse-midwives attended 69 percent. Mothers chose unlicensed midwives because they had religious beliefs in common, or because they were the only providers available who would attend a home birth. (*Am J Public Health* 1990; 80:726–728.)

Introduction

In 1981, the Washington State legislature passed a Midwifery Act which established standards for direct-entry (i.e., without prior nursing) midwifery education and practice. To become a licensed (direct-entry) midwife, the law specifies that applicants be graduates of a state accredited educational program wherein they receive at least three years of coursework and clinical experience. Only a handful of other states license direct-entry midwives. Washington's law is considered unique in the United States because its standards are consistent with those established by the International Confederation of Gynecology and Obstetrics and the International Confederation of Midwives.[1]

The Washington State Midwifery Act permits unlicensed midwives to practice as long as no fees are charged. In a recent review of the Act, the legislature added an amendment to allow unlicensed midwives with documented experience to challenge the licensing examination without completing the required coursework. The amended Act also stipulated that data be provided to determine the role of unlicensed practitioners in the provision of maternity services in the State of Washington. Accordingly, the present study was undertaken to determine the proportion of births that were attended by unlicensed midwives and other unlicensed attendants, and the reasons for mothers' choice of an unlicensed midwife.

Methods

Births which took place out-of-hospital (in a birth center, medical facility, home, or "other" setting) were selected from 1986 birth certificates (N = 1656). Those with an unlicensed person in attendance (mother, father, unlicensed midwife, other or unknown attendant) were identified (N = 423). Records

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TABLE 1—Birth Attendant Reported by Respondents Compared with Birth Attendant Recorded on Birth Certificate

Birth Attendant Recorded on Birth Certificate	Birth Attendant Reported by Respondents					
	Physician	Licensed Midwife/ Certified Nurse-Midwife	Father Mother	Unlicensed Midwife	Other	Total
Father/Mother	0	7	26	17	0	50
Unlicensed Midwife	0	9	0	12	1	22
Other/Unknown	47	11	15	38	23	134
Total	47	27	41	67	24	206

were checked by matching attendant's name on the birth certificate against state rosters of licensed providers.

Eight births were excluded because of a missing address or because the birth mother was not the infant's legal guardian. A questionnaire was mailed to the remaining 415 mothers, asking for verification of type of birth attendant and place of birth, whether they had planned an out-of-hospital birth, and the reasons for their choice of birth attendant. Follow-up telephone calls were made to non-respondents for whom telephone numbers could be found. A second questionnaire was sent to those who could not be reached by telephone.

Results

The response rate was 49.6 percent. Compared to respondents, non-respondents were more likely to be teenagers (11 vs 3 percent), other than White race (12 vs 5 percent), and unmarried (26 vs 7 percent). No differences were found in the proportion of mothers living in rural counties or having professional occupations.

The attendant category on the birth certificate agreed with the mother's response in only 30 percent of the cases (Table 1). Sixty-seven respondents said an unlicensed midwife attended their birth but only 12 birth certificates were correctly coded as such; 47 women said their birth attendant was a physician and 27 said the attendant was a licensed or certified nurse-midwife, for a combined total of 74 (36 percent) reporting that their birth attendant was actually licensed.

Table 2 shows the attendants and birthplaces for the 1656 out-of-hospital births with attendant classifications changed to reflect the 206 survey responses. The majority of these births occurred at home. Of the 76 births classified as having an unlicensed midwife attendant, 75 were home births. Of the 1,030 home births, licensed midwives accounted for the

TABLE 2—Out-of-Hospital Births by Type of Attendant and Place of Birth, Washington State, 1986

Birth Attendant	Out-of-Hospital Birthplace				
	Birth Center	Other Medical Facility	Home	Other	Total
Licensed Physician	155	97	78	8	338
Licensed Midwife	21	7	583	7	618
Certified Nurse-Midwife	267	2	131	0	400
Unlicensed Midwife	0	1	75	0	76
Mother/Father	0	1	65	2	68
Other	0	51	49	3	106
Unknown	0	0	49	1	50
Total	443	162	1030	21	1656

SOURCE: Washington State Vital Data, 1986, and 206 survey respondents

TABLE 3—Sociodemographic Characteristics of Mothers Who Gave Birth at Home By Attendant Type Washington State, 1986

Characteristics	Birth Attendant Types				
	% Unlicensed Midwife	% Licensed Midwife	% Nurse-Midwife	% Licensed Physician	% All Other*
Total	N: 75	N: 583	N: 131	N: 78	N: 163
Maternal age <20	1	2	2	9	7
Other than white race	4	4	3	26	13
Unmarried	15	11	5	29	18
Professional occupation (Father)	15	26	27	19	15
Professional occupation (Mother)	11	11	12	10	7
Rural county of residence	27	3	2	1	10

*Mother, Father, Other, Unknown
Source: Washington State Vital Data, 1986, and 206 survey respondents

greatest proportion of attendants; unlicensed midwives attended only 7 percent.

Licensed midwives and certified nurse-midwives were more likely than unlicensed midwives to serve urban, married professionals (Table 3), while unlicensed midwives attended mothers residing in rural counties.

Only 13 percent of respondents said their out-of-hospital birth was unplanned. All such respondents were attended by a mother, father, friend, or other person.

Reasons for the choice of an unlicensed midwife include: "I could not find a licensed birth attendant in my community willing to attend an out-of-hospital birth" (31 percent), and "A major factor in my decision was to have someone to care for me with religious beliefs similar to my own" (31 percent).

Most respondents attended by unlicensed midwives were satisfied with the care they received, although 25 percent reported that they would have preferred a licensed midwife or doctor. Sixty-seven percent said their unlicensed midwife charged a fee for her services.

Discussion

These results suggest that unlicensed midwives attend a small proportion (0.11 percent) of births in Washington State. However, for several reasons, our data may underestimate the true proportion of unlicensed midwife-attended births. If it is assumed that all unlicensed attendants of out-of-hospital births were actually unlicensed midwives, approximately 18 percent of the 1,656 out-of-hospital births would have been attended by unlicensed midwives, 0.43 percent of the state's total births. Even at this upper end of the range of plausible estimates, the proportion of unlicensed midwife-attended births is still quite small.

If the mother's questionnaire response is correct, the extent of attendant misclassification on birth certificates was surprising. Although we expected to find unlicensed midwife attendants concealed within the other unlicensed categories, there were also numerous instances of births attended by licensed providers that had been attributed to unlicensed attendants on the birth certificate. In these cases, birth certificates were either inaccurately completed or coded incorrectly.

Apparently many respondents chose an unlicensed midwife because they were unable to locate a licensed provider who would attend an out-of-hospital birth. This is consistent with the finding that women attended by unlicensed midwives are more likely to reside in rural counties than their counterparts attended by licensed midwives or certified nurse-midwives.

Shared religious beliefs was another reason cited for the choice of birth attendant. It was later learned that some unlicensed midwives regard their practice as a ministry to serve fellow churchwomen.

That two-thirds of mothers attended by unlicensed midwives paid a fee for service suggests that in most cases this was a professional relationship, not simply one friend helping another. Technically, unlicensed midwives who charge a fee are practicing illegally. However, because they are responding to the needs of particular groups of women, they are likely to continue to practice in spite of legal prohibitions. Legislation prohibiting unlicensed midwives from practicing is rarely enforceable.[2] Indeed, the real effect of such legislation has been to force unlicensed midwives to work in secret or to prevent them from seeking education and training.[2,3] Providing an avenue to legal midwifery practice may be a more appropriate course of action so long as competence, equivalent to other licensed providers, can be demonstrated.

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Occupational Physical Activity and other Risk Factors for Preterm Birth among US Army Primigravidas

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Abstract: We examined the relation of occupational physical activity to the risk of preterm birth among US Army active-duty primigravidas between 1981 and 1984 using 604 cases (preterm deliveries; ≤ 37 weeks gestation) and 6,070 controls (term and post-term deliveries). Women employed in the highest physical activity levels had increased odds of preterm delivery ranging from 1.69 to 1.75. The relation was not changed by adjustment for the effects of age, marital status, socioeconomic status, or education. Missing data suggest cautious interpretation. (*Am J Public Health* 1990; 80:728–729).

Introduction

Relatively few studies have examined the relation between physical activity on the job and the risk of preterm birth, and the findings have been mixed. In a French case-control study, risk of preterm birth was related to the number of sources of occupational fatigue and with a work-week longer than 40 hours.[1] This finding was not confirmed in a case-control study in New Haven, Connecticut;[2] decreased risk of preterm birth was seen among women participating in sports or physical fitness exercises during pregnancy.

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The population of US Army active-duty gravidas presents an unusual opportunity to study the relation between preterm birth and occupational physical activity because of its large size and homogeneity, and because US Army military occupational specialties (MOS) had undergone physical demand assessments.

Methods

In 1977, entry level specialties for enlisted personnel were categorized by maximum upper body strength requirements as required for "combat conditions" performance (see Appendix).[3] This physical activity measure was not a specific measure of actual physical activity during pregnancy.

Data for this study were obtained from Patient Administration Systems and Biostatistics Activity, Fort Sam Houston, Texas for length of gestation (from maternal and infant inpatient records) and from US Army Military Personnel Center, Alexandria, Virginia for physical activity and other demographic data (from fiscal year personnel files).

Each subject was assigned to her MOS physical activity level during pregnancy using the fiscal year corresponding to her mid-gestation date. US Army policy forbids the reassignment of women during pregnancy.

The population of US Army active-duty gravidas delivering at US Army medical treatment facilities numbered 22,450 during the four-year period 1981–84. This period was chosen because of differences in coding schema for inpatient records prior to 1981; data for deliveries after 1984 were incomplete at the time of this study. Eligibility criteria for the study sample included primiparity, singleton delivery, and enlisted paygrade status. Officer occupations had not been assessed for physical demand requirements.

Cases were defined as preterm deliveries (≤ 37 weeks gestation); controls included term and post-term deliveries.

Risk factors studied included age, race, pay grade, length of military service, marital status, education, occupational aptitude score, job skill level, and type of military unit. The latter two variables were included to control for the initial focus of the MOS physical demand analysis: "entry level skills in a combat environment." Military duties performed in a field unit closely resemble how they would be performed in a combat environment. The occupational aptitude score was included since it is also used to determine MOS eligibility.

Odds ratios were computed for each separate risk factor. The dose-response relation between occupational physical activity and preterm birth was assessed with the Mantel test for trend.[4] We also fitted a logistic regression model and estimated population attributable risk.[5]

Results

Of the 22,450 US Army active-duty deliveries between 1981–84, 20,584 were to enlisted women. Of these, 20,218 were single live births. Using the mothers' Social Security Numbers (SSN), 12,911 of the 20,218 (64 percent) maternal inpatient records could be matched to infants' inpatient records. Seventy-two percent (9,262) of these matched records met the primiparity criterion. Using the maternal SSN, 7,821 were matched with personnel records. Of these, 1,147 records could not be used due to MOS data entry errors. The remaining sample of 6,674 included 604 cases and 6,070 controls.

Odds ratios for each risk factor are presented in Table 1. Risk factors for preterm birth included the two highest physical activity categories, age less than 20 years, other than White race, pay grades E1 to E3, less than two years' total military service, a never-married status, and an occupational aptitude score less than 100. The level of physical activity was positively related to risk. Condensing the five-category physical demand model to three categories, we found only minor changes in odds ratio estimates and chi-square values by controlling for other risk factors one at a time using a stratified Mantel trend test.*

The analysis of a multifactorial model using logistic regression resulted in three significant predictors of preterm birth: physical activity, race, and military pay grade. The combined risk of a preterm birth for all three factors ranged from 3.3 for those employed in "heavy" physical activity to 3.6 for those employed in "very heavy" physical activity. The population attributable risk for the risk factor occupational physical activity was estimated as 17.5 percent when adjusted for pay grade and race.*

TABLE 1—Estimates of Relative Risk for Preterm Birth

Risk Factors	Number	Percent Preterm	Odds Ratio	95% CI
Physical demand				
Low	429	6.1	1.0	
Medium	1949	8.5	1.43	0.92,2.25
Moderately Heavy	940	8.4	1.42	0.88,2.31
Heavy	1825	9.8	1.69	1.08,2.64
Very Heavy	1531	10.1	1.75	1.12,2.75
(p-value, trend: 0.0056)				
Age (years)				
≥20	5925	8.5	1.0	
<20	749	13.8	1.73	1.37,2.18
Race				
White	2927	7.7	1.0	
Other	3747	10.1	1.34	1.12,1.60
Paygrade				
E4–E8	4361	7.6	1.0	
E1–E3	2313	11.8	1.64	1.38,1.95
Years of service				
≥2	4132	7.9	1.0	
<2	2542	10.9	1.42	1.20,1.69
Marital status*				
Ever Married	3434	8.1	1.0	
Never Married	3239	10.0	1.26	1.06,1.50
Aptitude Score*				
≥100	3871	7.6	1.0	
<100	2583	11.1	1.50	1.26,1.79
Years of education*				
>12	839	7.9	1.0	
≤12	5832	9.2	1.19	0.90,1.57
Job skill level				
Levels 2–5	1437	8.1	1.0	
Entry	5237	9.3	1.17	0.94,1.45
Type of unit				
Garrison	2430	9.2	1.0	
Field	4244	9.0	1.02	0.86,1.22

*Missing data

Discussion

The results of this study should be interpreted with caution for several reasons. First, a considerable amount of information was lost when maternal inpatient records were not matched to their infants' inpatient records or their personnel records. Approximately one-fifth of the lost information was from the births occurring in civilian hospitals which do not forward information pertaining to the infant to the nearest military facility. The remaining four-fifths of data were lost because social security numbers did not match. Prior to 1985, there was no uniform policy regarding the entry of social security number on an infant's inpatient record when both parents were active-duty. The impact of the lost information may be small, however, since the dependent variable, length of gestation, and the primary independent variable, physical activity, were obtained from two independent sources, and there is no reason to suspect any systematic bias with regard to these two variables.

A greater concern is that information was not available to this study on potential confounders such as nutrition practices, smoking habits, alcohol consumption, and pregnancy complications.

Other studies have shown activity-duty gravidas to be at high risk for adverse reproductive outcomes, particularly when compared to wives of active duty males.[6,7] Active-duty gravidas are required to continue work until delivery; although military physicians provide recommendations to the pregnant soldier's commander regarding restrictions, these are often general in nature and rarely address physical activity beyond prohibiting participation in regular unit physical fitness programs.

The strength of this study was the consistency of the observed relationship between occupational physical activity and the risk of preterm birth even after controlling for risk factors such as age, race, and socioeconomic status. Additional research in this area seems called for.

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The views presented here are of the authors and do not purport to reflect the position of the US Department of the Army or the Department of Defense.

*Data available upon request to author.

APPENDIX

US Army Physical Demand Categories

Category	Maximum Lift Criteria (lbs)*	
	Frequent or Occasional	Constant
Light	20	10
Medium	50	25
Moderately heavy	80	40
Heavy	100	50
Very heavy	>100	>50

*Occasional: <20% of the time
Frequent: >20% but < 80% of the time
Constant: >80% of the time

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Pawtucket Heart Health Program Point-of-Purchase Nutrition Education Program in Supermarkets

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Abstract: Point-of-purchase nutrition education in supermarkets is one intervention strategy of the Pawtucket Heart Health Program, a community cardiovascular disease prevention program in Pawtucket, Rhode Island.

Using consumer intercept interviews, awareness of shelf labels and their effect on purchase behavior have been continuously evaluated. Between 1984 and 1988, the percent of shoppers who could identify correct labels increased from 11 percent to 24 percent (95% confidence intervals of difference: 7,17). The percent who reported they were encouraged to purchase the identified foods increased from 36 percent to 54 percent (95% CI of difference: 5,41). (*Am J Public Health* 1990; 80:730–731.)

Introduction

Because 57 percent of the adult population has blood cholesterol levels that put them at moderate or high risk for cardiovascular disease (CVD),[1] many Americans need to change their dietary intake of saturated fat and cholesterol to reduce their blood cholesterol level and CVD risk.

To reach this large number of people, Blackburn and Kottke delineate the need for population strategies.[2,3] The Pawtucket Heart Health Program, a comprehensive community heart disease prevention study,[4–7] developed the "Four Heart Program," a point-of-purchase (POP) nutrition education program in supermarkets and restaurants, as one of its population strategies. We report the results of a study of consumer awareness of the supermarket program and its influence on self-reported purchase behavior.

Methods

The Four Heart supermarket program was started in three supermarkets owned by two different companies and one small family-owned market in 1983–84. The name, Four Heart, represents foods that are tasty and contain less fat, cholesterol, and sodium. The basis of the program is the placement of brand-specific shelf labels next to the unit price tags of qualifying foods. Original multi-colored shelf labels that identified products low in sodium, fat, and/or calories were replaced in 1986 with labels of uniform color with the messages: "low-fat", "low-sodium", "low-fat, low-sodium", and "fat ratio OK." Collateral and support materials include signs with health messages in areas where shelf labels cannot be applied; "Look for the Labels" posters with lucite holders for brochures; free recipe cards; periodic promotions such as contests and blood pressure and cholesterol screening, counseling and referral events (SCORES); and training of store and department managers and of lay volunteers as program monitors.

Criteria used to determine the eligibility of foods for these labels were adapted from the Minnesota Heart Health Program[8] and Food and Drug Administration (FDA) food labeling regulations and recommendations[9,10]

Whereas FDA-sponsored descriptive programs labeled all products in the store that met their nutrient criteria,[11,12] we were interested in studying a more prescriptive program. Only those foods included in meal pattern recommendations based on the US Dietary Guidelines for Americans[13] are labeled.

Consumer intercept interviews were conducted at approximately yearly intervals over four years in two supermarkets from two chains. Interviews were administered in a similar manner across all stores and time periods.

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Participants

Individuals exiting a store who were judged to be at least 18 years old were eligible for the interview. Once selected, an individual exiting the store was asked if s/he had purchased something in the store. Those who indicated that they had made a purchase were interviewed.

Procedure

Shoppers were selected for interviews based on a time sampling procedure. Using a stop watch, the interviewer selected a potential respondent by approaching the first age-eligible shopper to exit the store at five-minute intervals.

Instrument

Data on frequency of shopping were obtained. To assess awareness of shelf labels, a placard containing four sets of labels, three of which were bogus sets, was shown to the respondent. The shopper was asked if s/he saw any of these labels inside the store. If the answer was yes, s/he was asked to identify the set seen. Those shoppers who reported seeing the labels actually on the shelves were counted to assess the influence of the labels on purchase behavior.

Results

Awareness of Labeled Items

Interviews with 1,807 shoppers were conducted over the four-year period. Fifteen percent more shoppers reported seeing any labels on the store shelves between 1984 to 1988 (Table 1).

Awareness was related to gender; 32 percent of female shoppers versus 24 percent of male shoppers reported seeing one of the labels shown to them (95% CI = 4,12). Thirteen percent more shoppers were able to identify the correct label between 1984 and 1988 (Table 1). Female shoppers identified the correct labels 23 percent of the time compared with 12 percent for males (95% CI = 7,15).

Influence of Shelf Labels on Purchase Behavior

Encouragement to purchase labeled foods was reported more frequently in 1988 than in 1984 among shoppers who correctly identified Four Heart labels (Table 1). Encouragement to purchase labeled products did not differ appreciably between male and female shoppers.

TABLE 1—Consumer Awareness and Effect of Food Product Shelf Labeling

	Total Surveyed	Yes[a]	Correct[b] Label	Encouraged[c] Purchase
	n	n %	n %	n %
1984	355	72 (20)	39 (11)	14 (36)
1986	454	104 (23)	70 (15)	36 (51)
1987	418	150 (36)	98 (23)	58 (59)
1988	531	185 (35)	129 (24)	70 (54)
		95% confidence intervals of difference		
'88 vs '86		6, 18	4, 14	
'88 vs '84		9, 21	8, 18	7, 35
'87 vs '86		7, 19	3, 13	
'87 vs '84		10, 22	7, 17	5, 41

[a] Shoppers reporting having seen any label shown of total surveyed
[b] Shoppers correctly identifying the "4-Heart labels" of total surveyed
[c] Shoppers stating that label encouraged purchase of those who saw correct label

Discussion

The supermarket program reached large numbers of people with low fat, low sodium eating pattern messages over a four-year period of time. In order to quantify this reach, we applied findings from our study to industry averages obtained from the Food Marketing Institute, Washington, DC (personal communication). In 1987, the mean weekly store sales in US supermarkets was $184,210 with an average transaction of $16.30. This translates to 11,301 transactions per store per week.

Based on our interviews, customers shopped an average of 2.3 times a week. Therefore, we estimate that the participating supermarkets served 4,913 customers a week.

Projecting our findings from these averages, by 1988 each week an estimated 1,179 customers recognized Four-Heart shelf labels while shopping and 636 of them were encouraged to purchase labeled products. It would be difficult and expensive to achieve this exposure to health messages and change in purchase behavior with one-to-one or small group methods of nutrition education.

Although a larger percentage of females than males identified the correct labels, approximately one-half of both males and females reported that they were encouraged to purchase the labeled foods. This is of particular interest because males have lower participation rates in traditional nutrition education programs than females[14,15] and are at higher risk for CVD—one in three will have clinical signs of CVD before the age of 60.[16]

Two important caveats must be given to these results. The first is that this program evaluation occurred within the context of a large, broad-based intervention effort to raise awareness of, and stimulate changes in, CVD risk behaviors. Secondly, other secular trends such as the increasing media coverage of cholesterol-related topics, and the efforts of the National Cholesterol Education Program and American Heart Association in this area, may also have added to the observed increases.

Yet, our data do support the findings of other studies which have demonstrated that point of purchase nutrition education in supermarkets is feasible and effective in increasing consumer awareness of nutrition messages over time[11,17,18] and in exerting a positive influence on self-reported purchase behavior.[18] Because of the ambiguous results of studies of actual consumer purchase behavior,[11,12,17,19–22] further controlled studies of this intervention in supermarkets need to be undertaken.

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Infant Feeding Practices: An Evaluation of the Impact of a Health Education Course

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Abstract: We assessed the impact of a health education course on infant feeding practices in the West Bank territories by comparing mothers who had attended the course (n = 102) with mothers not exposed to the course (n = 133). After adjustment for child's age, maternal age and education, parity, and birth site, course participants were more likely than non-participants to breastfeed, as well as to start supplementation by semi-solid foods at the recommended time. (*Am J Public Health* 1990; 80:732–733.)

Introduction

A comprehensive project of health education for mothers of young children in the villages of the West Bank territories, a population of low socioeconomic status, was designed as part of a food distribution activity.[1,2] Prior assessment of needs and problems of the local population indicated that a growing number of the mothers wean their children too soon, and introduce semi-solid foods either too early or too late. One of the main objectives of the course was to change these feeding practices. The course was taught in small groups by specially trained local instructors who adapted teaching materials developed for the course to the specific questions and issues raised by their groups. The course focused on nutrition, hygiene, child development, and first aid. The process of course development, design, methods, and content were described elsewhere.[1,2]

From 1985 to 1987, the program offered 972 courses in 152 villages (about one-third of the 443 villages in the West Bank) and in five cities throughout the West Bank. Participation of villages in the program was voluntary. In participating villages, most mothers of young children joined the course. Participating mothers received a small supply of basic food commodities. Overall, 19,984 mothers participated in these courses.

The present paper assesses the impact of the course on infant feeding practices, by comparing a sample of mothers who had attended the health education classes with a sample of mothers who had not been exposed to the course.

Methods

A survey was conducted in 92 villages throughout the West Bank, 46 which participated in the program, and 46 which had not participated. The sample of villages was randomly selected in proportion to the population distribution in six districts. Approximately six mothers in each village were interviewed. In experimental villages, the respondents were randomly selected from the list of course participants. In the comparison villages, respondents were selected in most cases from among the mothers whose children were attending school at the time of the survey. Structured interviews were conducted between August and December 1987 by female field workers experienced in village habits and familiar with local customs and culture. The present analysis includes 235 mothers whose youngest child was one year or less. Of these, 102 attended the course and 133 mothers did not attend the course. Statistical analysis

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includes comparisons of crude and age-adjusted proportions. Confidence intervals (CI) for differences of crude and standardized proportions were calculated by methods suggested by Rothman.[3] Mantel-Haenszel chi square was used to assess statistical significance of stratified analysis.[4] Multivariate logistic regression was performed to assess the effect of the health education course on feeding practices, controlling for other variables.[3]

Results

There were no differences of consequence in maternal age, number of children, and maternal level of formal education between the two groups (Table 1), but 51.1 percent of the course participants delivered at home assisted by a local midwife (Daya) or by a female relative, while only 35 percent in the comparison group delivered at home. All other women delivered in a clinic or a hospital.

Proportions of breastfeeding mothers were higher in the course group than in the comparison group, irrespective of child's age (Table 2). Introduction of semi-solid foods at 4–6 months (the recommended pattern) was more prevalent in the course group than in the comparison group (76 and 57 percent, respectively; 95 percent CI for the 19 percent difference = 7.1–30.9). Starting semi-solid foods at age 6 months was a more common practice in the comparison group than in the course group (16 and 1 percent, respectively).

Women who gave birth at home were more likely to start breastfeeding than women delivering in the hospital (97 vs. 74 percent), and to be still breastfeeding at 10–12 months (58 percent vs. 42 percent). Comparing rates of breastfeeders (adjusted for child's age) demonstrates that course participants were more likely to breastfeed, regardless of site of delivery (home vs. hospital or clinic). The difference, however, significantly exceeds 0 only among mothers delivering in the hospital (Table 3).

After controlling for child's age, site of delivery, maternal age, level of education, and parity, course participation continued to be associated with breastfeeding at the time of interview, as well as with starting supplementation with semi-solid foods at age 4 months (Table 4). Breastfeeding was also associated with child's age and home birth. The recommended pattern of supplementation with semi-solid foods, was associated with course participation, maternal education, and number of children (Table 4).

Discussion

The pattern of findings is consistent with the conclusion that the course was successful in influencing mothers' feeding habits. The results, however, could have been influenced by several biases inherent in the study design. Since our study is cross-sectional, and since the course was not randomly assigned, it is possible that health education project villages differed from the comparison villages in aspects relevant to child feeding practices for which no information was available. However, stratification on site of last delivery and multivariate adjustment for maternal age and education, site of delivery, number of children, and child's age suggest that course participation had a unique contribution to feeding behavior.

Another possibility is that women in the health education group report, but do not behave, differently than the comparison group. Even if some reporting bias does exist, the mere knowledge in itself about appropriate practices is a

TABLE 1—Comparison of Socio-Demographic Characteristics of Mothers to Children 0–1 Years Old in the Experimental Group and the Comparison Group

	Experimental Group		Comparison Group	
	$\bar{X}$	SD	$\bar{X}$	SD
Age	27.4	(6.6)	28.5	(6.2)
No. of children	4.6	(2.6)	4.9	(2.7)
Years of schooling	6.1	(3.4)	5.2	(4.2)
% of women delivering youngest child in hospital or clinic	34.6		51.1*	

*Difference 16.5% (95% CI = 3.1, 29.9).

TABLE 2—Percent of Women Breastfeeding by Child's Age and Course Participation

	Experimental Group		Comparison Group	
Child's Age (mos)	%	n	%	n
0–3	100.0	(25)	78.4	(29)
4–6	84.0	(21)	65.7	(23)
7–9	70.8	(17)	62.5	(20)
10–12	64.3	(18)	37.9	(11)

Mantel-Haenszel $\chi^2 = 6.802$, $p < .01$ (3).

TABLE 3—Percent of Women Breastfeeding at the Time of Interview, Adjusted for Child's Age,[a] by Site of Delivery, and Course Participation

	Home Delivery		Hospital Delivery	
	%	n	%	n
Experimental Group	79.1	(66)	83.1	(36)
Comparison Group	68.7	(65)	54.3	(68)
Difference	10.4		28.8	
95% CI[b]	(−3.5, 24.3)		(13.9, 43.7)	

[a]Rates were adjusted for child's age by the direct method, using the age group distribution of the entire sample as the standard population.
[b]95% confidence intervals for standardized rates.[3]

TABLE 4—Results from Multiple Logistic Regression Analysis Predicting Infant's Feeding Mode

	Breastfeeding at Present (Yes/No)		Introducing Semi-Solid Food at 4–6 Months* (Yes/No)	
Variables	Odds Ratio	95% CI	Odds Ratio	95% CI
Course participation (yes/no)	2.81	2.03, 3.88	2.63	1.89, 3.70
Home delivery (yes/no)	1.67	1.23, 2.28	1.37	0.98, 1.93
Mother's age[a]	1.10	0.89, 1.35	0.81	0.64, 1.02
Mother's education[b]	1.10	0.94, 1.28	1.44	1.21, 1.71
N of children[c]	1.00	0.82, 1.22	1.32	1.05, 1.65
Child's age[d]	1.85	1.61, 2.13	1.03	0.85, 1.26

[a]Age: (<25, 25–29, 30–35, >35)
[b]Years of schooling: (0, 1–5, 6–8, >8)
[c]Number of children: (1–2, 3–4, 5–6, >6)
[d]Months: (0–3*, 4–6, 7–9, 10–12)
*Only mothers whose youngest child was 4 months or older at the time of the interview were included in this part of the analysis.

worthwhile benefit of the course.

Our findings are consistent with a previous study showing a beneficial effect of special class on breastfeeding among mothers of low socioeconomic status.[5] These findings are encouraging in light of the trend observed among mothers of low socioeconomic groups,[6,7] and mothers in third world countries of declining rates of breastfeeding.[8]

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Pertussis Immunization in Eight-Month-Old Children in North Carolina

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Abstract: Between 1984 and 1987 reported pertussis cases in North Carolina increased threefold. Pertussis immunization rates were examined for those years in three one-year cohorts drawn from a random selection of North Carolina birth records. The percentage of children immunized with three DTPs at eight months of age was 58.1, 58.6, and 56.7 for the three cohorts. Only 20.5 percent of 117 reported pertussis cases in children 9–36 months of age during the last 10 years were adequately immunized. The low pertussis immunization rate may have contributed to the recent increase in pertussis cases in North Carolina. (*Am J Public Health* 1990; 80:734–735.)

Introduction

The United States has reported a recent increase in the number of pertussis cases.[1,2] In North Carolina the annual average of reported pertussis cases increased from 40 for the 10-year period 1975–85 to 88 and 123 cases in 1986 and 1987 respectively.

Although it is estimated that over 95 percent of North Carolina children entering primary school have the pertussis immunizations required recently by state law, a 1980 sample survey in North Carolina estimated that only 66 percent of two-year olds had received the recommended four DTP (diphtheria/tetanus/pertussis) injections.[3] Even less is known about the immunization status of children under one year of age who are at the greatest risk of severe pertussis disease and account for the majority of the cases reported and hospitalized.[4]

Our study was designed to examine the immunization status of eight-month old children in North Carolina during the time period 1984 to 1987, immediately preceding and during the pertussis epidemic.

Methods

A systematic random sample of 600 birth records was selected from all recorded North Carolina births between May 1, 1984 and April 30, 1985. Information on DTP immunization status was obtained using mailed questionnaires; with telephone or health department record follow-up for non-responders. From each of two birth cohorts: 5/1/85–4/30/86 and 5/1/86–4/30/87, 327 birth records were similarly selected. A 10 percent random sample was selected from those not responding for telephone interview. The telephone information was extrapolated to represent a similar fraction of the entire nonresponder group in each cohort to give an adjusted immunization rate.[5]

A child was considered properly immunized if three DTP injections were received by 244 days (eight months) of life. A few extra weeks beyond the recommended age (six months of age) for the third DTP immunization was allowed to avoid depressing the primary series completion rate by those who were only minimally delayed in obtaining immunization.

Confidence intervals for proportions were calculated using the normal approximation to the binomial distribution when n was sufficiently large. Hypothesis tests were performed using the normal approximation to the binomial for proportions, chi-square test statistic, Fisher's exact test or chi-square test for trend as appropriate.[6,7]

Results

The response rate in the 1985/86 and 1986/87 cohorts (41 percent and 49 percent, respectively) were lower than the response rate in the 1984–85 cohort (70 percent). Data were obtained by telephone from 44.4 percent of the 1985/86 and from 61.9 percent of the 1986/87 subsamples of nonresponders and used to adjust the final rates.

The adjusted immunization rates were similar for the three study years; 58.1 percent (1984/85), 58.6 percent (1985/86), and 56.7 percent (1986/87) (chi-square test for linear

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trend p = .737). Racial characteristics of the adjusted cohorts were similar to those of North Carolina in 1985 and did not play a role in the differences between cohort groups. There were significant differences, however, between the immunization rates for White (1984/85 63.2 percent, 1985/87 62.7 percent) and Black (1984/85 48.3 percent, 1985/87 39.9 percent) children in the adjusted rates.

There were 505 cases of pertussis reported in North Carolina between 1978–87, 350 (69.3 percent) were children less than one year of age (Table 1), the majority occurring in children less than nine months of age (Figure 1). Table 1 shows the immunization rates for the cases by age; 20.5 percent of children aged 9–35 months of age between 1978–87 and 21.4 percent of children for the years 1986/87 were adequately immunized. The median age of cases from 1978 to 1986 was five months and the median age for cases in 1986/87 was seven months.

Extrapolation from the World Health Organization (WHO) nomogram[8] for percent of cases vaccinated and vaccine efficacy (estimated to be 80 percent) gives a population vaccination rate of 55–60 percent.

Discussion

The DTP immunization rates for young North Carolina children as determined in this study were disturbingly low between 1984 and 1987, but remained constant over the three-year period. Therefore, our findings do not substantiate a decreasing rate of pertussis immunization during the study period. Due to the poor response rate, however, particularly in the 1985/87 cohorts, an undetected immunization rate change may exist. The finding that nonresponders located were less well immunized than the initial responders suggests that the rate of pertussis immunization may have been closer to 50 percent if all nonresponders had been contacted.

The recent increase in reported pertussis cases may be due to causes other than failure to immunize. Increased case reporting alone is unlikely because the number of children under one year of age with pertussis requiring admission to major hospitals in North Carolina also increased during 1986/87. The fact that over 40 percent of the reported cases of pertussis were under five months of age also suggests that the low immunization rate in young children was not the sole cause of the increasing incidence of pertussis. However, inadequate immunization of preschool-aged siblings could increase the chance of transmission to young infants. Immunizations are required to be up-to-date when children enroll

TABLE 1—Immunization Status of Reported Pertussis Cases in North Carolina 1978–87

Age at Disease (months)	# DTPs Required	# Immunized	%
<2	0	NA/48	NA
2–4	1	53/161	32.9
5–8	2	27/113	23.9
9–21	3	18/84	21.4
22–36	4	6/33	18.2
37–72	4	11/16	68.8
>72	5	21/50	43.8
TOTAL		136/505	26.9

NA = not applicable. If the 48 children in this group are removed from the denominator because they probably would not have been immunized, the overall immunization rate in the total group would be 136/457, 29.8 percent.

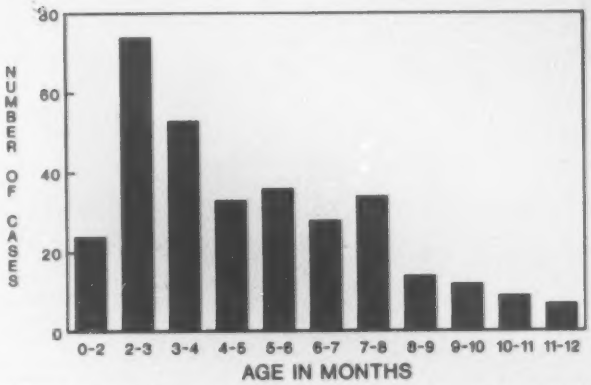

FIGURE 1—North Carolina Pertussis Cases Less than One Year of Age, 1978–87

at certified day care centers in North Carolina but continued updating of records is not normally required and may not be performed. A gradually cumulative increase in this pool of preschool-aged susceptibles could have propagated transmission to younger children during this epidemic.

Bass[1] has suggested that part of the reason for the recent increase in reported pertussis cases in the United States is that there is an increasing proportion of the population that is vaccine-immune to pertussis which is gradually losing its immunity. If older children and adults acquire asymptomatic infection, they could be a source of infection for others not yet immune, particularly unimmunized young children. It is of interest that the median age of pertussis cases increased from five months to seven months after 1985, coincident with the increase in the number of pertussis cases. It is also possible that an increasing use of day care facilities provided increased opportunities for incompletely immune children to be exposed to asymptomatically infected children and adults.

The increase in the number of pertussis cases and the delay in immunization that this and other studies[9] document is cause for concern. The disparity between the immunization rate of White and Black children is also of concern. It is likely, however, that race is a surrogate for socioeconomic status.

The task now is to better define groups of children that are poorly immunized, evaluate the barriers that prevent timely immunization, and formulate appropriate interventions.

ACKNOWLEDGMENTS

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Loss of Maternally Acquired Measles Antibodies in Well-Nourished Infants and Response to Measles Vaccination, Peru

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Abstract: Clinical, anthropometric, and serological evaluations were conducted at birth and at 3, 6, 9, and 10 months (post measles vaccination) in 34 well-nourished Peruvian infants. Seroconversion rate after measles vaccination was 94 percent. The rate of antibody loss was a direct function of birth titer; at age 9 months, all children had identical mean titers regardless of their titer at birth. Differences in maternally acquired measles antibodies at birth were important only during the first six months of life. (*Am J Public Health* 1990; 80:736–738.)

Introduction

Infants in developing countries become susceptible to measles infection at younger ages than do infants in the industrialized nations.[1–3] Seroconversion rates after measles vaccination among infants younger than 9 months of age in Africa and Latin America are higher than in US infants of the same age.[4,5] Thus, maternally acquired antibodies against measles do not appear to confer the same length of protection in third world infants as in infants from developed countries.

It has been proposed that a lower maternal level of antibody, hence a lower circulating level in the newborn, may be responsible for the early susceptibility to measles infection in infants from low-income regions.[6] Alternatively, Halsey, *et al*,[7] have suggested that third world infants may have an increased rate of measles antibody loss. Elucidation of the reasons for early susceptibility to measles may allow design of more appropriate vaccination schedules.

Methods

The investigation was conducted in San Martin de Porres, a poor district of the city of Lima, Peru. One hundred full term, normal birthweight infants born at Cayetano Heredia Hospital were included in this longitudinal study at the time of birth. The parents agreed to participate in this investigation and brought their children to the hospital for evaluations every three months. Children were examined by a physician at birth and at 3, 6, 9, and 10 months of age. At every visit, mothers were interviewed to assess past infection with measles and other major illnesses. At age 9 months, all participating children were vaccinated against measles and one month later their response to the vaccine was assessed.

Weight and height measurements were made at each visit. Weight for age, weight for length, and length for age were calculated using the National Center for Health Statistics and Waterlow's classification.[8,9]

A venous blood sample was obtained from the mothers at the time of delivery. A small blood sample was obtained from each child at the time of recruitment and at each visit. The last sample (at 10 months) was used to assess seroconversion to measles vaccination. All samples were frozen at −70 °C until processed together.

Circulating IgG specific against measles was measured by ELISA using measles-infected Vero cells and peroxidase-conjugated rabbit immunoglobulin to human IgG according to the method originally described by Rice, Casali and Oldstone.[10] Each serum was diluted serially in saline containing 10 percent fetal bovine serum and 0.05 percent Tween 20 and aliquots of each dilution were assayed. Absorbance was read using a manual EIA plate reader. Positive and negative control sera were included in each assay plate. Also, negative control wells using uninfected Vero cells were included in all assay plates. Absorbance was plotted against log of serum dilution. Using linear regression, the intercept with the abcisa was determined and this dilution value, at zero absorbance, was taken to be the titer.

Attenuated live measles virus vaccine (Connaught, manufactured in Canada) was obtained from the Ministry of Health of Peru and stored at 4C. Each dose contained not less than 1,000 TCID50 of measles virus.

Results

Thirty-four subjects, 17 boys and 17 girls, completed the study and only the samples corresponding to these infants were processed. The nutritional status of the children was normal throughout the study with the exception of three girls who showed mild wasting at 3 months of age but recuperated quickly.

Table 1 shows the mean circulating measles antibody titers for mothers and infants at birth and at 3, 6, and 9 months of age and at post vaccination (10 months of age).

In order to follow the decline of maternally acquired measles antibody, infants were grouped into high, intermediate, and low birth titer. Mean levels of measles antibody were plotted as a function of age for the three groups of infants as shown in Figure 1. Infants with higher birth titers

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TABLE 1—Mean Geometric Measles Antibody Titers (GMT)[a,b]

	Boys	Girls	All
Mother	1502 ± 211 (7.31)	1622 ± 374 (7.39)	1561 ± 217 (7.35)
Infants			
at birth	2538 ± 400 (7.84)	1833 ± 580 (7.51)	2146 ± 357 (7.67)
at 3 months	393 ± 37 (5.97)	341 ± 70 (5.83)	364 ± 41 (5.90)
at 6 months	244 ± 40 (5.50)	228 ± 27 (5.43)	236 ± 24 (5.46)
at 9 months	80 ± 12 (4.38)	72 ± 8 (4.28)	76 ± 7 (4.33)
Post-vaccination	3290 ± 484 (8.10)	2975 ± 515 (8.00)	2982 ± 370 (8.00)

[a]mean geometric titer ± standard deviation
[b]values in parentheses = log2 GMT

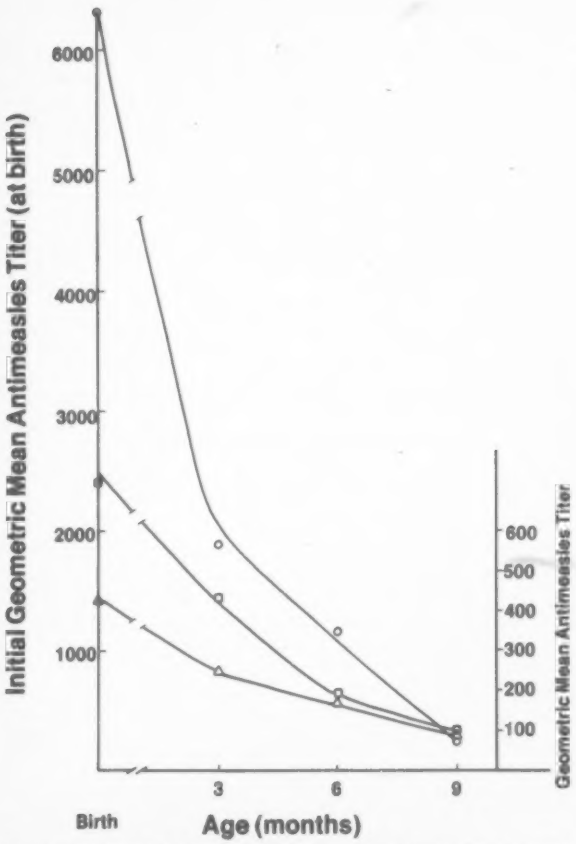

FIGURE 1—Geometric Mean Antimeasles Titer at Birth (left ordinate) and at 3, 6 and 9 Months (right ordinate) Plotted as a Function of Age
The break in the lines indicates that the points to the right of the break correspond to the scale on the right ordinate. Open circles (○) correspond to the group of infants with a high birth titer; open squares (□) to infants with intermediate birth titer; open triangles (△) to infants with low birth titer.

lost their antibodies more rapidly than infants with lower birth titers.

The half-life of maternally acquired measles antibody, calculated as described by Lennon and Black,[11] is shown in Table 2. The mean half-life for the three groups of infants was inversely related to birth antibody titer.

Seroconversion rate after measles vaccination was 94 percent among these children. Due to the method used to calculate antibody titer that led to a high sensitivity for our ELISA assay, seroconversion was arbitrarily defined as a post-vaccination titer above 1,000 and at least an order of magnitude higher than the prevaccination value.

TABLE 2—Half-life of Maternally Acquired Measles Antibody for Infants grouped into Low, Intermediate, and High Birth Titer

Group	n	Birth Titer [†]	Half-life (days)[*]	Post-Vaccination Titer
1	15	1000–2000 (1387 ± 64)	61 ± 13	3588 ± 489
2	9	2000–3000 (2346 ± 68)	59 ± 15	4098 ± 781
3	10	>3000 (6289 ± 1021)	46 ± 16	4067 ± 1404

[*]Calculated using circulating antibody titers at 3 months of age.
[†]Number in parentheses is mean of geometric titer at birth ± standard error for each group.

Discussion

Our results show that during the first three to six months of life, infants born with higher levels of measles antibody are better protected against the disease than infants born with lower titers. After 6 months of age this difference becomes tenuous and disappears completely by age 9 months.

Our results are in agreement with several studies that have suggested that the rate of degradation of circulating antibodies depends on the total IgG concentration.[12,13] This phenomenon has been reported in mice,[14,15] rabbits,[16] and human beings.[17–19] The half-life of passively administered gammaglobulin in children with agammaglobulinemia was 50–60 days whereas, in normal children, it was 20–40 days.[20] Cloonan, Hawkes and Stevens[21] have reported that babies having high rubella titers at birth lose their antibody rapidly, whereas in babies with low birth titers, antibody declines over a longer period of time.

The antibody half-life estimate from our data, 56.3 days, is similar to that calculated by Black, *et al*,[6] for Taiwanese children (53.3 days) but longer than that observed in children from Kenya (46.8 days).[22]

It is interesting to note that boys showed higher antibody titers than girls at all ages, although this may not hold up with larger samples.

Black, *et al*.[6] have used maternal and cord measles data from 15 populations from nine countries to demonstrate a strong inverse correlation between per capita income and the age at which children respond to measles vaccination. The authors have observed also a correlation between maternal antibody titer and the age at which children seroconverted. These findings led them to postulate that children in developing countries become susceptible to measles infection at an earlier age than children in industrialized countries because they receive less antibodies from their mothers.

Our study, even though it involved a small number of healthy and well-nourished children, suggests that infants appear to have equal protection after eight months of age regardless of what their antibody level was at birth. The fact that no correlation was observed between infant titer at birth and post vaccination titer strengthens the validity of our findings. If the length of protection conferred by maternal antibody as suggested by Black, *et al*,[6] were primarily a consequence of infant titer at birth, one would expect vaccination titer to be an inverse function of birth titer. This was not the case in our study. Other factors are probably involved in the early loss of passive measles immunity in children from developing countries.

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The Prime Time Diet: A Content Analysis of Eating Behavior and Food Messages in Television Program Content and Commercials

MARY STORY, PHD, RD, AND PATRICIA FAULKNER, MS

Abstract: The purpose of this study was to identify and analyze messages related to food and eating behavior as presented on prime time television (8:00–11:00 pm) both in programming and commercials. Food references occurred an average of 4.8 times per 30 minutes of programming time. Over half (60 percent) of all food references in programs were for low nutrient beverages and sweets. The prime time diet is inconsistent with dietary guidelines for healthy Americans. (*Am J Public Health* 1990; 80:738–740.)

Introduction

The American population spends more time watching television than it spends in any other activity except sleep and work.[1] There is increasing concern about the impact of this powerful and pervasive medium on health and health-related behaviors.[1–6] While the exact nature of the impact of television on behavior is controversial and largely unstudied, there is no doubt that many health-related images and messages are constantly conveyed to viewers, and may influence health behaviors. Because of the accumulating knowledge of the relationship of diet to health and chronic diseases, we thought it would be interesting to see how food and eating behaviors are portrayed on television, and whether these messages and behaviors are consistent with dietary recommendations for good health. The purpose of our study was to identify television messages related to food and eating behaviors both in programs and in commercials. Prime time television (8:00–11:00 pm) was selected for content analysis because it attracts the largest viewing audience among all age groups, with the exception of preschoolers.

Methods

The 1988 Nielsen Report on Television[7] was used to select the top nationally ranked prime time evening network programs for analysis. From the 15 top ranked shows among total US television households, only those featuring dramatic or situational comedy series were selected for analysis. Eleven programs were analyzed: the Bill Cosby Show; A Different World; Cheers; Growing Pains; Who's The Boss; Murder She Wrote; Golden Girls; L.A. Law; Moonlighting; Family Ties; and Alf.

Each program was video taped on two separate occasions during a nine-week period in the summer of 1988. A trained assistant viewed each video tape and coded all references to food and all eating occasions in programs and

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commercials on an instrument adapted from one developed by Kaufman.[8] The coding was checked for reliability by double coding a subsample of programs by an independent observer. For each reference to food or drink in programming, the coder identified the food or drink involved, whether it was a verbal or visual reference, or an actual eating scene, and the context in which food was eaten (meal or snack). Incidental visual references to food (e.g., a basket of fresh fruit on a kitchen counter) were counted as food references. Verbal references using food in a figurative sense, such as referring to someone as "the big cheese" were not included. All commercials aired during the show were recorded but only those advertising food or beverages were analyzed. The following information was coded: the product name and company, any explicitly expressed messages or claims about the food being advertised, and food shown other than that being advertised.

Results

Food references in prime time shows (excluding commercials) occurred an average of 4.8 times per 30 minutes of programming time (134 food references). References to low nutrient beverages (coffee, alcohol, soft drinks) occurred at a higher rate than any other category of food (Table 1). Over half (60 percent) of all food references in prime time programs were for low nutrient beverages and sweets.

TABLE 1—Rates of Occurrences of References to Food in Television Programs by Food Category*

Food Category	Total References (n)	Rate (per Half Hour)
Low-Nutrient Beverages		
Coffee	26	0.93
Alcohol	18	0.64
Soft drinks	12	0.43
Total	56	2
Sweets		
Cakes, cookies, desserts	13	0.46
Donuts, sweet rolls	8	0.28
Candy	2	0.07
Ice cream	1	0.04
Total	24	0.86
Meats		
Beef	9	0.32
Poultry	1	0.04
Fish	1	0.04
Sandwiches	6	0.21
Total	17	0.61
Fruits and Vegetables		
Fruits	3	0.11
Fruit juice	3	0.11
Vegetables	7	0.25
Total	13	0.46
Salty Snacks		
Popcorn	4	0.14
Peanuts	3	0.11
Pretzels	2	0.07
Total	9	0.32
Breads and Cereals		
Cereal	4	0.14
Breads	2	0.07
Muffins	1	0.04
Crackers	1	0.04
Total	8	0.29
Dairy Products		
Cheese	2	0.07
Milk	5	0.18
Total	7	0.25

*A total of 28 half-hour segments were viewed.

TABLE 2—Number and Rates of Food Commercials Shown during Prime Time Television Programs by Category*

Types of Food Advertised	Total References (n)	Rate per 30 minutes	Rate per Minute
Restaurants			
Fast food	23	0.82	0.10
Family style	1	0.04	0.00
Low-Nutrient Beverages			
Soft drinks, regular	7	0.25	0.03
Soft drinks, diet	4	0.14	0.02
Coffee/tea	3	0.11	0.01
Beer/wine	5	0.18	0.02
Cereals			
Sugared	1	0.04	0.00
Unsugared	17	0.61	0.08
Sweets/Desserts			
Ice cream, frozen treats	5	0.18	0.02
Cake, candy, cookie, etc.	7	0.25	0.03
Entrees			
Convenience entrees	5	0.18	0.02
Dairy Products			
Cheese	1	0.04	0.00
Yogurt	3	0.11	0.01
Fruits			
Orange juice	2	0.07	0.01
Raisins	1	0.04	0.00
Salty Snacks	2	0.07	0.01
Miscellaneous			
Butter	2	0.07	0.01
Condiments	2	0.07	0.01

*A total of 28 half-hour segments were viewed.

In the 22 shows, there were 86 occasions of eating or drinking. Almost three-fourths (72 percent) of the food eaten on prime time shows is eaten between meals. Snacking occurred at a rate of 2.2 times per half hour of programming versus .86 times for meals. The most frequent snack food was sweets (44 percent), followed by salty snack foods (25 percent). Less than 10 percent of snacks were fruits (3 percent) or vegetables (6 percent). Coffee (43 percent) was the most frequently consumed beverage, followed by alcohol (28 percent) and soft drinks (17 percent).

Of the 24 episodes involving meals, 12 were evening meals and six were breakfast and six lunch. Breakfast foods consisted of either cereal and toast (50 percent) or sweet rolls and donuts (50 percent). Fruit was shown three times at breakfast and once at lunch. In almost one-third (33 percent) of the noon and evening meal episodes, the only food portrayed was dessert (with either coffee or wine). Other foods shown for these meals were sandwiches (28 percent); salads (11 percent); entrees (28 percent), consisting of stir fry vegetables and chicken which the children refused to eat; lobster with butter; hot dogs and veal with potatoes. One-third of all meal episodes included alcohol.

Of the 261 commercials aired, 91 (35 percent) were food ads. Table 2 shows the number and rate of food references in commercials by food category. Commercials for fast food restaurants occurred at a higher rate than any other food category. The fast foods advertised were hamburgers, fried chicken, pizza, and breakfast sandwiches. None of these commercials advertised salads or salad bars. Only three commercials advertised fruit, and none advertised vegetables; however, fruits and vegetables were incidentally shown in 36 percent of all food ads (e.g., orange juice in an ad for breakfast cereals; fruit shown in an ad for candy).

The most frequently expressed message in commercials were claims of "good taste" and food being "fresh and natural." Few commercials made explicit nutritional claims. Those that did tended to be ads for unsweetened breakfast cereals, claiming that the products were nutritious, low in fat and sodium, and high in fiber.

Discussion

References to food were pervasive, both in prime time programs and in accompanying commercials. The "prime time diet" primarily consisted of foods low in nutritional quality, such as low nutrient beverages, sweets, and salty snack foods. Foods were typically consumed as snacks rather than meals. This reflects the eating pattern of the American population: diets high in sugar, sodium, and fat and low in fruits, vegetables, and fiber.[9–12] Recent data,[13,14] also indicate that both adults and children are snacking more frequently, and that families are eating fewer meals together. In light of the scientific evidence on the relationship of dietary factors to health and chronic disease risk, several studies, as well as numerous national reports and papers, have pointed to the wide-spread need for public education and changes in dietary behavior.[9–12,15]

Given our findings that messages related to eating practices are conveyed frequently during prime time television viewing, and that the television diet exemplifies poor nutritional practices, an important but unanswered question is whether television exposure to food references has any impact on actual eating behavior. Modeling and social learning theory would suggest that frequent, regular TV exposure might influence eating habits. However, this is speculative and further research is needed.

We believe media can be important in creating social norms and promoting healthy eating practices. Much of the eating on TV programs is trivial and incidental, used for "stage business," that is something to do with one's hands. Although producers, writers, and directors are in the business of delivering entertainment, and not with projecting health images, there is an opportunity to present health promoting messages by having characters model good dietary practices. Health professionals should initiate and work with the TV industry to encourage the inclusion of healthy eating patterns in programming.

ACKNOWLEDGMENTS

The authors thank Anne Ganey for technical assistance. This study was supported, in part, by the MCH Grant #MCJ-009118-01-0.

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NIH Consensus Panel Issues Conference Report on Treatment of Destructive Behaviors in Persons with Developmental Disabilities

In September 1989, the National Institute of Child Health and Human Development co-sponsored a Consensus Development Conference on "Treatment of Destructive Behaviors in Persons with Developmental Disabilities." The panel's report, which contains recommendations and conclusions concerning treatment of such behaviors, has been printed and single copies are available free from: William H. Hall, Director of Communications, Office of Medical Applications of Research, National Institutes of Health, Building 1, Room 259, Bethesda, MD 20892. Tel: 301/496-1143.

Seat Belt Use on Interstate Highways

JoAnn K. Wells, BA, Allan F. Williams, PhD, Adrian K. Lund, PhD

Abstract: More than 5,000 miles of limited-access highways in the eastern United States and Canada were traveled to observe seat belt use. Overall belt use was 58 percent in the United States and 79 percent in Canada. The data indicate that belt use in the United States follows a different pattern on interstate highways than on other streets and roads, with relatively high belt use rates (over 50 percent) appearing to be somewhat independent of belt use law provisions. (*Am J Public Health* 1990; 80:740–742.)

Introduction

Information on seat belt use is typically based on observations of motorists on city streets. For example, the National Highway Traffic Safety Administration (NHTSA) has surveyed belt use in 19 major cities annually since 1975.[1] However, belt use is known to vary according to where observations are made, and one location where belt use typically is higher than on city streets is interstate highways. In surveys in four states, belt use was 10–20 percentage points higher on interstate highways connecting the cities than in the cities.[2] Systematic information on seat belt use in vehicles traveling outside cities is lacking. In the four-state study it was collected incidentally as observation teams traveled between cities.[2] The present study provides information on belt use on interstate highways in the eastern United States and compares use rates in states and Canadian provinces with and without belt use laws.

Methods

Seat belt use observations were made by trained observers sitting behind the driver of a passenger van that traveled slower than the prevailing traffic in the far right lane. From that location, observers could clearly see shoulder belt use of front seat occupants of cars, light trucks, and vans passing in the adjacent lane. Lap belt use was not observed. Most cars on the road (all vehicles beginning with model year 1974) have integral lap/shoulder belts in the driver and right front seating positions. However, pre-1974 cars were not excluded so belt use is slightly underestimated.

Belt use observations were made on 5,129 miles of interstate highways in 21 eastern states and on limited-access highways in two eastern Canadian provinces from 9 am to 4 pm weekdays in August 1988. Appendix I provides information on the jurisdictions and highways surveyed and belt use law status.

It should be noted that the kinds of travel on these highways can vary tremendously from state to state. For example, in New York state, I-95 is entirely urban with short distances between access points and large volumes of local traffic; in South Carolina the same interstate is entirely rural with relatively long distances between access points.

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Observed differences in belt use for different populations were tested by χ^2 test of independence; only differences $\alpha = 0.05$ are reported.

Results

Seat belt use by the 14,354 front seat occupants observed on the highways surveyed averaged 58 percent in the United States and 79 percent in Canada. Belt use ranged from 48 percent to 66 percent in the 21 states and was 76 percent in Ontario and 84 percent in Quebec (Table 1). In the United States use was higher in states with laws (60 percent) than states without laws (56 percent), but the range of use rates for states with and without laws overlapped considerably. There was no significant difference in belt use between states with laws permitting primary and secondary enforcement.

The majority of occupants (57 percent) were in vehicles registered in the state or province in which they were observed. Of those traveling in out-of-state vehicles, motorists from jurisdictions with seat belt laws were more likely to use belts than motorists from non-law jurisdictions, whether observed in states with laws (66 vs 57 percent), provinces with laws (81 vs 74 percent), or non-law states (66 vs 56 percent).

Belt use was higher among drivers; in cars, utility vehicles, and passenger vans; among those estimated to be age 25 or older; and among women (Table 2).

Discussion

Belt use on interstates is higher than belt use in cities in the same state. Even in states without laws, belt use on interstates was greater than 50 percent, about 20 percentage

TABLE 1—Interstate Belt Use, by Jurisdiction

Jurisdiction	Belt Use %	Belt Use (N)	Standard Error of Percent Belt Use
United States			
Law	60	(7,943)	0.6
Connecticut	58	(526)	2.2
Florida	56	(2,149)	1.1
Illinois	55	(257)	3.1
Indiana	58	(311)	2.8
Maryland	66	(697)	1.8
Michigan	59	(581)	2.0
New Jersey	65	(477)	2.2
New York	54	(98)	5.0
North Carolina	59	(490)	2.2
Ohio	64	(453)	2.3
Pennsylvania	66	(605)	1.9
Tennessee	51	(476)	2.3
Virginia	63	(823)	1.7
No Law	56	(3,311)	0.9
Delaware	64	(59)	6.2
Georgia	59	(1,265)	1.4
Kentucky	64	(140)	4.1
Maine	52	(711)	1.9
Massachusetts	49	(404)	2.5
New Hampshire	54	(63)	6.3
Rhode Island	48	(212)	3.4
South Carolina	60	(457)	2.3
Canada			
Ontario	76	(2,042)	0.9
Quebec	84	(1,058)	1.1

TABLE 2—Interstate Seat Belt Use by Front Seat Occupants by Vehicle and Motorist Age, Sex, and Seating Position

Characteristics	Belt Use %	Belt Use (N)
Vehicle Type		
Car	66	(11,447)
Utility	61	(471)
Pickup	36	(1,100)
Passenger Van*	73	(663)
Other Vans	42	(644)
Unknown		(29)
Estimated Age (years)		
<25	56	(2,412)
25 or older	64	(11,921)
Unknown		(21)
Sex		
Female	66	(5,694)
Male	61	(8,620)
Unknown		(40)
Seating Position		
Driver	64	(9,519)
Right front passenger	61	(4,835)

*The following passenger vehicles in this category are: Nissan Van, Toyota Van, Mazda MPV, Chevrolet Astro, GMC Safari, Ford Aerostar, Dodge Caravan, Dodge Grand Caravan, Plymouth Voyager, Plymouth Grand Voyager, and Mitsubishi Van. The majority of these vans are sold as passenger vehicles, but the current data include observations of cargo versions as well.

points higher than in cities in these same states surveyed by NHTSA.[1] The laws do make a difference, however, not only in elevating use rates within the states, but motorists with vehicles registered in states with laws are more likely than motorists registered in states without laws to use seat belts wherever they travel. Nevertheless, variations in belt use laws affected belt use less on interstates than such laws are known to do on other highways, i.e., city-based surveys have found belt use to be higher in states with primary enforcement laws.[1,3] This should not be seen as an indication that belt use law differences are unimportant but that there is a different, and higher, pattern of belt use on interstates.

The reasons for higher interstate belt use are not clear. The population of motorists on interstates may be different than on other roads; for example, on average they may be of higher socioeconomic status, which is strongly related to belt use.[4] Also, people often claim in surveys that they are more likely to buckle up for long trips on high speed highways, although no observational studies have adequately tested this claim.[5]

Use rates are high both in Canadian provinces in general and on their limited-access highways.[6] Belt use in Canada was less than 50 percent in the 1970s.[7] Extensive use of programs combining publicity and enforcement during the past decade is thought largely responsible for Canada's present high belt use.[8,9] Such programs have been run successfully on a limited basis in the United States but have not been used extensively enough to increase overall belt use.[10,11]

ACKNOWLEDGMENT

This work was supported by the Insurance Institute for Highway Safety.

APPENDIX I
Highways and Jurisdictions Surveyed

Jurisdictions	Routes	Mileage	Belt Use Law* at Time of Survey
United States			
Connecticut	I-95	119	Primary
Delaware	I-95, I-295	16	No
Florida	I-75, I-95	859	Secondary
Georgia	I-75, I-95	499	No
Illinois	I-94, I-57, I-24	371	Secondary
Indiana	I-94, I-80	44	Secondary
Kentucky	I-24	101	No
Maine	I-95	298	No
Maryland	I-495, I-270, I-70, I-95	225	Secondary
Massachusetts	I-95	91	No
Michigan	I-75, I-94	283	Secondary
New Hampshire	I-95	20	No
New Jersey	NJ Turnpike, I-95	138	Secondary
N. Carolina	I-95	180	Primary
New York	I-95	23	Primary
Ohio	I-76, I-80, I-80/90, I-75	224	Secondary
Pennsylvania	I-70, I-70/76, PA Turnpike, I-76	192	Secondary
Rhode Island	I-95	38	No
S. Carolina	I-95	193	No
Tennessee	I-24	199	Secondary
Virginia	I-495, I-95	192	Secondary
Canada			
Ontario	Rt. 401	512	Primary
Quebec	Rt. 20	314	Primary

*Primary: Motorist can be stopped or cited for a seat belt law violation alone. Secondary: Motorist can be cited for seat belt law violation only if stopped for other traffic offense.

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Ciprofloxacin-Resistant Methicillin-Resistant *Staphylococcus aureus* in New York Health Care Facilities, 1988

LAWRENCE D. BUDNICK, MD, MPH, SAM SCHAEFLER, PHD, AND THE NEW YORK MRSA STUDY GROUP*

Abstract: The emergence in 1988 of ciprofloxacin-resistant methicillin-resistant *Staphylococcus aureus* (MRSA) in New York City was studied in nine hospitals and eight nursing homes. Of the 43 hospitalized patients studied, 21 were admitted from home, while nine of the 12 nursing home patients were transferred from a hospital. Twenty-four of the 55 patients had been treated previously with ciprofloxacin, and 26 had an identifiable risk factor for a nosocomial infection. MRSA was a contributing factor in at least five of the 21 deaths. MRSA resistance to ciprofloxacin was detected within three months of the drug's commercial availability, apparently emerged independently at a number of the health care facilities, and has become widespread. If such resistance is found in a health care facility, ciprofloxacin may not be useful as a first line antibiotic. (*Am J Public Health* 1990; 80:810–813.)

Introduction

Staphylococcus aureus caused a pandemic of nosocomial infections in the 1950s.[1] Shortly after methicillin was introduced into clinical practice, methicillin-resistant *S. aureus* (MRSA) emerged as a prominent cause of nosocomial infections first in Europe and later in the United States.[2–7] Patients can introduce MRSA into a health care facility from the community and cause intra- and inter-institutional spread.[8–11] Ciprofloxacin, an oral fluoroquinolone antibiotic that became commercially available on October 22, 1987, has bacteriocidal activity *in vitro* against MRSA[12] and is reportedly useful for the treatment of infections with susceptible staphylococci of the skin and skin structures,[13] for which it is marketed, bones,[14] and the lower respiratory tract.[15] This report examines the emergence of MRSA resistant to ciprofloxacin in New York City hospitals and nursing homes in 1988.

Methods

The Bureau of Laboratories Phage Typing Unit of the New York City Department of Health is the reference laboratory in New York City for bacteriophage typing of *S. aureus* isolates voluntarily submitted by hospitals, nursing homes, and other laboratories. All facilities do not submit isolates for typing and individual facilities do not necessarily submit all their isolates. The method of phage typing has been previously described in detail.[16–18] In summary, isolates are initially tested for phage susceptibility using the international set of *S. aureus* phages (at routine or 100X routine test dilution concentration) and then are tested using an experimental set of phages (at 100X routine test dilution). In addition, isolates are tested for acid production from mannitol, for growth response on mannitol salt agar plates (Difco Laboratories), and for susceptibility to a standard panel of antibiotics.[17–19] Ciprofloxacin susceptibility has been tested since May 1987 and resistance to ciprofloxacin was defined as a minimal inhibitory concentration ≥4.0 μg/mL. Isolates that differed by ≥3 of 13 findings in their antibiogram and biochemistry profiles were categorized as different.

The Phage Typing Unit MRSA laboratory records were reviewed for all isolates submitted between May 1, 1987 and June 30, 1988 that were resistant to ciprofloxacin. Isolates from 67 patients in 14 New York City hospitals and 14 patients in nine nursing homes (seven in New York City, two in bordering counties) were identified. The infection control practitioners of these 23 facilities were requested to complete for each patient a standardized data collection form based on the Centers for Disease Control National Nosocomial Infections Surveillance System Infection Worksheet. Data were collected concerning demographic factors, medical history, surgical procedures, cultures obtained, antibiotic treatment, hospital course, and outcome. Data were also collected concerning any patient at the facility who had a culture that revealed MRSA resistant to ciprofloxacin that was not submitted to the Phage Typing Unit.

Data on 43 patients were submitted by nine (64.3 percent) of the hospitals which are located in Manhattan (five) and Brooklyn (four) [the five nonparticipating hospitals are in Manhattan (two), Queens (two), and Brooklyn (one)]. Data on 12 patients were submitted by eight (88.9 percent) of the nursing homes which are located in the Bronx (four), Manhattan (one), Queens (one), Westchester (one), and Nassau (one); (the nonparticipating nursing home is in Brooklyn). Patients with positive cultures from normally sterile body sites or who were treated with antibiotics for positive cultures were considered infected; all others were considered colonized.

The data were coded and computerized using DBase III software. Statistical methods used included chi-square tests

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for independence and Fishers exact test. Odds ratios (OR) and 95% confidence intervals (CI) were calculated.

Results

The Patients

The first positive culture for ciprofloxacin-resistant MRSA in New York City was obtained from a patient in late January 1988. Prior to that time, ciprofloxacin-resistant MRSA had not been reported in New York City. Of the 55 patients on whom we have reports, 30 were males, 25 were females, and the age range was 23 to 96 years, with a mean of 67.5 years and a median of 73 years. The mean age of the nursing home patients (71.8 years) was five years greater than that of the hospital patients. The principal diagnoses at admission were cerebrovascular accident (16.4 percent), pneumonia (12.7 percent), atherosclerotic heart disease (9.1 percent), sepsis (9.1 percent), and gangrene/cellulitis (7.3 percent), with other diagnoses accounting for two or fewer patients each. Two patients had the acquired immunodeficiency syndrome (AIDS). In addition, seven patients had insulin dependent diabetes mellitus and four had a reported history of intravenous drug use; none were on hemodialysis.

Almost half of the hospitalized patients were admitted from home, while three-fourths of the nursing home patients were transferred from a hospital (Table 1). The median time from admission to the first positive culture for MRSA was 25 days for 38 of the hospital patients and 340 days for the nursing home patients. Of the nine hospital patients who had a positive culture within one week of admission, five were admitted from home, including the two patients with a positive culture on the day of admission, three were admitted from a nursing home, and one was transferred from another hospital. The one nursing home patient who had a positive culture within one week of admission had been transferred from a hospital. At least 26 (47.3 percent) of the patients had at least one prior culture negative for ciprofloxacin-resistant MRSA and 14 patients had previous treatment with systemic antibiotics.

The culture source of each patient's initial MRSA isolate is listed in Table 2. All eight bacteremic patients and 20 of the 22 patients with positive pulmonary or sputum cultures were hospitalized. Of the 22 patients with a sputum or pulmonary source, 13 had definite radiologic evidence of pneumonia and three had possible radiologic evidence of pneumonia. Almost half of the patients had an identifiable risk factor for a nosocomial infection. Of the 21 deaths, MRSA was estimated by the clinical staff to have contributed to the deaths of at least five.

At least one antibiotic had been used for 52 of the 55 patients and 24 were treated with ciprofloxacin, 19 of whom began taking ciprofloxacin at least two days before the first MRSA positive culture was obtained. None of eight patients with a culture positive for MRSA within one week of admission received ciprofloxacin (data were unavailable for one patient). Twenty-two additional patients who did not receive ciprofloxacin were infected or colonized with ciprofloxacin-resistant MRSA. Ciprofloxacin use among the nursing home patients (75.0 percent) was greater than its use among the hospital patients (OR = 5.6, 95% CI = 1.1, 27.0).

TABLE 1—Places from Which Patients Were Admitted, by Patient's Facility at Time of Culture

Admitted From	Facility at Culture: Hospital, n	Facility at Culture: Nursing Home, n	Total, n
Home	21	1	22
Hospital	6	9	15
Nursing Home	12	1	13
Other/Unknown	4	1	5
Total	43	12	55

TABLE 2—Initial Source of Ciprofloxacin-resistant Methicillin-resistant *Staphylococcus aureus*, Site-Specific Risk Factors, and Deaths

Methicillin-resistant *Staphylococcus aureus* Source	Number	Nosocomial Risk Factor	Number with Risk Factor	Number Who Died
Blood	8	Central venous catheter	7	3
Pulmonary/sputum	22	Ventilator	8	15
Urine	8	In-dwelling catheter	5	2
Wound	14	Previous incision	4	1
Other	3	Invasive procedure	2	0
Total	55	Any noted above	26	21

The MRSA Isolates

The phage susceptibility of the ciprofloxacin-resistant MRSA are listed by facility in Table 3. The distribution of phage types differed between the hospitals and the nursing homes, with 57 percent of the isolates from the hospital patients that were typed susceptible to experimental phage 88 and 64 percent of the isolates from the nursing home patients that were typed susceptible to phages of International Group III. When facilities were grouped by geographic location, no singular or unique pattern was identified.

Ten facilities identified more than one patient with ciprofloxacin-resistant MRSA. The antibiograms, biochemistry tests, and phage typing of the isolates from nine of these facilities are available. Table 4 lists the number of different phage types and antibiogram/biochemistry patterns by facility. Three hospitals and one nursing home identified isolates with multiple phage groups and multiple antibiogram/biochemistry patterns. In one hospital (M-2) there was a close association between the phage groups and the antibiogram/biochemistry patterns, in two of the facilities (hospital K-1 and nursing home B-1) there was a variable association, and in one hospital (M-4) there was no association.

Discussion

In this investigation, we document the emergence of ciprofloxacin-resistant MRSA in New York City health care facilities, which was first noted in 1988.[19] The data are incomplete and represent a minimal account, however, because not all facilities report MRSA and submit all isolates to the NYCDOH and not all facilities that did submit isolates participated in this survey. In addition, this retrospective analysis was limited to MRSA isolates and not other *S. aureus* isolates. Finally, the patients may have been misclassified as being infected or colonized, because patients could have been treated with antibiotics inappropriately and because not all culture data were provided on each patient. Nevertheless, several important conclusions can be reached.

The ciprofloxacin-resistant strains appear to have emerged independently at a number of the health care facilities. This was also seen in the broader survey of reported quinolone-resistant *S. aureus* isolates in New York City.[19]

TABLE 3—Phage Susceptibility of Ciprofloxacin-resistant Methicillin-resistant *Staphylococcus aureus* by Facility

Facility*	International Group 2	International Group 3	Experimental Phage 88	Other Experimental[a]	Not Typable	Not Typed	Total
Hospitals							
M-1		2				9	11
M-2		5	1		1	1	8
M-3			8				8
M-4			2		1		3
M-5			1				1
K-1			2	1	3		6
K-2						3	3
K-3			2				2
K-4			1				1
Subtotal		7	17	1	5	13	43
Nursing Homes							
B-1	1	1	1				3
B-2		1					1
B-3		1					1
B-4		1					1
X-1			1	1			2
X-2		1				1	2
X-3		1					1
X-4		1					1
Subtotal	1	7	2	1		1	12
Total	1	14	19	2	5	14	55

*Facilities: M = Manhattan, K = Brooklyn, B = Bronx, X = Other locations for nursing homes (1 each in Manhattan, Nassau, Queens, Westchester)

[a]Experimental phages excluding phage 88

The distribution of ciprofloxacin-resistant MRSA detected may represent either actual differences between hospitals and nursing homes, differences in community flora, or selective underreporting by facilities in other New York City boroughs. If the difference between the facilities is real, it is possible that MRSA isolates with this resistance pattern are transferred only between similar facilities locally, and we did note a few patients who may have been the vectors of transmission between facilities. Although nursing home patients have been prospectively implicated as a source of MRSA in Chicago hospitals,[8] half of the hospital patients we studied came from their homes, and colonized nursing home residents did not appear to be the major source of ciprofloxacin-resistant MRSA in the hospitals. Similarly, although the majority of the nursing home patients came from hospitals, many had a prior culture negative for ciprofloxacin-resistant MRSA and this admission pattern may be the usual one for nursing home residents.

TABLE 4—Phage Group, Antibiogram, and Biochemistry Patterns of Ciprofloxacin-resistant Methicillin-resistant *Staphylococcus aureus* by Facility

Facility*	Number of Isolates	Number of Different Phage Groups	Number of Different Antibiogram/Biochemistry Patterns
M-1	11	1	3
M-2	8	3	3
M-3	8	1	1
K-1	6	3	3
B-1	3	3	2
M-4	3	2	2
X-1	2	2	1
K-3	2	1	2
X-2	2	1	1

*See footnote Table 3.

Ciprofloxacin-resistant MRSA, which contributed to almost one-fourth of the deaths and which resulted in an outbreak that eventually affected over 40 patients in one of the hospitals (Michael S. Simbercoff, MD: personal communication) appears as virulent as other *S. aureus* strains.[20] In this investigation, the majority of patients had MRSA resistant to ciprofloxacin without prior ciprofloxacin treatment. Therefore, ciprofloxacin may not be useful as a first line antibiotic, especially in facilities where ciprofloxacin-resistant MRSA is already prevalent.[21] Although data indicating that the prevention of resistance to ciprofloxacin by combination chemotherapy are few, ciprofloxacin may best be used in combination with other agents.[22–25]

We recommend that isolates of *S. aureus* be tested for susceptibility to ciprofloxacin in all health care facilities where ciprofloxacin is used. This increased testing provides clinicians with additional information necessary to prescribe appropriate treatment and allows additional surveillance opportunities to further define the spread of ciprofloxacin-resistant MRSA. In New York City, the Phage Typing Unit received 717 ciprofloxacin-resistant *S. aureus* isolates from 31 hospitals and 28 nursing homes from July 1 to December 31, 1988. 23 percent of the 3134 MRSA isolates submitted for phage typing (New York City Department of Health: unpublished data).

MRSA, in general, is becoming more problematic to health care facilities in New York. In a 1987 New York State Department of Health statewide survey, 40 (36 percent) of 111 acute care hospitals reported that they were adversely affected by MRSA. Furthermore, 23 outbreaks of nosocomial MRSA were reported in New York City health care facilities between July 1, 1987 and December 31, 1988 (New York State Department of Health: unpublished data). In response, the Department is promoting policies for health care facilities, based in part on those of the Los Angeles County Department of Health[26] and the Centers for Disease

Control[27,28] which emphasize the importance of traditional infection control measures, i.e., surveillance, handwashing, barrier precautions, epidemiologic assessment and appropriate cultures, individualized treatment, and intra- and inter-facility communication. Additional prevention and control measures have been described elsewhere[29] and have been discussed in a recent review.[30] Health care facilities may also find the surveillance of certain prescribed antibiotics and their control useful.[30–33] Finally, health care facilities need to work together to control the spread of MRSA and to provide the appropriate level of care for colonized or infected patients.

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Access to Obstetric Care in Rural Areas: Effect on Birth Outcomes

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Abstract: Hospital discharge data from 33 rural hospital service areas in Washington State were categorized by the extent to which patients left their local communities for obstetrical services. Women from communities with relatively few obstetrical providers in proportion to number of births were less likely to deliver in their local community hospital than women in rural communities with greater numbers of physicians practicing obstetrics in proportion to number of births. Women from these high-outflow communities had a greater proportion of complicated deliveries, higher rates of prematurity, and higher costs of neonatal care than women from communities where most patients delivered in the local hospital. (*Am J Public Health* 1990; 80:814–818.)

Introduction

A fundamental precept of modern obstetrics is that adequate prenatal care leads to improved perinatal outcomes for mothers and infants particularly for high risk populations.[1–3] For pregnant women to obtain timely and appropriate perinatal care, they must establish relationships with individual providers: obstetricians, family physicians, midwives, or public health nurses.

In the rural United States—accounting for 23 percent of the nation's population[5]—two-thirds of the obstetrical providers are family or general practitioners.[6] There has been a precipitous decline over the past several years in the number and proportion of family physicians offering obstetrical services in the United States. In 1988, only 29 percent of the members of the American Academy of Family Physicians were offering routine obstetrical care, down from an estimated 40 percent just two years previously.[7,8] Although approximately 43 percent of *rural* family physicians continued to offer obstetrics, this constitutes a 23 percent decline in their participation since 1980.[7,9] The Institute of Medicine reports that thousands of rural physicians stopped offering obstetrical care, leaving hundreds of rural counties without any local source of obstetrical services.[6] Although the cause of physicians eliminating obstetrical care from their practices is multifactorial, the cost of liability insurance and the fear of suits appear to play a major role in their decisions.[6,8–13]

Even in communities with adequate obstetrical care, a certain proportion of women either choose to leave these communities for obstetrical care, or are referred to different physicians or facilities because of specific complications of pregnancy. However, in towns with little or no obstetrical capacity, most women must travel to secure basic prenatal care as well as delivery. As a consequence it becomes less likely that those women will obtain adequate prenatal care.[14] Delays in care of early labor complications may also result.

This study investigates the extent to which local availability of obstetrics is related to perinatal outcomes. We seek to answer the following questions:

- What are the characteristics of rural communities in which the majority of women deliver at a facility other than their local hospital (outflow)?
- Using outflow as a proxy for access to care, is there any difference in the outcome or cost of care for women living in communities with diminished obstetrical access as compared to women who have ready access to local obstetrical care?

Methods

Population Studied

The study was based on all deliveries of women whose primary residence was in a rural area of Washington State and who gave birth during calendar year 1986. The following definitions were used:

- *Rural hospitals* were defined as all non-federal, short-stay, acute care, inpatient facilities of fewer than 50 beds and located more than 15 miles from a city of 30,000 population or greater. Thirty-three of Washington's 90 hospitals met these criteria.
- A *rural area* was defined as the medical service catchment area served by these hospitals.
- A *catchment area* was the aggregate of all zip code areas whose center was closer to a specific rural hospital by public road than to any other hospital facility.
- Distances were based on figures supplied by the Washington State Department of Transportation.

Stratification of Rural Areas by Location of Delivery

A file of all hospital discharges from non-federal, short-stay hospitals in the State of Washington includes data on the place of residence of the patient, hospital of discharge, the DRG (diagnosis related group), and hospital charges. Maternal residence was used to identify all patients living within the 33 rural medical service catchment areas. By comparing the place of residence with the location of the hospital of delivery, we could determine what proportion of all obstetrical deliveries occurred in facilities outside a woman's local hospital catchment area.

The 33 rural areas were stratified into three groups on the basis of these determinations. Areas in which more than two-thirds of deliveries occurred in the local hospital were designated as "low-outflow" communities. "High-outflow" communities were those in which fewer than one-third of deliveries to local women occurred in the local hospital.

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"Medium-outflow" communities were those between these two extremes.

Availability of Obstetrical Services

The availability of obstetrical services in each of the 33 communities was determined through telephone surveys of hospital administrators and directors of nursing. The response rate was 100 percent. Information was obtained as to the number and specialty of all physicians providing obstetrical services in each hospital during the study period (1986), as well as in 1985 and early in 1988. A physician was considered as providing obstetrical services if, in the opinion of the administrator and directors of nursing, the physician was routinely doing deliveries in the hospital; this excluded physicians who delivered only on an emergency basis.

Determining Pregnancy Outcome and Costs

DRGs were used as proxies for obstetrical outcome (Appendix). Maternal complications were defined as all discharges with DRGs 370 or 372. These DRGs are used to designate deliveries—both cesarean section and vaginal—associated with major intrapartum complications, as well as other conditions such as pregnancy-induced hypertension, diabetes, and anemia. The balance of the deliveries were assumed to be uncomplicated for the purposes of this study and included DRG 371 (uncomplicated C-section), 373 (normal vaginal delivery), and 374 and 375 (vaginal delivery with an operating room procedure, such as sterilization). Premature births were defined as babies discharged with DRG codes 386, 387, or 388 all of which referred to premature delivery. DRGs 389, 390, and 391 were defined as full term births.

Hospital charges for neonatal care, as well as source of payment, were also evaluated from the hospital discharge abstracts.

Statistical Analysis

Differences in outcomes between outflow groups were evaluated using chi-square for trend for each outcome measure. Hospital charges for newborns were evaluated by comparing the means of high and low outflow communities, and the confidence interval of the difference between them. In addition, for the same groups, the proportion of newborns with charges greater than $5,000 were compared using the associated 95 percent confidence interval of the difference between those proportions.

Results

Characteristics of Study Communities

Approximately 350,000 people, 8 percent of the population of Washington State, live in the 33 rural medical service areas defined by this study. The 33 hospitals serving these areas represent 37 percent of the 90 acute, short-term, general hospitals in Washington State, but account for only 8 percent of the total licensed acute care beds. These service areas are dispersed throughout the state; 23 of Washington's 39 counties encompass one or more of the study areas. The 5,554 births which occurred to residents of these areas in 1986 represent 8.1 percent of all births to residents of the state during that year. There were 108 physicians practicing obstetrics in the 33 communities during 1986, 93 percent of whom were family or general practitioners; there were eight obstetricians practicing in two low-outflow communities and two medium-outflow communities.

As can be seen from Table 1, both the rural communities and the hospitals that serve them are quite small, with the average service area encompassing 10,592 people and the average hospital having 32 beds. High-outflow communities—those in which more than two-third of all births to local residents did not occur in the local hospital—were smaller than communities where larger proportions of pregnant women delivered in their local community hospital. These high-outflow communities were also somewhat closer to other more sophisticated perinatal facilities, although even for this group the average distance to a Level II facility was 41 miles. Conventional measures of socioeconomic status, such as unemployment rate and the proportion of obstetrical patients enrolled in Medicaid, did not meaningfully differ among the three groups of communities.

The most striking difference between the communities in the outflow groups was the local availability of obstetrical care. By the end of the study period in 1986, only eight of the 13 high-outflow community hospitals still offered routine obstetrical services, with four of the closures occurring since mid-1984. Additionally, three of the eight hospitals which offered obstetrical services in 1986 suspended these services

TABLE 1—Characteristics of Study Communities Stratified by Differential Obstetrical Outflow, Washington State, 1986

Characteristics	Low Outflow (<33%) N = 8	Medium Outflow (33–67%) N = 12	High Outflow (>67%) N = 13	All Communities N = 33
Total number of hospital births to residents of service areas	1,155	2,781	1,618	5,554
Percent of births occurring outside community	19.9	48.8	80	52
Mean miles to Level II nursery	79	63	41	58
Mean beds in local hospital	37	33	27	32
Mean population of hospital service area	11,029	12,318	8,731	10,592
Mean percent of obstetrical patients enrolled in Medicaid	31.3	26	27.4	28.8
Mean percent county unemployment rate (1985)	12.6	11.8	11.1	11.7
Percent of births to women under 18 or over 35 (1986)	9.4	11.0	8.8	9.7
Infant Mortality Rates/1000 births (County rates 1980–83)	11.6	11.8	10.5	11.2

in either 1987 or 1988. The reason given in essentially all of these cases was the decision of local physicians to discontinue offering obstetrics. All of the low and medium-outflow communities continued to offer obstetrics. Figure 1 shows that high-outflow communities had relatively fewer obstetrically active physicians in the year before the study, and that this disparity has become more pronounced during the study year and in the succeeding year. By contrast, there was no significant attrition in obstetric availability in the comparison communities.

Obstetrical Outcomes

Obstetrical outcomes differed systematically across the three groups of communities. As Figure 2 demonstrates, there is a strong association between the proportion of deliveries that occur outside of the community and the rate of complications associated with childbirth. Women living in high-outflow communities were 34 percent more likely to experience birth-associated complications or comorbidity than women from medium-outflow communities, and 67 percent more likely than women from low-outflow communities.

Children of women from high-outflow communities have higher rates of prematurity, a trend significant at the .001 level. Neonatal length of stay—a measure presumably correlated with neonatal outcome—also shows a significant trend across community type.

Access to Care and Perinatal Costs

Table 2 illustrates the difference in hospital charges for newborns used as a proxy for cost for neonatal care across the three community groups. Newborn patients from high-outflow communities have dramatically higher average charges than their counterparts in better served communities.

Although the differences are impressive without regard to insurance type, it appears these differences are mainly the result of patients enrolled in the Medicaid program. It should be noted, however, that infants of lower income women with adverse birth outcomes generating high hospital charges are more likely to be encouraged, and even assisted in enrolling in Medicaid.

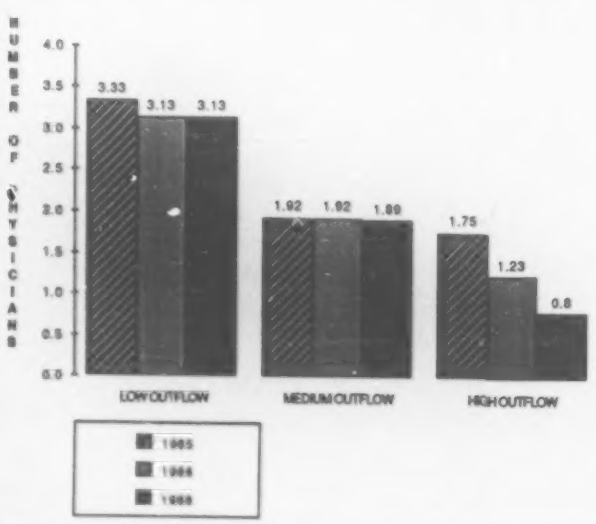

FIGURE 1—Physicians Practicing Obstetrics per 100 Births to Local Women by Community Outflow by Selected Year

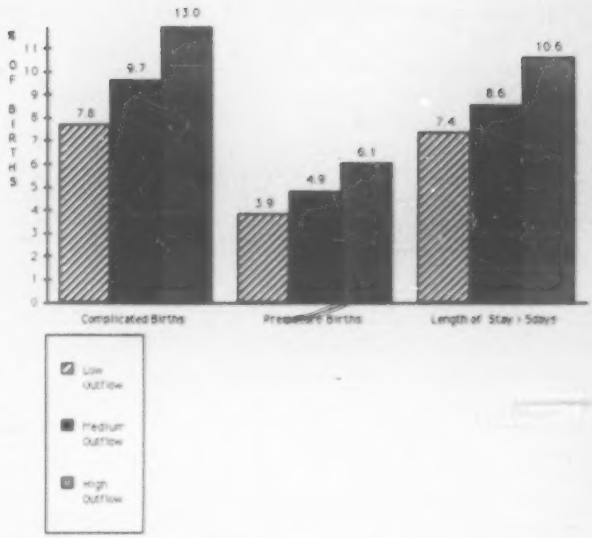

FIGURE 2—Percent of Adverse Birth Outcomes to Rural Washington State Residents by Community Outflow

TABLE 2—Newborn Hospital Charges to Rural Washington Residents Stratified by Community Outflow for Obstetrical Care and Payor Type

	Mean Charges		
	High Outflow N = 1587	Low Outflow N = 1210	Difference (95% CI)
All payors	$2,103	$1,046	$1057 (−$125, $2,239)
Medicaid	$4,627	$1,014	$3613 (−$422, $7,648)
Non-Medicaid	$1,128	$1,061	$67 (−$368, $502)
	Proportion of Newborns with Charges > $5,000		
	High Outflow N = 1587	Low Outflow N = 1210	Difference (95% CI)
All payors	3.6	1.7	1.9 (0.66, 3.14)
Medicaid	6.3	2.1	4.2 (0.54, 7.9)
Non-Medicaid	2.7	1.4	1.3 (−.03, 2.57)

Although a few outliers can generate enormous charges, dramatically increasing the mean, and they undoubtedly had an effect here, newborns with higher charges clearly occurred more frequently in high outflow community populations. As shown in Table 2, there are three times as many babies on Medicaid with charges exceeding $5,000 in the high-outflow communities compared to the low-outflow communities—a measure less affected than average charges by the influence of extreme outliers.

Discussion

The declining proportion of practitioners offering obstetrical services has had a disproportionate impact on rural areas. First, rural areas have fewer physicians per capita than urban regions and thus are more susceptible to changes in the spectrum of clinical services offered by those physicians who

do practice in rural America. Second, a larger proportion of deliveries in rural areas have been provided by general and family physicians, a group that has experienced a high rate of attrition from obstetrics.[6,7–13,15]

Out data demonstrate that women living in rural Washington state communities with little or no obstetrical care available locally tend to deliver in hospitals outside the community. These women are more likely to have complicated labor and premature deliveries, and their infants are more likely to have longer and more expensive hospital stays than the children of their rural counterparts who deliver in local facilities communities with greater access to care.

The design of this study does not permit us to conclude that there is a causal relation between impaired access to rural obstetrical care and adverse perinatal outcome, although this appears to be a tenable hypothesis. There are several possible mechanisms which could account for a causal relation between access to care and outcome, however. First, women living in communities without obstetrical services must travel to obtain routine prenatal care, a barrier associated with poorer prenatal compliance.[14] Women on marginal incomes or women without adequate transportation would be likely to delay or forego prenatal care, and might have more difficulty getting to all their prenatal visits. Even if these patients are able to arrange for transportation to other communities, they may encounter difficulties in obtaining obstetrical care in a state like Washington where most physicians limit the number of pregnant Medicaid patients for whom they will provide care.[10]

Second, obtaining obstetrical care and delivering outside one's local community may in itself constitute a risk factor for adverse outcome, even if obstetrical care has been arranged. Patients from remote, rural communities may have difficulty adhering to prenatal protocols or treatment regimens prescribed by physicians in distant communities. There may be significant delays in presentation to the hospital after the onset of labor. And the increased stress—physiological and psychological—associated with travel and parturition in unfamiliar settings may interfere with the normal process of labor.

Nevertheless, there are alternative explanations for the patterns observed here, the first being the possibility that inappropriate care was delivered by local physicians in high outflow communities. This is an unlikely cause for the results in this study for several reasons. First, there was little care available in these communities; five were without any routine obstetrical services. Second, for the 20 percent of the women from high-outflow communities who did deliver in a local hospital, outcomes were actually superior to local deliveries in medium and low-outflow facilities. Finally, of the non-local complicated births which occurred to women from high outflow communities, less than 5 percent came to those non-local hospitals as the result of a transfer.

A second explanation is that because the data are derived from hospital discharge abstracts, we lack precise information about the prenatal, intrapartum, and neonatal course of the patients in the study. DRGs and costs are used as proxies for outcome, and it is possible that there is a systematic bias in which larger urban hospitals are more likely to intervene medically during the intrapartum period, assign DRG codes denoting increased medical intensity, and keep neonates longer in their nurseries than smaller rural hospitals. Even if such a bias is the cause of the apparent differences in biological outcomes, the increased charges and lengths of stay associated with deliveries outside rural communities are real.

A third alternative explanation for the observed disparities is that women from communities with high-outflow are not comparable to the women living in medium- and low-outflow communities. Perhaps the 13 communities in this group have populations of women with higher risks for adverse perinatal outcomes. However, we found no indications that these high-outflow communities differ systematically from those with lower outflow for obstetrical care. Although the high-outflow communities are slightly smaller than the average rural Washington town or medical service area, the situation regarding previous county infant mortality rates (80–83), county unemployment, and the percent of obstetrical patients on Medicaid from these communities is similar to that of communities with less outflow and better outcomes.

A fourth explanation for these results may be that physicians in communities with increased rates of adverse outcomes transfer those high-risk patients to outside facilities prior to labor, thereby becoming a high-outflow community. However, if one notes that high-outflow communities were those in which more than two-thirds of the patients delivered non-locally, and that these communities had only 13 percent complicated births, it is clear that the transfer of an increased number of complicated patients would not change an otherwise low-outflow community into a high-outflow community.

Despite the limitations of this study, the data suggest that recent declines in the availability of obstetrical care in rural areas are associated with poorer perinatal outcomes. From the experience in Washington State it appears that the pivotal event is the decision by rural family physicians to discontinue providing obstetrical services. In many cases this leads to a substantial curtailment or total discontinuation of obstetrical services in the local hospital, causing women who may have previously delivered in their local hospital to travel to other communities for their obstetric care.

Although in this study loss of local services and associated higher rates of adverse outcomes were observed only in a handful of relatively small communities, this may be the leading edge of a more pervasive phenomenon. Although the problem is more graphic and easily demonstrated in rural populations, impaired obstetrical access may have the same social and biological consequences in urban settings.[16]

This study suggests that programs that maintain local availability of obstetrical care may improve perinatal outcomes in a cost effective fashion, since infants from high-outflow rural communities generated hospital charges twice as high as infants from low- and medium-outflow communities. The excess charges were more than $1.5 million during 1986 alone, most of it paid for by public subsidies through the Medicaid program. This does not include additional costs in the post-hospital period which have been shown to be higher for Medicaid infants whose mothers received inadequate prenatal care.[17]

In conclusion, this study demonstrates an association between diminished rural access to obstetrical care and perinatal outcomes for women who travel outside their local communities for that care. If a causal relationship exists, society would benefit both medically and economically from providing a solution to the problems which stem from a diminishing number of obstetrical providers.

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APPENDIX

Codification of Diagnosis Related Groups

DRG	Abbreviated Title
Coded as Complicated Birth	
370	Cesarean section with complications or comorbidity
372	Vaginal delivery with complicating diagnosis
Coded as Uncomplicated Birth	
371	Cesarean section without complications or comorbidity
373	Vaginal delivery without complicating diagnoses
374	Vaginal delivery with sterilization and/or D & C
375	Vaginal delivery with operating room procedure except sterilization or D & C
Coded as Premature Neonate	
386	Extremely premature neonate
387	Premature with major complications
388	Premature without major complications
Coded as Term Neonates	
389	Full term neonate with major problems
390	Neonates with other problems
391	Normal newborn

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'Late Breaker' Session on Injury Control Invites Abstracts

The Injury Control and Emergency Health Services special primary interest group of the American Public Health Association has announced it will again feature a "late breaker" session during the APHA upcoming 118th annual meeting in New York City. The session will be held on Tuesday, October 2, 8:30 am–10:00 am, and will feature work completed within the last few months--after the deadline for consideration in the regular symposia of the APHA annual meeting.

Abstracts of 250 words or less will be accepted by the Injury Control SPIG until August 15, 1990. Please send the abstract, title of the paper, authors' name, address and telephone number to: Richard Waxweiler, Division of Injury Control, Centers for Disease Control, Mail Stop F-36, Atlanta, GA 30333. Tel: 404/488-4695.

The Relation of Obstetrical Volume and Nursery Level to Perinatal Mortality

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Abstract: We investigated the relation of hospital delivery volume and nursery technology level to perinatal outcome in 226,164 White singleton births in Washington State, 1980–83. Level III facilities (neonatal intensive care unit) were defined by the state licensing commission. We defined the Level II (intermediate) and Level I (normal newborn) facilities using published criteria. Infants under 2000 gm born in Level III facilities had half the risk of perinatal death compared to those born in a Level I or II facility. No significant improvement was noted among level or volume groupings for normal birthweight infants. A loglinear regression model of hospital perinatal death rates showed that when birthweight and maternal risk were controlled, obstetrical volume added minimal explanatory power to level of nursery care. (*Am J Public Health* 1990; 80:819–823.)

Introduction

Many experts believe that the decreasing rates of neonatal and perinatal death observed in the United States over the past several decades are due to more effective technological interventions coupled to regionalization, a process which optimizes access to that technology.[1,2] Regionalization is based on a tiered structure of facilities linked by referral patterns whereby high-risk mothers and newborns are triaged to the central facilities with increased technology and staff. Most investigators have noted a twofold improvement in outcome for infants under 2500 gm when born in a high technology facility (Level III) compared to birth in a low technology facility (Level I).[3–7]

A close correlate of high technology is volume, since a high volume of patients is required to justify the expense of purchase, maintenance, and staffing of high technology. This high volume is also thought to lead to improved skill in the use of the technology. Work by Luft and others has shown a positive relation between the quality of care and hospital volume of some surgical procedures and medical diagnoses.[8–10] Increased obstetrical volume also appears to impart some benefit in obstetrical outcome, with authors noting the death rates for infants delivered in facilities with over 2,000 deliveries/year to be half of the rate of those born in facilities delivering less than 500 per year, particularly for the low birthweight infants.[3,11,12]

The relation among volume, technology level, and outcomes is unclear. Most of the studies of obstetrical volume are confounded by the presence of high technology. For example, when Williams evaluated the relation of hospital characteristics to mortality, obstetrical volume became insignificant when variables that normally accompany higher technology, such as higher number of specialists and C-sections, were added to the regression.[12]

This study was undertaken to establish more clearly the relation of hospital obstetrical volume and neonatal nursery technology to outcome by using the perinatal mortality rate as the outcome measure of quality. Also, we desired to explore the relation between obstetrical volume and the nursery level of care.

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Methods

Perinatal Data

Washington State birth-death linked records and fetal death records were obtained for 1980–83. The analysis was restricted to White singleton births because of the very small number of other racial births and their marked association with high volume hospitals. All White singleton births over 1000 gm which occurred in hospitals that routinely offer obstetrical care in Washington State were included. The hospital of birth (not of death) was identified from the record, and births were grouped by hospital. Perinatal death was defined according to the World Health Organization, i.e., all fetal deaths over 1000 gm and all live births over 1000 gm which died within one week of birth (early neonatal deaths).[13] Births under 1000 gm and those without a recorded birthweight were excluded from the study. The birthweight was stratified by 250 gram intervals for analysis, then grouped by 500 grams for presentation in the tables.

The fetal file required special handling because no birthweight was recorded for 450 cases (23 percent) of the fetal death file. We obtained 91 of the missing birthweights from a chart review being conducted by the Washington State Department of Social and Health Services, Division of Health, Epidemiology Section.[14] The last menstrual period was recorded on 223 of the records sans a birthweight, enabling us to calculate a gestational age. We elected to impute birthweights based upon this calculated gestational age, using the mean birthweight per gestational week from the 1981 live birth file as a standard. There were 171 fetal deaths without a birthweight or gestational age recorded. (Further details can be obtained from the first author.)

We controlled for maternal risk factors recorded in the certificate which might influence the choice of hospital.[15,16] A high-risk status was declared if any of the following were noted on the certificate: all forms of diabetes, hypertension, pyelonephritis, pregnancy-induced hypertension, acute urinary tract infection, surgery, tumor, epilepsy, prior fetal death, pre-eclampsia, eclampsia, Rh-sensitization, abruptio placenta, placenta previa, or "other medical problems."

Hospital Characteristics

The obstetrical volume per year was calculated as the average of all deliveries performed in that facility over the four study years, 1980–83.

All six Level III facilities currently recognized and licensed by the state as Level III were assigned Level III status. We assigned Level II status using published guidelines

(see Appendix).[17] The remainder of the facilities were considered Level I facilities.

Data Analysis

Simple descriptive analyses were used to examine the relation between variables and to calculate the relative risks of death. Confidence intervals were developed using the Mantel-Haenszel Chi-square statistic test-based method.[18] A log-linear model was created, using the Generalized Linear Model (GLIM) statistical package. The dependent variable was the likelihood of death in each hospital and the independent variables were obstetrical volume, nursery level, maternal risk status, and birthweight. The model additions were tested for significance using the deviance based method, where the model is compared to the nested model by comparing the difference in scaled deviance and difference in degrees of freedom in a Chi-square contingency table.[19]

Results

Data Description

Washington State Department of Vital Statistics recorded 280,801 live births, 1,377 early neonatal deaths, and 2,046 fetal deaths for the study years 1980–83. We excluded live births and fetal deaths that were delivered in non-obstetrical hospital settings (6%, 6% and 6%), multiple gestations (i.e., twins) (2%, 11% and 9%), and of non-White race (12%, 12% and 17%). White singletons born in an obstetrical hospital that had a birthweight under 1000 gm (live births (.3%), neonatal deaths (41%) and fetal deaths (32%)) or were missing a recorded birthweight (live birth (.4%), neonatal deaths (4%), and fetal deaths (4%)) were excluded from analysis. There were 225,287 live births, 495 early neonatal deaths and 877 fetal deaths of White singletons which occurred in the 90 Washington state hospitals which routinely offer obstetrical care. This made a total of 226,164 births and 1,372 perinatal deaths.

Table 1 shows the distribution of the 90 hospitals between facility level and obstetrical volume groups. A high degree of correlation is noted between level and obstetrical volume groups. The distribution of births and perinatal deaths between level and obstetrical volume along with the crude perinatal mortality rate is shown in Table 2. Level III facilities had the highest crude death rate.

Stratified Analyses

The birthweight distribution was highly related to the hospital's level of technology. The distribution of births among the three levels is shown in Table 3, showing a significant shift in birthweight distributions across level. The lower the birthweight, the more likely the infant to be born in a Level III setting. While Level III hospitals deliver only 22 percent of all births, almost 40 percent of low birthweight infants (1500–2500 gm) and 67 percent of very low birthweight infants (1000–1500 gm) were born in Level III settings. The distribution of births among the five volume groups is similar to the distribution among levels, with low birthweight babies concentrated in larger volume facilities.

The birthweight-specific perinatal mortality rate by level of facility is shown in Table 4. The infant under 2000 gm was twice as likely to die if born in a Level I or II facility compared to a Level III. For the infant over 2000 gm, level of care had little influence on survival rates. The birthweight-specific perinatal mortality rate is compared across obstetrical volume groups and is similar to that across level of facility. The infant under 2000 gm had twice the mortality rate in facilities with less than 2,000 deliveries per year when compared to those born in facilities with more than 2,000 deliveries per year, while little difference existed between obstetrical volume groups for infants over 2000 gm.

TABLE 1—Distribution of Hospitals Among Nursery Levels and Obstetrical Volume Groups

	Volume Groups (births/year)					
Nursery Type	<200	201–500	501–1000	1001–2000	>2000	Total
Level I (normal)	31	22	13	7	0	73
Level II (intermediate)	0	0	1	5	5	11
Level III (intensive care)	0	0	0	0	6	6
Total	31	12	14	12	11	90

TABLE 2—Crude Perinatal Mortality for the Level and Volume Groups

	Deaths	Births	Crude PNMR (deaths/1000 births)
Level I (normal)			
<200 births/yr	65	8,267	7.8
200–500 births/yr	201	23,275	8.6
500–1000 births/yr	269	30,928	9.3
1000–2000 births/yr	301	40,518	7.4
Level II (intermediate)			
500–1000 births/yr	20	2,337	8.5
1000–2000 births/yr	303	27,827	11.2
>2000 births/yr	314	43,261	7.2
Level III (intensive care)			
>2000 births/yr	793	51,161	15.5

TABLE 3—Distribution of Births Among Level by Birthweight

	Level			Total	
Birthweight	I (%)	II (%)	III (%)	(%)	(n)
1001–1500 gm	17	16	67	100	924
1501–2000 gm	25	20	55	100	1,829
2001–2500 gm	39	29	32	100	6,214
2501–3000 gm	45	31	24	100	27,136
3001–3500 gm	46	32	22	100	79,229
3501–4000 gm	46	33	21	100	76,900
>4001 gm	46	34	20	100	33,932
Total	45	33	22	100	226,164

TABLE 4—Birthweight-Specific Perinatal Mortality Rates by Level (Deaths/1000 births)

	Level			
Birthweight	I	II	III	Total
1001–1500 gm	459	358	175	253
1501–2000 gm	170	150	68	110
2001–2500 gm	31	38	39	36
2501–3000 gm	7.8	7.6	11.4	8.6
3001–3500 gm	3.5	2.8	3.0	3.2
3501–4000 gm	1.8	1.5	2.3	1.8
>4001 gm	2.2	2.8	3.7	2.7

Loglinear Regression

The loglinear regression models are shown in Table 5. The nested model used for comparison to determine the significance of the new variable is noted in the second column. As expected, the main effect of birthweight (Model B) was by far the most important variable, with a smaller contribution made by the addition of risk status (Model D), nursery level (Model E) but not the addition of obstetrical volume (Model F). The two major interactions noted in the stratified analyses between "birthweight—risk" (Model H) and "birthweight—level" (Model J) made significant improvements in the model. The addition of volume (Model K) made a significant addition to the model, indicating that volume had an effect above and beyond nursery level.

The model using the interaction between level and volume (Model L) was discarded since the large number of empty volume-level combinations left only one interaction term in the model (see Table 1). The addition of interaction between "volume-risk" (Model M) was also significant, but made the model very complex.

Unfortunately, there is no method currently available to calculate confidence intervals for the various models, since the models include interaction terms. Because the data is so large, most variable additions were statistically significant. However, when parameter estimates were converted into perinatal mortality rates and compared for Model K and Model M, the clinical significance was minimal. Based on this finding, Model K was chosen as the final model.

To provide some clinical perspective of this complex model created by the loglinear analysis, we calculated the number of deaths that would have occurred if all infants had been born in one type of a facility. For infants weighing 1000–2500 gm, the Level III facilities with over 2,000 births/year appeared most advantageous, and for infants over 2500 gm the Level I facilities with 1,000–2,000 births/year appeared most advantageous. Table 6 shows the number of deaths that would have been expected if all births had occurred in either Level I facilities with 1001–2000 births/year or in Level III facilities with >2000 births/yr.

TABLE 6—Observed Deaths and Deaths Predicted from the Model If All Births Occurred in One Type of Facility

Birthweight	Observed	Level I Volume 1000–2000	Level III Volume >2000
1000–1499 gm	234	425	213
1500–1999 gm	201	316	130
2000–2499 gm	221	204	264
2500–2999 gm	234	232	306
3000–3499 gm	251	301	281
3500–3999 gm	139	143	179
4000–4500 gm	47	35	81
>4500 gm	45	45	62
Total	1372	1701	1517

Discussion

The potential limitations of this study include the quality of the vital statistics data. First, missing data, inaccuracies, and underreporting could bias the results, especially if they occurred preferentially in selected level or volume group facilities. However, the failure to record birthweight was evenly distributed among all levels and volume groups of hospitals, minimizing this concern. Other studies comparing medical records to Washington State Vital Statistics records have noted no correlation between type or size of facility and recording omissions.[14,*] Second, the elimination of cases without birthweight creates an underestimation of the true birthweight-specific perinatal death rates, while the imputation of fetal cases using standards from live births would overestimate these rates. Third, certain variables of interest that may have affected the risk classification, such as smoking status of the mother and educational attainment, were not available on these records.

Our study's main purpose was to explore the relation between obstetrical volume and nursery level of care. While obstetrical volume and nursery level are highly correlated, the loglinear analyses indicate that nursery level has a stronger influence on the perinatal mortality rate. Technology, rather than the volume effect, appears most beneficial in lowering perinatal mortality rates.

*Connell FA, Persing JP: Needs Assessment Study of High-risk Perinatal Services. Perinatal Data Base Project Assessment Report No. 5, unpublished report, May 1985.

TABLE 5—Loglinear Models Predicting Mortality Rates

	Comparison Model	Change in Scaled Deviance	Change in Degrees of Freedom	p value
Main Effects Models				
A. Grand mean	—	—	—	—
B. Birthweight	A	3523	20	<.001
C. Risk	A	290	1	<.001
D. Birthweight + Risk	B	18	1	<.001
E. Birthweight + Risk + Level	D	14	2	<.001
F. Birthweight + Risk + Level + Volume	E	0	6	>.999
Interaction Models				
G. Birthweight + Risk * Level	E	31	2	<.001
H. Birthweight * Risk	D	117	20	<.001
I. Birthweight * Risk + Level	E	116	14	<.001
J. Birthweight * Risk + Birthweight * Level + Risk * Level	I	117	40	<.001
K. " + Volume	J	141	4	<.001
L. " + Volume * Level	K	52	1	<.001
M. " + Volume * Risk	K	55	4	<.001

*Indicates the inclusion of the main effects of both variables plus the interaction term (Z * Y = Z + Y + (Z × Y).

The benefits from technology (and to a lesser extent, volume) were not enjoyed by all groups of infants. The greatest benefits were achieved by low birthweight infants (<2500 gm) with a two-fold improvement in perinatal mortality rates noted when born in Level III nurseries compared to Level I or II. This finding is similar to other evaluations of regionalized nursery systems over the past decade.[3–7] The normal birthweight infant, on the other hand, realized no net benefit from the technology-intense Level III facility. In fact, they had marginally better survival when born in the low technology setting of Level I hospitals than if born in a Level III facility, but this difference was not statistically significant. This finding is very similar to an analyses of the term, normal birthweight infant in an urban setting (New York City),[20] but different from Rosenblatt's finding in New Zealand[21] of statistically higher mortality for normal birthweight infants in Level III, high volume facilities. The lack of benefit for the normal birthweight infant in a high volume, technology intense setting could possibly be due to the failure of Level III facilities to recognize and record high-risk conditions. Because this potential ascertainment bias among nursery levels seems counter-intuitive, other explanations for the lack of benefit, and even possible harm, should be explored.

Perhaps the most interesting finding of this study was that Level II facilities did no better with infants weighing under 2000 grams than did Level I facilities. Level III facilities are approved by state authorities according to set criteria in Washington State, but no such external independent review occurs for Level II facilities. The published criteria differentiating a Level II is quite vague, specifying only that a "selected number of other services" would be offered above and beyond a normal nursery. Our Level II classification system included facilities that self-designate themselves "Level II+" and excluded hospitals offering fewer types of Level II services. Our conservative classification system should have exaggerated any possible difference between Level I and II. Prior evaluations of the influence of level have noted no improvement of Level II over Level I.[6,20,21] No evaluation to date has justified the division of nurseries into a three-level system.

The improvement in perinatal mortality rates in Level III compared to Level I facilities has been credited to the presence of neonatal intensive care units, but the specific features of the unit that ensure this improvement are unknown. Other factors which may influence survival include resuscitation capabilities (due to a practice effect), lower rates of "untreatable" conditions such as malformations, or a selection bias where less severe conditions present with sufficient time for transfer to a Level III, while more severe conditions present acutely to the Level I facility, prohibiting transfer or effective treatment.[22,23] Small area population based studies would assist in clarifying the influence of these factors.

The benefits and risks of regionalization should be evaluated from a system-wide perspective, taking into account all segments of the population. Our model simulation with all births occurring in Level I facilities predicted a marked increase in perinatal mortality, experienced especially by the low birthweight infant. This is consistent with the observations that perinatal mortality rates have improved as low birthweight infants have been centralized into Level III facilities. The model also predicts a higher number of deaths if all infants were centralized to a Level III facility, mainly from a large number of normal birthweight infants dying at a slightly higher rate. We suggest that the current system of intrauterine transfer is closer to an optimum composition, with most of the low birthweight infants being born in Level III facilities, and the majority of low-risk, term infants being born in Level I facilities. Further efforts to centralize the system, i.e., closing smaller hospitals, will be of no benefit to the normal, low-risk infant, and may even pose a risk to the normal birthweight infant.

The relation of volume to perinatal outcome is of more than academic interest to states with large rural populations such as Washington State. In 1984, over one-third of Washington State hospitals had less than 500 deliveries per year, the cutoff recommended by federal health planners in 1977 to ensure hospital quality.[24] These small hospitals now find themselves affected by the public's belief that big and better equipped hospitals offer better quality care and are bypassed by low-risk, term women who seek delivery in a Level III facility. This drain of obstetrical cases, compounded by other financial pressures and the loss of obstetrical providers, has forced many rural hospitals to eliminate their obstetrical services, resulting in access problems and even adverse outcomes for rural women.[25] Health planners and patients should be reassured with the knowledge from this and other evaluations[26] that the low volume Level I facility is an appropriate and safe location for birth for the term, low-risk woman.

In summary, the effect of hospital technology upon perinatal mortality appears to be greater than the effect of volume, but this benefit is enjoyed mainly by the low birthweight, high-risk infant. The evaluation of practices and policies which encourage transfer of obstetrical patients between facilities must consider the outcomes for all segments of the population to guide wise policy decisions and efficient resource allocation.

APPENDIX

Criteria for Level I and Level II Facilities

Criteria to differentiate Level II from Level I facilities were based upon guidelines published by the American College of Obstetrics and Gynecology and the American Academy of Pediatrics.[17] Our Level II criteria included:

- the presence of obstetric and pediatric medical staff,
- 24 hour in-house capability of resuscitating a newborn,
- maintenance of peripheral or umbilical lines, and
- short-term respiratory support.

A hospital had to meet all of the above criteria to be classified as a Level II facility. Information on nursery services and practices during 1980–83 was obtained by telephone interview with the hospital staff. Eleven hospitals met our criteria for Level II designation, and the remainder were labeled as Level I. The designation was reviewed and approved by the neonatologist who chairs the Washington State regionalization program.

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Call for Abstracts

APHA 'Late-Breaker' Epidemiology Exchange Session

The Epidemiology Section will sponsor a "Late-Breaker" Epidemiology Exchange on Wednesday, Oct. 3, 1990, 2:00–5:00 pm, at APHA's 118th Annual Meeting to be held in New York City, Sept. 30–Oct. 4. The Exchange will provide a forum for presentation of investigations, studies, methods, etc., which have been conceived, conducted, and/or concluded so recently that abstracts could not meet the deadline for submission to other Epidemiology Sessions. Papers submitted should report on work conducted during the last 6–12 months.

Abstracts should be limited to 200 words; no special form is required. Abstracts should be submitted to Robert A. Gunn, MD, Division of Field Services, Epidemiology Program Office, Building 11 South, Mailstop F-15, Centers for Disease Control, Atlanta, GA 30333—Telephone: 404/639-0336; FAX 404/639-0277, and must be received by *August 13, 1990*. Receipt of abstract will be acknowledged by postcard and acceptance will be decided by August 17, 1990.

Infectious Disease Control in a Long-term Refugee Camp: The Role of Epidemiologic Surveillance and Investigation

CHRISTOPHER J. ELIAS, MD, BRUCE H. ALEXANDER, MS, AND TAN SOKLY

Abstract: This report demonstrates the role of epidemiologic surveillance and investigation in the control of infectious diseases in a long-term refugee camp. The applications of simple epidemiologic methods in a refugee camp on the Thai-Cambodian border are described for a one-year period. The development of a Health Information Office facilitated the collection of demographic and vital statistics data, administration of a disease surveillance system, regular monitoring of hospital and outpatient discharge diagnoses, and investigation of disease outbreaks. This office also organized community health education campaigns and disease control efforts. Examples of specific disease investigations are provided to demonstrate the utility of epidemiologic surveillance in the control of infectious disease. We conclude that simple epidemiologic methods play an important role in health planning in long-term refugee camps. (*Am J Public Health* 1990: 80:824–828.)

Introduction

There are currently more than 12 million refugees throughout the world.[1] Disease and famine often accompany the movements of refugees, as health and nutritional systems are disrupted by strife. Refugee camps are generally perceived as temporary structures requiring immediate, short-term assistance. Unfortunately, persistent war in the Middle-East, Afghanistan, Sub-Saharan Africa, and Southeast Asia has created refugee camps that require long-term assistance. These camps have become permanent settlements and are now home for the majority of the world's refugees. The health care problems of long-term camps differ from those of short-term camps; the primary tasks are health maintenance, nutritional support, community development, and training of refugee health workers.[2] These tasks are often more difficult than the highly publicized crisis management necessary during the emergency phase of refugee relief operations. The permanence of these camps, combined with the difficulty in obtaining ongoing support, demands innovative planning to effectively allocate scarce health resources.

The importance of epidemiologic assessment for public health planning and resource allocation has been emphasized in emergency assistance situations.[3,4] The successful use of simple epidemiologic methods has been demonstrated in the emergency phase of the Cambodian and Ethiopian refugee crises.[5,6] Epidemiologic assessment is equally important for dealing with the health concerns of long-term camps.[7] Effective disease surveillance and outbreak investigation can help direct public health interventions. Analysis of mortality trends and nutritional status can be used to evaluate the effectiveness of assistance programs.[8,9] Knowledge of disease patterns can inform clinical practice and guide resource allocation decisions. Finally, this information may be useful in designing training programs and orienting expatriate personnel. This paper reports our experience with epidemiologic surveillance of infectious diseases in the Site 2 South encampment for Cambodian refugees on the Thai-Cambodian border.

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Methods

Following the Vietnamese invasion of Cambodia in 1979, there was a large influx of Cambodian refugees into camps in Thailand. These refugees were physically, mentally, and nutritionally exhausted after nearly a decade of war and four years of hardship under the Khmer Rouge. Several reports have described the health conditions in these early camps.[10–12] Ten years later there are still approximately 350,000 refugees in eight camps along the Thai-Cambodian border. Site 2, home to over half these refugees, is divided into North and South. Site 2 South has a population of approximately 70,000, or roughly 20 percent of the border refugee population.

The age distribution of Site 2 South is skewed toward the very young. Twenty-three percent of the population is under 5 years of age, and 43 percent is under 15.[13] The camp is severely overcrowded, with only 33 sq.m./person including public lands.[14] There is no ground water in Site 2, requiring that water be brought long distances by truck at considerable expense and resulting in a chronic shortage of water for personal hygiene. The average water ration is only 20 liters/person/day, significantly below the ideal of 100 liters/person/day recommended by the World Health Organization.[15] There is no running water, and the sewer system consists of open ditches between rows of houses. In the dry season these sewers are stagnant pools of waste. In the rainy season they quickly overflow, flooding nearby houses.

The population is totally dependent on external food supplies. The acute malnutrition rates in children under age 5 years, which were very high (>13 percent) in the acute phase of the refugee crisis, had stabilized at approximately 5 percent by 1985.[15] The prevalence of chronic malnutrition (stunting), however, remains high.[13] The diet consists primarily of rice and canned fish. The United Nations World Food Program supplies a food basket calculated to supply sufficient protein and calories. It is common, however, for camp residents to barter away some of their food aid on the black market, gaining variety at the cost of nutritional value. The persistence of significant malnutrition despite the provision of adequate protein and calories arises in part from this use of rice as the primary market exchange commodity; a phenomenon exacerbated by severe under-employment.

Humanitarian assistance for these refugees is provided through the UN Border Relief Operation (UNBRO), which supports and coordinates several voluntary agency health service programs in the camps. The American Refugee Committee has the responsibility for providing curative health services for Site 2 South; their hospital and two

outpatient clinics are the only curative health facilities serving Site 2 South. Maternal child health services, including antenatal screening, routine immunizations, and growth monitoring, are provided by a separate voluntary agency and are not covered by this report.

The hospital has dirt floors and lacks electricity or refrigeration. There are five wards: adult medicine, surgery, pediatrics, obstetrics, and a feeding center for severely malnourished children. During the year described by this report, the hospital had an average daily census of 223 patients. All wards are staffed and supervised by Cambodian health workers, nurses, midwives, and medical assistants. The expatriate staff, consisting of three nurses, two doctors, and a midwife, serve primarily as consultants and teachers. Laboratory facilities are limited to malaria smears, ova and parasite examinations, Gram and Acid Fast stains, hand-spun urinalyses, and colorimetric hemoglobin determinations. There are no chemistry, serology, or microbiologic culture facilities available. A mobile radiologic facility is available one morning a week.

A Health Information Office was developed by the American Refugee Committee to coordinate epidemiologic surveillance and investigation. This office was staffed by Cambodian and expatriate epidemiologists working closely with hospital and outpatient clinic staff, community leaders, and community service providers. The responsibilities of the epidemiologists included: collection of demographic and vital statistics data, administration of a reportable disease surveillance system, compilation of hospital and outpatient discharge diagnoses, outbreak investigation, and facilitation of clinical studies. The office also organized community health education campaigns and served as a source of information for the many programs involved in health promotion activities in Site 2.

Vital Statistics

Demographic data, obtained from the Cambodian camp administration, were derived from a family registration system maintained for the purpose of distributing relief materials. Monthly figures reflected population changes resulting from births, deaths, in-migration, and out-migration. This information was used as the basis for vital statistics. The receipt of additional relief supplies served as a powerful incentive to complete birth registration. Monthly consultations with the Buddhist monastery operating the camp's only crematorium, and the Khmer Moslem Association, served as a mechanism for death verification. Names and ages of deceased individuals were recorded, but reliable information concerning the cause of deaths occurring outside the hospital was typically unavailable. This method of death ascertainment was generally complete; however, some underreporting of neonatal deaths may have occurred because of the occasional practice of burying very young infants.

Reportable Diseases

The Health Information Office also administered a program to allow the prompt identification of diseases requiring immediate public health interventions: cholera, diphtheria, tetanus, measles, pertussis, encephalitis, dengue hemorrhagic fever, rabies, polio, typhoid, meningococcemia/meningitis, and sexually transmitted disease. The occurrence of any of these conditions was to be reported to the office within 24 hours, along with atypical presentations of more common diseases (e.g., the occurrence of malaria in a child). Contacts of incident tuberculosis and leprosy cases were investigated by a separate program. Given the extremely high prevalence of tuberculosis in Southeast Asia, a comprehensive program for the management of this disease had been developed previously, and is reported elsewhere.[16]

Hospital Discharge Data

At the time of hospital discharge a senior Cambodian medical assistant or midwife, in consultation with an expatriate health professional, recorded a discharge diagnosis. This diagnosis was entered into a log book in each ward, along with the patient's age, sex, name, and address. Precarious security conditions and emergency camp relocations have prevented the development of a permanent medical record system. Hence, the ward log books provided the only data available for retrospective analysis. Diagnostic accuracy and complete reporting were encouraged by random review of charts by clinical supervisors. Unfortunately, the volume of patients seen in the outpatient facilities (average monthly visits >18,000) prohibited complete recording of outpatient diagnoses. On Monday of each week all outpatient diagnoses were recorded and reviewed. During disease outbreaks, the office attempted to identify all outpatient cases, conducted home visits, and was involved in public educational campaigns targeted at curtailing the spread of disease.

Analysis

The results reported below were drawn from the activities of the Health Information Office, June 1987—May 1988. Population figures for January 1, 1988 were used in the computation of age-specific mortality and hospitalization rates. Analysis of the monthly population totals indicated that the population increased steadily throughout the year, and that this mid-year estimate was within 1 percent of the actual annual population average. By applying proportions from 1988 UNBRO demographic data[17] to these population figures, estimates for specific age intervals were obtained.

Results

Vital Statistics

During the study year, 3,261 births and 256 deaths were reported. The estimated crude birth rate was 46.3 per 1,000 population, the crude death rate 3.6/1,000 population. The infant mortality rate (infant deaths/1,000 live births) was estimated to be 27.3. The estimated under-5 mortality rate (deaths in children under-5/1,000 live births) was 33.4. Both the infant and under-5 mortality rates can potentially be biased by migration. During the study year there was a net in-migration of 4,024 people; however, only approximately 5 percent of these new arrivals were children under-5.

Table 1 shows the age- and sex-specific mortality rates. In general, mortality rates were low and resulted primarily from infectious diseases. Mortality rates for males were greater than those for females in the under-5 and some adult age groups. The higher mortality in male children was largely attributable to prematurity, neonatal sepsis, and acute respiratory illness.

Analysis of the cause-specific mortality rates for adults revealed the excess in male deaths to be entirely attributable to violence or malaria. There were 11 deaths from falciparum malaria in men and none in women. Epidemiologic investigation of exposure history revealed that most cases of falciparum malaria occurred in persons who had recently been outside the camp. These were mostly men involved with military or market-related activities who were exposed to the malarial parasite in Cambodia and subsequently returned to

TABLE 1—Age- and Sex-Specific Mortality Rates for Site 2 South, Thailand, June 1987 through May 1988

Age Group (years)	Population		Deaths/1,000/year*	
	Male	Female	Male	Female
<1	2075	1755	28.9 (60)	18.2 (32)
1–4	6630	6377	2.7 (15)	1.6 (10)
5–14	6953	7321	1.3 (9)	1.0 (7)
15–24	6702	4825	1.6 (11)	1.7 (8)
25–34	7992	6850	1.1 (9)	1.6 (11)
35–44	3046	3611	3.3 (10)	2.2 (8)
45–54	1290	1350	10.9 (14)	8.2 (11)
55–64	789	979	10.1 (8)	8.2 (8)
≥65	358	675	53.1 (19)	13.3 (9)
TOTAL	35835	33743	4.3 (155)	3.1 (104)

*Number of deaths in parentheses.

the camp for treatment. There was no evidence of sustained malarial transmission in the camp.

Reportable Diseases

An outbreak of dengue hemorrhagic fever in the summer of 1987 demonstrated the importance of maintaining a reportable disease surveillance system. This disease is a relatively rare, but potentially life-threatening complication of dengue. In 1987 there was a large outbreak in Thailand, with over 63,000 cases reported from 60 provinces in six months.[18] Cases began appearing in Site 2 in May 1987. Figure 1 plots the course of this outbreak in Site 2 South. The reportable disease system allowed the Health Information Office to quickly identify the first cases of the outbreak.

Prompt efforts to control the Aedes mosquito vector included pesticide spraying of houses bordering those of index cases, larval control efforts, and an extensive community education program. The latter included "dengue day" on which the primary school children were given the day off to collect larvae in competition for prizes. The combined effect of these efforts was to plateau the incidence of dengue hemorrhagic fever in the camp.

Only two cases of tetanus were reported, and no cases of pertussis, diphtheria, measles, or polio. The Health Information Office identified six isolated cases of meningococcal illness and promptly located and treated household and clinical contacts with antibiotic prophylaxis. No outbreaks of meningococcal illness occurred.

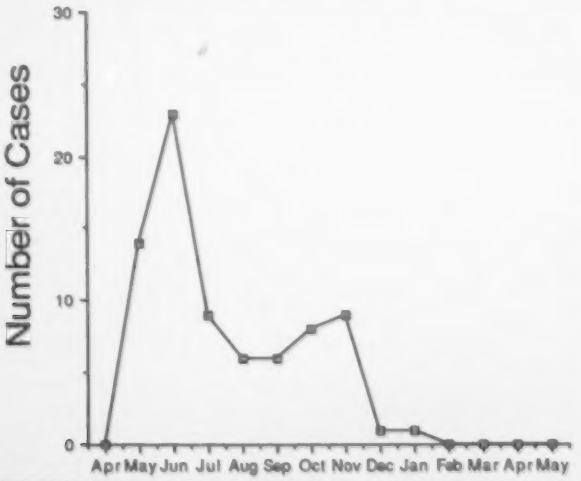

FIGURE 1—Presumed Cases of Dengue Hemorrhagic Fever in Site 2 South: 1987–88

The Health Information Office was also involved in the control of rabies, which is endemic in the dog population of Thailand. In October 1987, a dog in Site 2 was found to have rabies, confirmed by examination of the brain at the Science Division of the Thai Red Cross Society in Bangkok. Dogs that had bitten one or more persons and were stray, unwanted, or died during the period of observation were submitted for pathologic examination. Approximately 30 percent of dog brains submitted for pathologic analysis over the next several months were confirmed to have rabies. An investigation by the office revealed that few people sought medical attention for dog bites. The office began an extensive community education effort emphasizing the importance of post-exposure immunization. In October 1987, only four people were seen for treatment of dog bites. Beginning in November, the number of visits increased to approximately 30 per month and stayed at that level.

The Health Information Office also conducted a community survey to assess the dog population in the camp. It was determined that the dog population was comprised of mostly pet animals. Less than 20 percent of bites were traced to stray dogs, justifying an animal vaccination campaign. In the year following this campaign, none of the suspect dogs submitted for pathologic examination were found to have rabies.

Hospital Discharge Data

The vast majority of pediatric admissions were for acute infectious diseases: acute respiratory illness, skin infections, otitis media, and diarrheal disease (Table 2). Continuous monitoring of hospital discharge diagnoses provided definition of the seasonal variation of infectious diseases in the camp. Acute respiratory illness and diarrheal disease were most common in the rainy season (June—September). Skin infections were common throughout the year.

Monitoring of hospital discharge diagnoses also facilitated the early identification of outbreaks of these common pediatric illnesses. For example, there was an outbreak of acute respiratory illness in August and September. At the peak of the epidemic, the outpatient clinics, in Site 2 South saw 1,361 cases of pneumonia in a 17-day period. Most cases

TABLE 2—Hospital Discharge Diagnoses—June 1987 to May 1988

Diagnosis	Hospitalization Rate* Cases per 1,000 per year	Proportional** Morbidity (%)
Children (<15 years)		
Acute Respiratory Illness***	45.22 (1445)	34.3
Skin Infections	28.91 (924)	21.9
Otitis Media	9.33 (298)	7.1
Diarrheal Disease	8.58 (274)	6.5
Other	41.01 (1276)	30.3
Adults		
Prolonged Fever	94.27 (3623)	55.1
Skin Infection	22.43 (862)	13.1
Falciparum Malaria	7.49 (288)	4.4
Urinary Tract Infection	6.45 (248)	3.8
Other	40.32 (1551)	23.6

*Number of cases in parentheses.
**Percent of hospitalized cases.
***Predominantly pneumonia, bronchiolitis, and croup.

occurred in infants or young children. Clinically, these children had diffuse pulmonary rales, with occasional wheezing. Chest X-rays performed in severe cases revealed diffuse interstitial pneumonia. A viral etiology was presumed on the basis of this information. Antibiotics were used conservatively and children were carefully observed for signs of hypoxia.

A large outbreak of diarrhea was seen in December and January. Almost all cases occurred in children under two years of age, with 56 percent of the cases occurring in infants. A spot map of cases failed to reveal any specific foci of infection. Chlorine levels in the water supply were found to be adequate to prevent microbiologic contamination. Clinically, children were afebrile and had watery, non-bloody diarrhea. This was presumed to be a rotaviral outbreak and efforts focused on the education of mothers in the preparation and use of oral rehydration therapy.

The majority of adult hospital admissions were also for acute infectious diseases. The most common diagnoses were "prolonged fever," skin infections, falciparum malaria, and urinary tract infections (Table 2). The term "prolonged fever" designates a collection of infectious diseases that are not easily distinguished with the diagnostic facilities available at Site 2. It is applied when the patient has 48 hours of documented fever >38.5 C, three negative malaria smears, and no signs or symptoms suggesting localized infection. Occasionally the clinical presentation is precise enough to allow a specific diagnosis, such as scrub typhus or typhoid fever, to be made. More often the patient is treated empirically and the discharge diagnosis is simply "fever of unknown origin." Analysis of the monthly discharge diagnoses of "scrub typhus" and "fever of unknown origin," however, showed an inverse relationship, suggesting considerable variation in the convention used for assigning these diagnoses.

There was no clear seasonal variation in the incidence of prolonged fever, skin infections, or urinary tract infections. Falciparum malaria occurred principally in November through January. These are the months following the rainy season during which men leave the camp to resume fighting in Cambodia.

Discussion

The demographic indicators in Site 2 South reveal some of the anomalies of long-term refugee camps. The crude birth rate is very high and comparable to that seen in the least developed countries.[19] On the other hand, the crude death rate is extremely low; lower than even in the most developed countries. (For comparison the US crude death rate is 9.) This reflects an age distribution skewed toward the very young and possibly the ready access to effective health services. Furthermore, the residents of long-term refugee camps are survivors. The more vulnerable members of a refugee population often perish during forced migration or the emergency phase of a relief effort.

The estimated infant and under-five mortality rates are middle range and comparable to those seen in countries such as Korea and Panama.[19] They were lower than the figures reported for Thailand (infant mortality rate = 41, under-five mortality rate = 53), despite the fact that the level of health care in Site 2 is designed to be comparable to that available in surrounding Thai communities. The geographic localization of the population, the greater intensity of health services, and the absence of financial barriers make this care much more accessible, however.

The ability to accurately characterize the demographics of a refugee population is important for optimal planning of humanitarian assistance efforts. For example, the high birth rate and low death rate in Site 2 indicate that, even without new arrivals, the population will expand at over 4 percent a year. Given the already overcrowded conditions in the camp, and the ill effects of this overcrowding on physical and mental hygiene, long-range planning must include identifying more land on which to shelter this population. These data also underscore the need for effective family planning efforts as part of an overall program for maternal and child health. Analysis of mortality trends can identify vulnerable subpopulations and indicate specific diseases requiring control or surveillance.

The importance of maintaining a reportable disease system that can readily identify outbreaks of illness requiring immediate public health intervention is essential in the overcrowded environment of a refugee camp. This is well illustrated by the Health Information Office's experience with the 1987 dengue hemorrhagic fever outbreak. While it is impossible to know what the course of this outbreak would have been without intervention, the large number of cases seen in nearby urban areas of Thailand, where disease control efforts were more difficult to implement, suggests the office's activities may have averted a much larger outbreak.[18] The experience of the Health Information Office in confronting rabies in the dog population of Site 2 demonstrates the role of such an office in coordinating a community response to a serious public health threat.

The successful provision of health services in a long-term refugee camp requires a strong community orientation. The training of refugee health providers and the briefing of expatriate health professionals must emphasize the public health context within which patients develop illness. The use of simple methods of epidemiologic surveillance and investigation can greatly facilitate the development of this perspective by documenting geographic, seasonal, and epidemic patterns of disease in the community. In an environment lacking the facilities for laboratory confirmation, therapy is often administered empirically based on the clinician's "best guess" as to the likely etiology. Consequently, clinicians aware of the epidemiologic patterns of disease in the community are better equipped to provide optimal care to the individual patient. The regular monitoring of hospital and outpatient discharge diagnoses by the Health Information Office allowed prompt characterization of disease outbreaks, as well as long-term or seasonal trends.

There has been little experience with epidemiologic investigation of non-infectious diseases in the camp. Occasionally outbreaks of illness have been traced to toxic ingestion of plants or black market medications. Recently, monitoring of hospital discharge data led to the recognition of a steady increase in the number of patients with trauma. Further investigation revealed a dramatic increase in suicide and domestic violence rates, symptomatic of the growing crisis in mental health in Site 2.[20] These are late signs, however, and not adequately sensitive for surveillance activities. As experience is gained, the use of simple epidemiologic methods will be extended to surveillance and investigation of non-infectious and chronic diseases, such as hypertension and cervical cancer.

In the last several decades, the world has seen ever-growing numbers of refugees. Increasingly these refugees are

becoming "long-stayers", living in subsistence camps for indefinite time periods. As the nature of refugee environments changes, the providers of assistance must mature and learn from their experiences. Unfortunately, refugee situations are often dominated by well-intentioned, but short-term, poorly prepared personnel operating in duplicative and loosely coordinated efforts.[1,3,10] Refugee health care should not be a hobby. It requires the coordinated efforts of refugee and expatriate health planners and providers sensitive to the public health needs and resource scarcities of long-term refugee camps. Our experience demonstrates that epidemiologic surveillance and investigation have an important role in shaping the clinical and public health response to infectious disease in these camps.

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Child Vaccine Injury Claim Deadline is October 1

Health and Human Services Secretary Louis W. Sullivan has issued a reminder that the deadline is October 1, 1990 for a certain class of claimants who are eligible to use the recently authorized National Vaccine Injury Compensation Program. The program, which went into effect October 1, 1988, is intended to serve as a faster and less expensive procedure than a civil suit for resolving claims for certain vaccine-related injuries. Using this no-fault system, petitioners do not need to prove that those who manufactured or administered the vaccine were at fault. They must prove only that the vaccine is related to the injury. The federal program provides compensation to individuals who suffered injuries associated with one of the following vaccines: diphtheria and tetanus toxoids and pertussis vaccine (DTP); measles, mumps and rubella vaccine; oral polio vaccine; inactivated polio vaccine.

For injuries that occurred before October 1, 1988, claimants must file by October 1 of this year to use this program. Although claimants may not use the program if they file later, they still may pursue their claims in civil court. For injuries occurring on or after October 1, 1988, claimants must file within certain periods prescribed by the National Childhood Vaccine Injury Act of 1986 as amended.

As of April 5, 1990, some 263 petitions have been filed, 254 of which stemmed from injuries that occurred before October 1, 1988. Sixty awards totaling $31.5 million had been made as of April 5 under this program, with amounts ranging from $86,000 to $2.8 million.

For persons seeking information on the National Vaccine Injury Compensation Program, the Health Resources and Services Administration (HRSA) operates a toll-free hotline. The number is 800/338-2382.

Physician Breach of Patient Confidentiality among Individuals with Human Immunodeficiency Virus (HIV) Infection: Patterns of Decision

JUDITH A. SCHWARTZBAUM, PHD, JOHN R. WHEAT, MD, MPH, AND ROBERT W. NORTON, PHD

Abstract: To determine whether the sex, race, or sexual preference of a patient infected with immunodeficiency virus (HIV) influences a physician's decision to breach patient confidentiality, Tennessee primary care physicians were mailed a questionnaire containing a case study in which an HIV-infected patient presented a risk to a third party. Eight different descriptions of the sex, race, and sexual preference of the hypothetical patient were distributed randomly among the physicians, one description to each physician. The physicians were asked to decide whether to maintain confidentiality, notify the health department, or inform the patient's partner. Responses of 199 White male physicians were analyzed using an unconditional saturated logistic regression model. The odds ratios for these physicians saying they would send the patient's antibody status to the health department extend from 18.4 (95 percent confidence interval: 1.3, 260.1) for Black homosexual males to .5 (95 percent CI: 0, 11.5) for White homosexual females. The odds ratios for White male physicians saying they would inform the patient's partner range from 7.5 (95 percent CI: .8, 69.2) for Black heterosexual males to 1.0 (reference category) for Black homosexual females. The results suggest that when physicians decide to protect a third party by breaching an HIV-infected patient's confidentiality, their decision may be influenced in some cases by the race, sex, and sexual preference of the patient. (*Am J Public Health* 1990; 80:829–834.)

Introduction

One of the perceived ethical dilemmas associated with the acquired immunodeficiency syndrome (AIDS) epidemic is whether a physician should breach the confidentiality of a patient infected with the human immunodeficiency virus (HIV) to inform a third party at risk of infection from the patient but unaware of that risk. The American Medical Association has recommended that "if persuasion fails, the authorities should be notified; and if the authorities take no action, the physician should notify and counsel the endangered third party."[1] In this statement and similar statements[2,3] and discussions[4,5] of the policy of third-party protection, the assumption is that the demographic characteristics or sexual preference of the patient and third party are not relevant and that implementation of the policy is independent of the patient's identity. Kelly, *et al*,[6,7] have shown that AIDS patients are stigmatized by physicians. Because the policy of third-party protection requires that the physician judge the degree of danger presented by an HIV-infected patient to a third party and because that judgment could be influenced by social factors, we extended the work of Kelly, *et al*.[6,7] The purpose of our research was to evaluate the effect of a patient's demographic characteristics and sexual preference on a physician's decision to breach an HIV-infected patient's confidentiality to protect a third party.

Methods

Sample

A stratified random sample of 628 practicing primary care physicians was identified[8] from a population of 2,804 primary care physicians practicing in Tennessee in 1986. The strata used were specialty (family practice, general practice, obstetrics and gynecology, or internal medicine) and size of the community in which the physician practiced (urban or rural). Randomization was successful so that the distributions by specialty and community size observed in the sample were similar to those observed in the population. Both the sample and the population consisted of approximately 27 percent family practitioners, 15 percent general practitioners, 20 percent obstetricians and gynecologists, and 40 percent internal medicine specialists. Rural physicians constituted approximately one-third of both of the sample and the population. Each physician in the sample was mailed a questionnaire, with instructions to respond anonymously. To improve the response rate, a second questionnaire was sent to the entire sample, with instructions to ignore the second mailing if the first questionnaire had been returned. The combined response rate to the two mailings was 37.1 percent (233 questionnaires returned). Besides sending questionnaires, further efforts to contact individual nonrespondents could not be made due to the anonymity of the respondents.

Questionnaire

The questionnaire elicited information about the physician's demographic characteristics, knowledge of HIV infection and AIDS, and attitudes toward individuals infected with HIV. One of eight variations of the following description of a hypothetical patient, based on the work of Kelly, *et al*,[6,7] was presented to each physician: "John (Joan) is a 30-year-old Black (White) heterosexual (homosexual) male (female) who tested positive for HIV by the ELISA antibodies test and confirmed by the Western Blot test. He (she) has not been getting along with his (her) partner. He (she) has asked you not to tell her (him, i.e., the partner) the results of the test because he (she) believes that the knowledge would complicate matters." The subject was described as one of the eight possible combinations of sex, race, and sexual preference (for example: Black homosexual female) until the eight possible combinations of sex, race, and sexual preference were exhausted. The eight vignettes, identical except for the description of the hypothetical patient, were distributed randomly among the 628 physicians. The algorithm for randomization was the same for the first and second mailings so that each physician received a second questionnaire with a description of the same hypothetical patient as was in the first. Each of the eight vignettes was followed by these progressively intrusive five statements (the first of the five

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statements was intended to infringe on the patient's privacy the least and the last statement the most):

1. The knowledge of the antibody status would remain between my patient and me.
2. I would attempt to persuade the patient to inform any partners who might be infected.
3. The antibody status, but not the name, would be reported to the health department.
4. The name of the person and the antibody status would be reported to the health department.
5. If the person would not inform any partners who might be infected, I would attempt to do so if the person identified them.

Physicians were asked to indicate which of these statements would characterize their actions. More than one selection was permitted.

Analysis

Of the 233 respondents to the questionnaire, 11 were not considered for the analysis because they did not report the action they would take. If a physician indicated more than one decision, the decision that most violated the patient's privacy was used as a dependent variable in the analysis. The responses of the three physicians who said that the knowledge of the patient's antibody status would remain confidential were combined with the responses of the 53 physicians who said they would try to persuade the patients to inform their partners. We used unconditional logistic regression[9] to compare the hypothetical patients' characteristics among the physicians' decisions. The dependent variable for three separate models was coded "0" if the physician would maintain patient confidentiality or persuade the patient to inform his or her partner. The dependent variable was coded "1" in these three situations: (1) if the physician would notify the health department of the patient's antibody status (the first model) or (2) if the physician would provide the health department with the patient's antibody status and name (the second model) or (3) if the physician would inform the patient's partner (the third model). The independent variables were the hypothetical patients' sex, race, and sexual preference. Also included in each model were the potentially confounding variables: physician age, year of graduation from medical school, physician specialty, and the urban or rural location of each physician's practice. The change in estimate criteria[10] was used to decide whether to retain the confounding variables in the final models.

In a second set of three logistic regression models, the independent variables were represented by seven dummy variables corresponding to seven of the eight combinations of sex, race, and sexual preference of the hypothetical patients. The category of Black homosexual female was used as a reference category and therefore not represented by a dummy variable. The category of Black homosexual female was selected as a reference because doing so made most of the odds ratios positive and therefore easier to interpret. These three regression models were also adjusted for confounding using the change in estimate criteria.[10]

In addition to including confounding variables in the regression models, confounding was addressed by assigning the descriptions of the hypothetical patients at random and by restriction of the regression analysis to White male physicians. The prevalence odds ratios presented in this paper are based on a cross-sectional survey and are therefore not estimates of relative risks.

Finally, to determine whether the physician's age, year of graduation from medical school, specialty, residence, knowledge of HIV infection and AIDS, and attitude toward individuals with AIDS modified the effect of race on the ethical decision, we used logistic regression models containing race-physician characteristic interaction terms.

Results

The distribution by specialty and location of practice of the 222 responding physicians (Table 1) was similar to that of the 628 physicians in the sample. Because half the questionnaires sent to the physicians described White hypothetical patients, half described Black, half described homosexuals, and so on, lack of response bias would be suggested by the return of questionnaires in the proportions in which they were sent. Of the returned questionnaires, 50 percent described female patients, 49.1 percent described Black patients, and 49.6 percent described homosexual patients. Further, each of the eight patient descriptions constituted approximately 12.5 percent of the sample of the returned questionnaires. To eliminate the physician's sex and race as sources of confounding, the subsequent analysis was confined to the 199 White male physicians in the sample.

Table 2 shows the physicians' characteristics associated with each type of hypothetical patient. While the relatively small numbers of observations in each category show some variation, there appear to be no systematic patterns of association. Similarly, Table 3 illustrates an unexpected consistency of physician age across the different decisions.

The unadjusted percentages of decisions for each type of hypothetical patient are shown in Table 4. The range of the percentage of physicians who said they would maintain the patient's confidentiality varies from only 8.7 percent for Black heterosexual males to 44.4 percent for Black homosexual females. Black males and Black heterosexual females were each associated with a relatively high percentage of physicians who said they would report their antibody status to the health department (17.4, 21.4, and 17.4 percent Black

TABLE 1—Distribution of the Characteristics of the 222 Responding Physicians

Variables	Percentage in Each Category
Age (years)	
21–30	0.5
31–40	31.5
41–50	21.2
51–60	20.3
60+	27.0
Race and Sex	
Black female	0.5
White female	6.8
Black male	3.2
White male	89.6
Specialty	
Family practice	27.9
General practice	11.7
Obstetrics and gynecology	20.7
Internal medicine	36.5
Missing	3.0
Community	
Rural	33.8
Memphis	19.8
Nashville	20.3
Chattanooga	11.3
Knoxville	14.9

TABLE 2—Percentage Distribution of Potentially Confounding and Effect-modifying Variables for the Categories of the Hypothetical Patients

Physicians' Characteristics	Black Homosexual Female (*n* = 18)	Black Homosexual Male (*n* = 28)	Black Heterosexual Female (*n* = 23)	Black Heterosexual Male (*n* = 23)	White Homosexual Female (*n* = 30)	White Homosexual Male (*n* = 24)	White Heterosexual Female (*n* = 27)	White Heterosexual Male (*n* = 26)
Age (years)								
21–40	5.6	28.6	56.5	30.4	33.3	37.5	37.0	15.4
41–60	50.0	42.9	26.0	30.4	46.7	29.2	37.0	61.4
61+	44.4	28.6	17.4	39.1	20.0	33.3	25.9	23.1
Year MD degree received (median)	1954	1960	1972	1968	1968	1963	1959	1960
Specialty[a]								
Family practice	27.8	10.7	26.1	39.1	26.7	33.3	37.0	34.6
General practice	22.2	17.9	8.7	17.4	10.0	4.2	18.5	7.7
Obstetrics and gynecology	11.1	17.9	21.7	21.7	20.0	16.7	18.5	26.9
Internal medicine	38.8	46.4	40.1	21.7	33.3	37.5	44.4	26.9
Community								
Rural	22.2	21.4	21.7	8.7	23.3	12.5	11.1	26.9
Urban	77.8	78.6	78.3	91.3	76.7	87.5	88.9	73.1

a) Percentages do not always add to 100% because 7 missing values are not shown.

TABLE 3—Percentage of Physicians' Decisions by Categories of Potentially Confounding and Effect-modifying Variables

Physicians' Characteristics	Would Maintain Confidentiality (*n* =56)	Would Report Antibody Status to Health Department (*n* = 21)	Would Report Name to the Health Department (*n* = 78)	Would Inform Patient's Partner (*n* = 44)
Age (years)				
21–40	30.4	33.4	32.1	29.5
40–60	35.7	38.1	42.3	45.5
60+	33.9	28.6	25.6	25.0
Year MD degree received (median)	1963	1959	1964	1961
Specialty[a]				
Family practice	23.2	38.1	25.6	38.6
General practice	16.1	9.5	9.0	18.2
Obstetrics and gynecology	21.4	9.5	26.9	9.1
Internal medicine	33.9	28.6	34.6	34.1
Community				
Rural	17.9	9.5	21.8	18.2
Urban	82.1	90.5	78.2	81.8

a) Percentages do not always add to 100% because 7 missing values are not shown.

TABLE 4—Percentage Distribution of Patient's Category among the Physicians' Decisions

Patient Category	Would Maintain Confidentiality		Would Report Antibody Status		Would Report Name		Would Inform Patient's Partner		Total
	n	%	*n*	%	*n*	%	*n*	%	(100%)
Black homosexual female	8	(44.4)	1	(5.6)	6	(33.3)	3	(16.7)	18
Black homosexual male	7	(25.0)	6	(21.4)	8	(28.6)	7	(25.0)	28
Black heterosexual female	7	(30.4)	4	(17.4)	8	(34.8)	4	(17.4)	23
Black heterosexual male	2	(8.7)	4	(17.4)	12	(52.2)	5	(21.7)	23
White homosexual female	10	(33.3)	1	(3.3)	15	(50.0)	4	(13.3)	30
White homosexual male	7	(29.2)	2	(8.3)	7	(29.2)	8	(33.3)	24
White heterosexual female	8	(29.6)	2	(7.4)	11	(40.7)	6	(22.2)	27
White heterosexual male	7	(26.9)	1	(3.9)	11	(42.3)	7	(26.9)	26
Total (%)	56	(28.1)	21	(10.6)	78	(39.2)	44	(22.1)	199

heterosexual males, Black homosexual males, and Black heterosexual females, respectively). Physicians said they would directly inform the partners of White homosexual males more often than they would individuals in other categories. The overall association between the hypothetical patients' characteristics and the physicians' decisions was not statistically significant ($\chi^2[21] = 20.2$, $p = .51$). Because the data were sparse, a Monte Carlo estimate of the exact *p*-value was run.[11] The estimated *p*-value was identical to the large sample *p*-value.

The ratios odds shown in Table 5 represent the results of three separate logistic regression models. In each of these models, physicians who said they would maintain the patient's confidentiality are compared to those who made one of the other three decisions shown in the column headings in Table 5. The odds for physicians saying they would send the antibody status of Blacks to the health department are 10.7 times (95 percent CI: 2.2, 52.0) those for saying they would send the antibody status of Whites. Males, Blacks, and heterosexuals have odds ratios greater than 1.0 for each of the three comparisons.

TABLE 5—Influence of Hypothetical Patient Category on White Male Physician's Decisions

Patient Category	Would Report Antibody Status		Would Report Name		Would Inform Patient's Partner	
	Odds Ratio	(95% CI) (n = 77)[a]	Odds Ratio	(95% CI) (n = 134)	Odds Ratio	(95% CI) (n = 100)[b]
Male	2.0	(.5, 7.3)	2.6	(.9, 8.0)	2.8	(1.2, 6.8)
Female	1.0		1.0		1.0	
Black	10.7	(2.2, 52.0)	3.6	(1.2, 11.0)	1.3	(.5, 3.1)
White	1.0		1.0		1.0	
Heterosexual	1.5	(.4, 5.9)	1.9	(.6, 5.7)	1.2	(.5, 2.9)
Homosexual	1.0		1.0		1.0	

a) Adjusted for physician speciality, year of graduation from medical school, and location of practice. Four missing values for specialty excluded from the analysis.
b) Adjusted for physician specialty. Three missing values for specialty excluded from the analysis.

To see if the average effects (e.g., Black) shown in Table 5 were masking the modifying effects of the patient's sex, race, and sexual preference on one another, we ran a second set of regression equations using dummy variables to represent seven of the eight categories of patient sex, race, and sexual preference (Table 6). The category Black homosexual female was selected as the reference category because of the high percentage of physicians who said they would maintain confidentiality in this category (44.4 percent, Table 4). Using Black homosexual females as a reference category made most of the odds ratios greater than 1.0 (Table 6). The odds of White male physicians saying they would report the antibody status of Black homosexual males, as opposed to maintaining their confidentiality, were 18.4 times (95 percent CI: 1.3, 260.1) those of saying they would report the antibody status of Black homosexual females (Table 5). Considering the small sample size, the magnitude of the odds ratios (OR) for reporting the antibody status for Black heterosexual males (OR 10.7, 95 percent CI: .7, 192.4) and Black heterosexual females (OR 9.3, 95 percent CI: .7, 132.1) should also be noted. When the patient was described as a Black heterosexual male, the odds of physicians saying they would report a patient's name to the health department (OR 8.0, 95 percent CI: 1.3, 50.0) or inform a patient's partner (OR 7.5, 95 percent CI: .8, 69.2) were highest. The odds of informing the partners of White homosexual males were 4.8 (95 percent CI: .7, 30.8) times the odds of informing the partners of Black homosexual females.

The analysis of the modifying effects of the physicians' characteristics on the race of the patient showed that family practitioners said they would send the names of Blacks (OR 19.1, 95 percent CI: 2.5, 143.9) to the health department more often than they would send the names of Whites (OR 1.0, reference). None of the other patient characteristics that we examined (e.g., age, location of practice, knowledge of AIDS) appeared to modify the influence of the patient's race on the physicians' decisions.

If the physician indicated more than one decision, preceding analyses used the physician's decision that most infringed on the patient's privacy as the dependent variable. To determine whether the patient's characteristics influenced the progression of the physicians' decisions from less to more intrusive, we compared the responses in which physicians indicated more than one decision (n = 120) with those in which physicians indicated they would directly report the patient (n = 23). Physicians more often said they would attempt to persuade women to inform their partners before intruding on their privacy (OR 2.5, 95 percent CI: .9,6.8) than they would men (OR 1.0, reference).

We could not evaluate the importance of the characteristics of the hypothetical partner in predicting the physicians' decisions because the assumed characteristics of the partner were highly correlated with the sex-sexual preference interaction term; models including both the characteristics of the patient and of the hypothesized partner could not be estimated. Those models including the characteristics of the partner only yielded results similar to those containing the characteristics of the patient. Because the sex, race, and sexual preference of the hypothetical partner were not known and those of the hypothetical patient were, we modeled only the patient characteristics.

To estimate the degree to which being a White male (relative to being a Black male) would influence physicians' underreporting of HIV-infected patients to the health department, we calculated the population attributable risk.[9] We defined the outcome of interest as not being reported to the health department; we defined the exposure as being a White male. Using the data in Table 4, the estimated cumulative incidence of physicians saying they would not report White

TABLE 6—Influence of Hypothetical Patient Category on White Male Physicians' Decisions

Patient Category	Would Report Antibody Status		Would Report Name		Would Inform Partner	
	Odds Ratio	(95% CI) (n = 77)[a]	Odds Ratio	(95% CI) (n = 134)	Odds Ratio	(95% CI) (n = 100)[a]
Black homosexual female	1.0	(Reference)	1.0	(Reference)	1.0	(Reference)
Black homosexual male	18.4	(1.3, 260.1)	1.5	(.4, 6.6)	4.2	(.7, 27.1)
Black heterosexual female	9.3	(.7, 132.1)	1.5	(.4, 6.6)	2.1	(.3, 15.6)
Black heterosexual male	10.7	(.7, 192.4)	8.0	(1.3, 50.0)	7.5	(.8, 69.2)
White homosexual female	.5	(.0, 11.5)	2.0	(.5, 3.8)	1.1	(.2, 5.7)
White homosexual male	.9	(.0, 20.1)	1.3	(.3, 5.9)	4.8	(.7, 30.8)
White heterosexual female	2.3	(.2, 35.2)	1.8	(.5, 4.0)	1.7	(.3, 2.3)
White heterosexual male	.7	(.0, 14.8)	2.1	(.5, 4.1)	2.3	(.5, 11.5)

a) Adjusted for physician specialty, 7 missing values excluded from the analysis. See footnote Table 5.

males to the health department was .40. The cumulative incidence of saying they would not report Black males to the health department was .23. If 33 percent of HIV-infected males are White,[12] then the percentage of HIV-infected males who would not be reported to the health-department because they are White is 20 percent.

Discussion

White male physicians' decisions to violate patient confidentiality to protect a third party were influenced in some cases by the race, sex, and sexual preference of a hypothetical patient. Presented with vignettes about hypothetical patients, these physicians said they would report Black homosexual and heterosexual men to the health department and Black heterosexual men to their partners more often than they would report hypothetical patients in the other categories. Black heterosexual women and homosexual men were also less likely to have their confidentiality maintained. Family practitioners said they would report the antibody status of Blacks to the health department more often than would practitioners in other specialties.

Although the response rate is low, the internal validity of the survey has not been compromised and may be better than it would have been had the survey not been anonymous. As noted in the results section, the questionnaires were returned in proportion to their distribution in the sample. As Greenland[13] points out, the equality of the proportion responding over the levels of the exposure variable is not sufficient to assure lack of response bias. Response bias could have occurred in this case if the physicians who would have said they would breach confidentiality had been more or less likely to have responded, depending on the description of the hypothetical patient they received. Since the description of the scenario occurred on the sixth page of the questionnaire, it is unlikely that the physician decision to return to the questionnaire depended on the characteristics of the hypothetical patient and even less likely that there were interactions among the decision to return the questionnaire, the characteristics of the hypothetical patient, and the physician response.

Another component of internal validity is information bias. It was to prevent information bias that the anonymous questionnaire was chosen, since we believed that questions about physician knowledge and ethical decisions could best be answered anonymously. This is the method used by Kelly, *et al*,[7] and we achieved a higher response rate than they did. While the proportion responding is an important criterion, the validity of a study should not be sacrificed merely to achieve a high response rate.

That the study is externally valid is suggested by the equality of the distribution of physician specialty and location of practice among the respondents to that of the sample. However, the respondents may differ from those who did not respond in ways that cannot be measured; what is important is that whoever these 222 respondents represent, some appear to be influenced by the sex, race, and sexual preference of the hypothetical patients when making an ethical decision. Even if the nonrespondents were completely impartial, the respondents constitute a large enough portion of the total sample to require consideration.

The fallacy of using statistical significance alone as a criterion for interpreting the results of observational studies has been discussed at length in this Journal.[14–18] Although there can be no question of the statistical significance of the odds ratios associated with Black homosexual and heterosexual males, one might question whether the borderline associations in the first and third column of Table 6 (having confidence intervals with lower bounds greater than or equal to .7 and less than 1.0) are meaningful. First, the borderline confidence intervals must be interpreted in the context of small sample sizes, multicollinearity, and the pattern of the odds ratios across the levels of the physician decisions. Of the five borderline confidence intervals, four are associated with a hypothetical patient who is Black. Because of the artifacts working against statistical significance, it would be improper to dismiss the borderline associations as uninformative. Most important, our results are consistent with the known stigmatization of Blacks,[19] more specifically of Black men, by Whites. It is in this context that our findings must be given serious consideration.

Whether the results can be attributed to prejudice or whether the different physician response patterns were motivated by consideration of the sources of HIV infection prevalent in the groups with which the hypothetical patients were associated cannot be determined from these data. Because Blacks who are infected with HIV are more likely to use intravenous drugs than are similarly infected Whites,[12] physicians may believe that they are less likely to be conscientious about informing their partners. Blacks are more often treated in the health department than are Whites[12]; therefore physicians who say they would send Blacks to the health department may merely reflect sociological reality rather than exhibit prejudice. However, saying they would send Black men to the health department more often than hypothetical patients in other categories is also consistent with racial prejudice.

Laumann *et al*,[20] found evidence for the underreporting of AIDS among middle class Whites. They suggest that the underreporting may be due, in part, to the diagnosis of Whites by private physicians, while Blacks are more often diagnosed by individuals directly associated with the health department. Our results are consistent with Laumann's findings and suggest one pathway for the underreporting of Whites. If accurate estimates of the number of individuals infected with HIV or diagnosed with AIDS are to be made, physicians should be made aware of this possibly unconscious bias in reporting.

Further research is needed to determine whether the observed patterns can be attributed to risk factors that are more prevalent among HIV-infected Black men (such as intravenous drug use) or with racial prejudice or both. Regional patterns of physician decision-making should be investigated, and the characteristics of physicians who make particular types of decisions should be identified. Because physicians may not be aware that their HIV-related ethical decisions are influenced by the characteristics of the patients, the results of such research can, in themselves, be educational and may also explain some of the patterns of underreporting of HIV-infected individuals.

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US Organ Transplant System Called 'Effective and Efficient'

The United States now has a "defined organ transplantation system, operated through the Organ Procurement and Transplantation Network, that appears to be effective and efficient," stated Dr. Robert G. Harmon, Health Resources and Services Administration (HRSA) Administrator, in testimony on April 20 before Rep. Henry Waxman's Subcommittee on Health, US House of Representatives.

The OPTN is operated by the United Network for Organ Sharing in Richmond, Virginia which has received HRSA contract funds since 1986 for its development and operation. All clinical transplant centers, organ procurement organizations, and tissue typing laboratories involved in organ transplantation are participating members of the network. Dr. Harmon cited a number of "significant accomplishments" made during the developmental period:

- The number of multi-organ donors increased by 30 percent since 1985, from 2,120 to 2,753 a year;
- The donor kidney discard rate decreased from 12.5 percent in 1986 to the present 4.6 percent;
- The number of kidneys exported overseas decreased by 93 percent, from 258 in 1983 to 17 in 1989, making more kidneys available to patients in the United States;
- A single source of data on organ donors, transplant candidates and transplant recipients has been established; and
- Procedures are in place to develop policies designed to assure equitable access to donate organs.

The number of heart and liver transplants and transplant centers has grown rapidly in recent years. From 1984 to 1989:

- Liver transplants rose from 308 to 2,160;
- Heart transplants rose from 346 to 1,673; and
- Pancreas transplants rose from 87 to 413.

The number of transplant programs also rose markedly. From 1984 to 1989:

- Heart programs increased from 37 to 147;
- Liver programs increased from 24 to 79; and
- Kidney programs increased from 170 to 221.

Despite the growing number of transplants, the number of people waiting for donor organs continues to increase. Currently more than 20,000 people are waiting for an organ transplant. The number of donors decreased from 4,090 in 1988 to 3,993 in 1989.

More information is available from the Division of Organ Transplantation, Bureau of Maternal and Child Health and Resources Development, Room 11A-22, HRSA, 5600 Fishers Lane, Rockville, MD 20857. Tel: 301/443-7577.

Effect of Changing Patterns of Care and Duration of Survival on the Cost of Treating the Acquired Immunodeficiency Syndrome (AIDS)

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Abstract: We performed a two-year cost of illness study of 240 AIDS patients (55 percent of all Massachusetts cases) diagnosed and treated at five hospitals from March 1984 through February 1986. Sociodemographic and clinical data as well as information on medical utilization were obtained from review of inpatient and outpatient hospital records.

The yearly inpatient cost per patient decreased by 28 percent from $38,369 in year one to $27,714 in year two. These changes were related to shorter lengths of stay (from 20.6 days to 16.8 days per hospitalization, mean difference of 3.8 days, 95% CI of the difference −.2, 7.8), and less costly hospitalizations (from $12,463 to $9,957, mean difference of $2,506, 95% CI of the difference $135, $4,877). The probability of hospitalization, however, was similar in both years. These patterns of care were still evident after controlling for transmisson category, race, site, mortality, insurance, age, gender, number and type of opportunistic diseases and time since diagnosis. Although the cost per patient per year decreased between years one and two, median survival increased by 70 percent (from 10 to 17 months). Hence overall estimated lifetime costs increased by 24 percent. (*Am J Public Health* 1990; 80:835–839.)

Introduction

As of December 1989, more than 100,000 cases of AIDS (acquired immunodeficiency syndrome) had been reported to the Centers for Disease Control (CDC).[1] It is estimated that there may be an additional 1.5 million people infected with the human immunodeficiency virus (HIV),[2] the virus that causes AIDS.[3–5] The CDC has projected that in the next three years, the number of AIDS patients may increase more than four-fold.[6]

Several studies examining the medical costs of treating AIDS have been performed, with lifetime medical care cost estimates ranging from $20,320 to $147,000 per patient.[7–16] Although these studies provide useful preliminary information, they suffer from a variety of deficiencies. Almost all of the studies to date have been based at single settings with small numbers of patients or have used the records of a single insurer. They have generally examined cross-sectional data over limited periods of time, hence were unable to measure changes in utilization over time. Temporal changes are important because medical and social treatment patterns may vary substantially for a newly discovered disease. In addition, previous studies have generally not included survival data which makes it difficult to estimate lifetime costs and have not provided information on the costs of treatment in relation to clinical characteristics.

This report describes a two-year study designed to address these issues. It includes data describing patterns of inpatient and outpatient medical utilization, survival and cost for patients with AIDS as well as changes in these patterns over the two-year period.

Methods

Selection of Patients

Study participants were selected by reviewing medical records at five major Massachusetts medical centers that care for patients with AIDS. Four Boston hospitals were selected because they provided care for the largest number of AIDS cases in Massachusetts; the fifth was selected because it had cared for the largest number of cases in the state outside of Boston. All persons receiving primary care for AIDS at one of the five study hospitals between March 1, 1984 and February 28, 1986, and who met the CDC criteria for AIDS[17] were included. Patients who met the 1986 CDC criteria for AIDS and did not receive their primary care at these institutions (those seen once for consult or only for therapeutic protocols) were excluded. The protocol for this study was approved by the Institutional Review Boards at all participating institutions.

A total of 240 cases were identified by the five medical centers, including 34 prevalent cases identified at the beginning (3/1/84) of the observation period and an additional 206 cases diagnosed during the two-year observation period. To assess completeness of patient identification, names of the study participants were phonetically coded by use of a Soundex code and compared with statewide AIDS surveillance records. The 206 incident cases represented 87 percent of the 236 cases that were diagnosed and reported to the AIDS surveillance program by the same hospitals and 55 percent of all AIDS patients reported to the state program during the two-year observation period. Due to concerns of confidentiality, we were not able to ask hospitals for records of the 30 patients that were discovered as having been reported to the surveillance program but not identified for this study. Hence we are unable to ensure that these 30 individuals were appropriate exclusions, i.e., diagnosed at the study site but recipients of primary care elsewhere. These 30 cases were similar in age, gender, transmission category, and presenting diagnosis to the 240 study subjects. Of the 240 cases, six cases (2.5 percent) were reported by more than one hospital and were credited for purposes of this study to the hospital in which the majority of utilization occurred; however, utilization from both hospitals was included in calculating overall costs.

Data Collection

Sociodemographic data (age, gender, insurance coverage, race) and clinical characteristics (opportunistic infection present at initial diagnosis, number of opportunistic infections developed during the study period, and risk factors for

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AIDS) were obtained by reviewing ambulatory and inpatient hospital records at the participating institutions. Use of outpatient services including office visits and diagnostic tests was evaluated by reviewing outpatient medical records at four of the participating institutions during the two year study period (March 1, 1984 to February 28, 1986). Because of administrative difficulties, the fifth institution, which cared for 23 percent of the study cohort, was able to provide data on outpatient utilization only for the first year of the study. Thus the denominator was reduced for calculations of outpatient utilization during the second year of the study.

Data on hospital utilization in terms of length of stay (LOS) and hospital charges were obtained from the billing departments of the participating institutions. Hospital costs were then estimated by applying the Massachusetts Rate Setting Commission year-specific/hospital-specific charge/cost ratios. Total inpatient costs per patient were derived by adding to hospital costs inpatient physician fees estimated using the following algorithim: 1) first inpatient day consisted of one admission visit, one consult visit, and one routine visit; 2) for each subsequent day, one routine visit fee was added.

Costs for ambulatory care and hospital doctor visits were estimated by using 1984 Blue Cross and Blue Shield customary reimbursement rates for office visits and diagnostic tests at hospital settings.

Data Analysis

Calculating Overall Cost—We examined cost of hospital care and ambulatory costs as well as the combined cost in terms of mean cost per patient per year during each of the two study years. Since every patient did not receive care for a full 12 months (some were diagnosed prior to the study period and some died during the study period) we tabulated a cost per patient per month for all patients alive in a particular month. For each year we calculated a mean for all patient months and multiplied by 12 to arrive at an annualized estimate.

Analyzing Incidence of Hospitalization—A two-part analysis was performed to better understand the trends and predictors of hospital admission lengths of stay and costs. We first examined overall rates of hospital admission and analyzed their association with patients' sociodemographic and clinical characteristics. Each month a subject in the study was coded as a binary variable depending on whether or not he or she was admitted to the hospital. Since more than one admission within a month was unusual (1.8 percent) and the second admission was usually related to the same episode of illness, the incidence of "hospital admission months" closely estimates the incidence of hospitalization. Crude rate ratios based on this index were calculated for study year, age, gender, transmission category (homosexual male, intravenous drug user, other), race, time since diagnosis, mortality (whether or not the patient died during the particular month), payor status, and number and type of opportunistic diseases.[18] We defined time since diagnosis as a binary variable based on whether the month was either the first or second since diagnosis, or a subsequent month. We made this determination by examining univariate plots which showed that the first two months after diagnosis were associated with a much higher incidence of hospitalization; thereafter rates were relatively similar. Next we examined the predictors of hospitalization simultaneously, estimating the likelihood of hospitalization during each month with a logistic regression model. The adjusted rate of hospitalization per month was estimated and 95% confidence intervals (CI) were calculated by the method of Miettinen.[19] For grouped variables, a likelihood ratio test of significance was performed.[20] This allowed us to assess the difference in rates of hospitalization each year for patients with the typical clinical and sociodemographic characteristics in our overall study sample. The probability of hospitalization per year was estimated from the adjusted coefficient of the maximum likelihood.[20]

Analyzing Length of Stay and Cost of Hospitalization—The second part of the analysis employed two linear regression models to examine temporal changes in the adjusted average length of stay and the average cost per hospitalization for those hospitalized. Using analysis of covariance we examined differences in cost between years one and two controlling for age, gender, transmission category, race, time since diagnosis, mortality, payor status, and the number and type of opportunistic infections.[21] As explained above, in less than 2 percent of months patients had more than one hospitalization. In order to use study "months" as our denominator we treated multiple hospitalizations during a month as a single hospitalization, combining the length of stay and costs related to the multiple events. Hence, our estimates for the likelihood of hospitalization are slightly low and our estimates for utilization and length of stay once hospitalized are slightly high. We know of no reason why this should be biased temporally. Because the distribution of the dependent variable was skewed, we performed a logarithmic transformation of length of stay and cost per hospitalization to assess statistical significance.

Survival Analysis—To determine overall cost per patient, a Kaplan-Meier survival analysis was performed on the cohort to determine the median survival.[22] A test of significance of the survival distribution comparing patients diagnosed during year one with patients diagnosed during year two was performed with the generalized Wilcoxon-Gehan Statistic.[22] Patient status (living or dead) was ascertained as of January 1, 1987 from vital records and mortality status was recorded as either dead, with the month and year of death, or alive.

To estimate lifetime costs, costs per patient month were multiplied by the median months of survival.

Evaluation of Outliers—A possible concern was the role of potential "outliers," i.e., individuals with unusually long or costly hospitalization, which may be related to temporal changes in the treatment of AIDS patients. To evaluate the potential impact of outliers, we examined the prevalence of 1 percent and 5 percent (based on the entire study population) length of stay outliers by study year. We then repeated both the length of stay and cost per hospitalization analyses with the 1 percent and 5 percent outliers removed.

Results

Table 1 displays demographic and clinical characteristics of the study cohort. The large majority were homosexual or bisexual men (81 percent); 16 percent were entirely uninsured, 24 percent had Medicare/Medicaid. Only 14 percent of patients presented with an initial diagnosis other than Pneumocystis *carinii* pneumonia (PCP) or Kaposi's Sarcoma (KS); 54 percent had multiple opportunistic diseases during the study period. There were no significant differences in demographic and clinical characteristics of individuals by year of diagnosis (Table 1).

Overall Utilization

The average length of observation was 7.6 (SD 5.3 months). Of the 240 patients with AIDS, 204 (85 percent) were hospitalized at least once during the study period. Members of the cohort were hospitalized 469 times (mean ±

TABLE 1—Demographic and Clinical Characteristics of AIDS Patients by Year of Diagnosis

Characteristics	Year 1* (n = 110) Number (%)	Year 2* (n = 130) Number (%)	Total (n = 240) Number (%)
Gender			
Male	103 (93)	120 (92)	223 (93)
Female	7 (7)	10 (8)	17 (7)
Risk Group			
Homosexual, Bisexual	88 (80)	106 (82)	194 (81)
Heterosexual/other/unknown	8 (7)	14 (11)	22 (9)
Intravenous drug use (IVDU)	9 (8)	6 (5)	15 (6)
Gay/Bisexual & IVDU	3 (2)	2 (2)	5 (2)
Blood Products	2 (2)	2 (2)	4 (2)
Insurance Status			
Blue Cross/Blue Shield	40 (36)	36 (28)	76 (32)
Public (Medicaid)	32 (29)	26 (20)	58 (24)
Commercial	17 (15)	24 (19)	41 (17)
None	13 (12)	25 (19)	38 (16)
Health maintenance organization	8 (7)	19 (15)	27 (11)
Presenting Diagnosis			
Pneumocystis carinii pneumonia (PCP)	51 (46)	63 (48)	114 (48)
Kaposi's Sarcoma (KS)	31 (28)	35 (27)	66 (28)
PCP and KS	15 (14)	12 (9)	27 (11)
Other Opportunistic infections	13 (12)	20 (15)	33 (13)
Number of Opportunistic Infections			
One	42 (38)	68 (52)	110 (46)
Two	32 (29)	32 (25)	64 (27)
Three	23 (21)	16 (12)	39 (16)
Four	13 (12)	14 (11)	27 (11)
Primary Care Hospital			
Site 1	44 (40)	55 (42)	99 (41)
Site 2	19 (17)	17 (13)	36 (15)
Site 3	7 (6)	6 (5)	13 (5)
Site 4	10 (9)	18 (14)	28 (12)
Site 5	30 (27)	34 (26)	64 (27)
Race			
White	87 (79)	96 (74)	183 (76)
Black	13 (12)	22 (17)	35 (15)
Hispanic, Other	10 (9)	12 (9)	22 (9)

*Year 1: 3/1/84–2/28/1985
Year 2: 3/1/85–2/28/1986

SD = 1.95 ± 1.8), with a mean length of stay of 17.3 (± 20.8) days (range 1–252). Mean cost per hospitalization was $10,868 (SD $12,813, Table 2).

Unadjusted medical care costs in year two were 28 percent lower than year one. Over 90 percent of these cost were related to inpatient utilization. Crude inpatient costs per patient per year were $38,369 in year one, compared with $27,714 in year two. Crude outpatient costs per patient per year were $2,706 in year one, compared with $2,215 in year two.

Rate and Predictors of Hospitalization

A stratified analysis of the rate of hospitalization showed that women, intravenous drug users (IVDU), Blacks, Hispanics, individuals who died, individuals on Medicare/Medicaid, and individuals at three of the five study sites were significantly more likely to be hospitalized. Individuals with only one opportunistic disease during the study period, or presenting with Kaposi's Sarcoma (KS) alone, were significantly less likely to be hospitalized during the study period.

When we examined age, study year, gender, risk, race, site, mortality, insurance, number and type of opportunistic diseases, initial diagnosis, and time since diagnosis simultaneously, we found that the number of opportunistic diseases, death during the study month, different categories of insurance status, time since diagnosis, and sites of care were predictors of hospitalization (Table 3).

TABLE 2—Overall Utilization during the Two-Year Study Period* (N = 240)

Description of Service	Mean ± SD	Range
No. of hospitalizations (N = 469)	1.95 ± 1.8	(0–10)
Length of Stay (days)	17.3 ± 20.8	(1–252)
No. of intensive care days	1.2 ± 5.0	(0–69)
Cost per hospitalization	$10,868 ± 12,813	($363–107,111)
No. of outpatient visits	7.0 ± 8.3	(0–60)
Cost per visit	$269 ± 307	($0–13,858)

*3/1/84–2/28/86

Temporal Changes in Utilization of Hospital Care

Table 4 shows the probability of hospitalization during each of the study years as well as the lengths of stay and the costs for hospitalization for those hospitalized. After adjusting for confounding variables, there was no important difference in rates of hospitalization between study years. However, once hospitalized, patients in year one had longer (3.1 days) and more costly ($2,432) lengths of stay.

Survival after AIDS Diagnosis and Lifetime Cost

Figure 1 contains the results of a Kaplan-Meier analysis of months of survival for two cohorts of patients, one cohort diagnosed in year one or earlier (n = 110) and the other diagnosed in year two (n = 130). Whereas individuals diagnosed in year one lived a median of 10 months after diagnosis those diagnosed diagnosed in year two lived a median of 17 months. We estimated lifetime hospital cost in each year as the product of unadjusted hospital cost and median survival. Although unadjusted hospital cost per year (inpatient and outpatient) decreased by 27 percent between year one and year two (from $41,075 to $29,929) (Table 2), estimated lifetime hospital cost increased by 23.8 percent (from $34,229 to $42,399).

Evaluation of Outliers

Interestingly, most of the observed temporal decrease in cost was related to the shorter length of stay and reduced cost for individuals hospitalized in year two. To evaluate the potential impact of outliers, we performed a test of significance of the prevalence of the 1 percent and 5 percent length

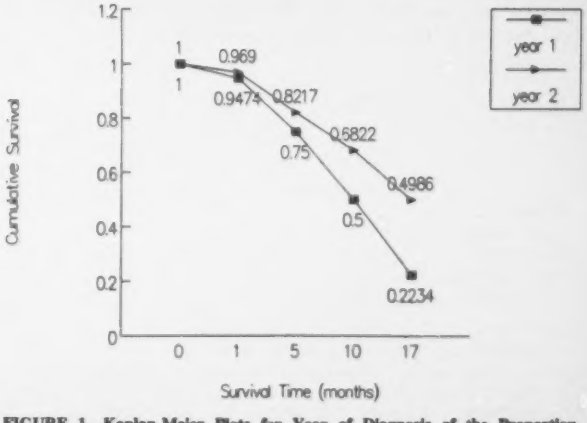

FIGURE 1—Kaplan-Meier Plots for Year of Diagnosis of the Proportion Surviving with AIDS

TABLE 3—Predictors of Hospitalization of AIDS Cases 3/1/84–2/28/86

Characteristics	Hospital Months	Total Months	Crude Relative Risk	Adjusted* Relative Risk	95% Confidence Interval	Exact# p-value
Age (Years)						
≤35	265	1144	-			.10
>35	169	679	1.07	1.23	(.95, 1.59)	
Gender						
Male	391	1687	-			.57
Female	43	136	1.36	1.04	(.60, 1.80)	
Transmission Category						
Homosexual/bisexual	329	1490	-			.15
IV Drug User	47	128	1.66	1.57	(.93, 2.67)	
Other	58	205	1.28	1.08	(.67, 1.73)	
Race						
White	302	1392	-			.40
Black	74	224	1.52	1.26	(.79, 2.00)	
Hispanic	58	205	1.29	1.38	(.94, 2.03)	
Site^						
Site 1	129	663	-			.12
Site 2	78	326	1.23	1.14	(.74, 1.76)	
Site 3	37	118	1.61	1.62	(.93, 2.80)	
Site 4	59	169	1.79	1.70	(.93, 3.13)	
Site 5	131	547	1.23	1.51	(1.09, 2.08)	
Mortality						
Alive	356	1710	-			.01
Dead	78	112	3.34	9.70	(6.25, 15.06)	
Insurance						
Blue Cross	128	545	-			.02
Commercial	59	313	.80	.73	(.50, 1.05)	
Public	139	482	1.23	1.17	(.85, 1.62)	
HMO	54	209	1.10	1.32	(.82, 2.14)	
None	54	224	.84	.70	(.46, 1.08)	
Number of Diseases						
More than one	282	1065	-			.01
One	152	758	.76	.64	(.48, .84)	
Initial Diagnosis						
P. carinii	211	821	-			.45
Kaposi's sarcoma	97	540	.70	.85	(.61, 1.18)	
PCP & Kaposi's	58	216	1.04	1.10	(.74, 1.65)	
Other OOI	68	246	1.08	1.12	(.77, 1.62)	
Time since diagnosis						
>3 months	283	1438	-			.01
≤3 months	151	385	1.99	2.92	(2.24, 3.79)	

*Controlling for all variables in the table.
#Comparing logistic models with and without the variable using an exact likelihood ratio test[20].
$Other = Heterosexual, Blood Related, or unknown categories.
^Site = Hospital which provided the majority of care.

of stay outliers by study year. The prevalence of both 1 percent and 5 percent outliers was significantly greater during the first year of the study. We then repeated both the length of stay and cost per hospitalization analyses with the 1 percent and 5 percent outliers removed. In the reanalysis we found that length of stay and cost per hospitalization remained significantly higher during the first year of the study.

TABLE 4—Temporal Changes in Use of Hospital Services

	Year One*	Year Two*	Crude** Difference	Adjusted*** Difference	Exact p-value
Probability of hospitalization per month	0.27	0.25	0.02	0.01	.13
Length of stay per hospitalization (days)	20.6	16.8	3.8	3.1	.04
Cost for these hospitalizations	$12,463	$9,957	$2,506	$2,432	.05

*Year 1: 3/1/84–2/28/85, Year 2:3/1/85–2/28/86.
**All differences are year one minus year two.
***Adjusted for age, number and type of opportunistic infections, transmission category, race, site, mortality, insurance, gender, and time since diagnosis.

Discussion

Our results illustrate the importance of multiple factors that produce temporal changes in the cost of treating AIDS. These include changes in: patterns of treatment, length of patients' survival, and characteristics of the patient population. Our findings suggest that although the cost per patient per year may have decreased, overall lifetime costs may have increased because of improved survival.

In our population, there was little temporal variation in patients' clinical and sociodemographic characteristics. However, this is not likely to be true in the future. Regions of the country such as New York City and Massachusetts have demonstrated a recent increase in the incidence of AIDS among groups such as intravenous drug users.[23,24] Our analysis suggests that such characteristics may also be important predictors of hospitalization and associated cost.

While the median survival time in our study population was short (14 months), it must be kept in mind that all of the

patients in our study were diagnosed prior to March 1986. Our cohorts' median survival was slightly longer than that observed in New York City during a similar period of time.[25] We assume that much of the increase in survival among individuals diagnosed during the second year is due to wider recognition of complications and changes in treatment. However, we were concerned some component may be due to lead time bias, an earlier diagnosis of disease, in which case our estimates for the differences in survival by year may be exaggerated. In order to assess this possibility, we reviewed surveillance records of patients diagnosed at the five study sites during the two time periods. We evaluated the method of diagnosis (n = 240), number of symptomatic months before diagnosis (n = 163), and the ratio of T-helper lymphocyte count to T-suppressor count (T-cell subsets) for those patients in which T-cell evaluations were performed and reported (n = 51). We found no significant differences in these indices by year of diagnosis. Hence, we feel that our findings are not likely to be a result of lead time bias.

Individuals with AIDS require a substantial amount of health care resources. In our study the mean cost of medical care was $37,404 per patient per year. Most other estimates have been in the $40,000 to $70,000 range.[8–16] These large costs have obvious implications for national health policy. Assuming an average lifetime cost of $42,399 per patient, the 110,000 AIDS patients already diagnosed will already require medical care expenditures of over $4 billion. The economic burden is substantial and may present a particular problem for teaching and public hospitals, where patients with AIDS tend to be hospitalized. It must be kept in mind that we assessed the direct medical care costs associated with treating AIDS and did not measure the indirect, nonmedical costs of AIDS, which may be enormous.

Several methodologic issues should be noted. As with virtually all cost-of-illness studies, we assessed average medical costs which may overestimate the incremental, i.e., marginal, medical cost of treatment. On the other hand, our results represent an underestimate of the total medical cost of treating a person with AIDS. Although we believe that the five hospitals in the study were the major source of medical care for the patients in our study, some patients may have received additional care at other sites. In addition, this study was performed before the licensure of AZT (azidothymidine). The annual cost of therapy with this medication is approximately $8,000 per patient per year[26,27] and it can be expected to have a major effect on both earlier diagnosis and survival after diagnosis.[28,29] We believe that both the methodology and results of our study will form a baseline to which later utilization studies may be compared.

Future studies of the economics of AIDS need to incorporate not only the actuarial estimates of the cost of care, but also clinical and organizational factors as well as life expectancy. The unique nature of this disease and its tremendous social and economic burden make such monitoring essential.

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The Impact of DRGs on Community-based Service Providers: Implications for the Elderly

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Abstract: We investigated changes in community-based agencies following the implementation of the Medicare prospective payment system for hospitals utilizing DRGs (diagnosis-related groups). Data were collected in 1986 and 1987 from 771 community service providers. There were five major findings: 1) hospital discharge planners, nursing homes, and home health agencies experienced DRG effects before other types of community providers studies; 2) the "reach" of DRG impact is widespread; 3) providers report a change in clientele toward a heavier-care client; 4) the impact of DRGs affects the types of services agencies provide; and 5) community providers have experienced a decrease in their ability to refer their clients both to hospitals and to each other. (*Am J Public Health* 1990; 80:840-843.)

Introduction

A number of research studies have focused on the short-term effects of the Medicare Prospective Payment System (PPS), particularly in terms of the utilization and cost of hospital services.[1-9] A limited number of studies have also focused on the effects of PPS on post-hospital care, although these have generally given primary attention to home health care and other Medicare-reimbursed services.[10-17] There has been little documentation, however, on the effects of PPS on a broader range of community-based service providers and, by extension, on the elderly population they serve. This important, but understudied, area of care is addressed here.

PPS-related early hospital discharge changed patterns of post-hospital service utilization, affecting community agencies, individuals and families.[18-26] The adequacy of the community-based service system to respond to new demands, either in the form of demand for new types of services or increased intensity of existing services, raises questions of access for the older adult population needing those services. While this study explores DRG (diagnosis-related groups) impact from an agency perspective and records organizational changes that service providers report making, it is possible, nonetheless, to suggest some of the ways in which these organizational changes will affect clients.

Methods

The research question asks post-hospital care organizations how DRGs have affected their staffing, service provision, and the types of clients served.

Data Sources

Study data were drawn from several sources:

- telephone survey interviews with a probability sample of seven types of service providers conducted in Spring of 1986 and again in Fall of 1987;
- telephone interviews with state and local policy makers and representatives of major provider associations ("key informants") in the states studied, conducted in 1988;
- detailed case studies and in-person interviews in two study communities;
- secondary data obtained from a variety of sources, including the Area Resource File, Census data, and the National Nursing Home Survey of 1985.

Site Selection

Sample States. States and SMSAs (standard metropolitan statistical areas) were selected purposively to assure some geographic variability. Each of the five states in the sample (California, Florida, Pennsylvania, Texas, and Washington) was the most populous state in its federal administrative region. They had also been identified by the regional directors of the Administration on Aging in 1976 as having the most innovative and generous aging programs in their regions. The states selected assured some diversity of sociodemographic and state economic conditions, factors that have been shown in other studies to affect expenditures and utilization of long-term care services. Our assumption is that, if we find shortfalls or gaps in these states, we would expect that the situation is likely to be as difficult or more so in smaller, less urbanized states.

Sample SMSAs. The SMSA was selected as the geopolitical unit from which to sample provider organizations because such catchment areas better represent the referral, market and interorganizational system for long-term care services than does a single city or county. In all cases one or two of the top four largest metropolitan areas (exceeding a population of one million) in the state were included in the study: Miami and Tampa/St. Petersburg; Philadelphia and Pittsburgh; Dallas/Ft. Worth and Houston; San Francisco/Oakland and San Diego; and Seattle. The sample represents nine of the 11 largest metropolitan areas in the five sample states and nine of the 24 largest metropolitan areas in the nation.

Provider Sample. The sample of provider organizations was selected randomly using probability sampling techniques, and the directors of each, or their designees, were interviewed by telephone for 30 to 60 minutes. The survey instrument contained both close-ended and open-ended questions covering the topics of: organizational structure; staffing patterns; clientele; service patterns; budgetary sources; opinion questions on policy issues; and perceptions of DRG effects in specific areas. This article presents the responses to the questions on DRG impact.

The service provider sample is comprised of a total of 771 respondents, representing seven different service providers interviewed in 1986 and again in 1987. This combined data set consists of: 178 discharge planners, 166 home health

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agencies, 166 nursing homes, 39 adult day care centers, 104 senior centers, 24 hospices, and 94 community mental health centers, rendering a total of 1542 interviews.

In 1986, respondents were asked to report on events and describe organizational changes occurring between 1984 and 1986 (to tap the immediate period of DRG implementation). In 1987, respondents were asked to report on events and changes occurring between 1986 and 1987. Thus, the data collected cover the period from 1984 to 1987.

Results

Five major findings emerged from the study (selected tables are in the text; further data may be requested from the authors).

- **First and Second Generation Effects.** In response to an open-ended question asking respondents to identify the policy that had most effect or impact on their organization during the time period studied, the policies that were most frequently nominated, in rank order, were DRGs and other Medicare policies (Table 1).

The types of DRG effects agencies described included increased demands as a result of earlier release of older hospital patients still needing some form of care. The other Medicare policies nominated were associated with reimbursement such as problems with fiscal intermediaries and inadequate reimbursement.

Table 1 also illustrates the lag time in reported DRG effect by different types of agencies. The "first generation" effects of DRGs (1984–86) were most immediately and directly experienced by hospital discharge planners, nursing homes, home health agencies and, to a much lesser degree, community mental health centers. In 1987, however, the percentage of these providers nominating DRGs as the most important policy affecting them declined, while adult day care centers and hospices reported being affected by DRGs for the first time.

While the one-fourth of adult day care centers reporting DRG effects is not large, the reason given for the impact, we believe, heralds an important trend. Adult day care centers reported that they were receiving more post-hospital patients referred from home health agencies after their Medicare benefits were exhausted, but before their care needs were met. This reason also explains the dramatic increase in the percentage of day care centers that reported being affected by other Medicare policies. At present adult day care services are paid for privately or by Medicaid, but not by Medicare. These centers believe the care they are now offering should be covered by Medicare as well.

- **The "Reach" of DRG Effects.** When we calculated the percentage of all providers reporting at least one type of DRG impact, we found that all seven types of providers reported being affected by DRGs in at least one of the following ways: types of clients served, services required or provided and/or referral patterns. The percent of providers reporting one or more types of DRG impact ranged from a low of 58 percent of senior centers to a high of 98 percent of discharge planners (table not shown). This is additional evidence that DRG effects were experienced not only by medical service providers but also by social service providers.
- **Change in Type of Client.** Providers were asked if they experienced changes in the types of clients they serve as a result of DRGs. As expected, the organizations most likely to receive patients directly following hospital discharge (home health agencies, hospices, and nursing homes) more frequently reported this type of DRG effect. Interestingly, however, even one-third of senior centers reported a change in clientele linked to DRGs (Table 2).

When asked to indicate *how* their clientele had changed, the predominant response across providers, was that clients were more frail, physically sicker and/or in need of more acute care services than in the previous study period. Overall, 60 percent reported this in 1986 and 57 percent in 1987.

- **Change in Services.** The increased tendency for clients to be heavier-care is also reflected in the responses to questions about DRG effects on services (Table 3).

Discharge planners differ from other providers in that their function is to assess and arrange for services needed by hospital patients upon discharge. Their most frequently mentioned service changes were in outpatient services and increases in the amount of discharge planning done by the hospital. For home health agencies, hospice programs, and nursing homes, the most frequently reported shifts in service were toward: more acute high-tech care; more skilled nursing; and more antibiotic intravenous therapy. Home health agencies reported this type of impact first (1984–86), while nursing homes and hospices reported more shifts of this type between 1986–87. Senior centers most frequently reported

TABLE 1—Percentage of Providers Reporting DRGs and Medicare Policies as the Most Important Policies Affecting Their Agencies

Agency Provider Type	1986*		1987**		
	DRG	Medicare	DRG	Medicare	N
	%	%	%	%	
Discharge Planners	90.4	10.7	62.3	12.4	(178)
Nursing Homes	28.2	12.4	24.1	13.2	(166)
Home Health Agencies	36.7	41.0	12.6	33.7	(166)
Community Mental Hlth	6.4	0.0	0.0	0.0	(94)
Adult Day Care	0.0	0.0	12.8	25.6	(39)
Hospice Programs	0.0	66.7	25.0	79.2	(24)
Senior Centers	0.0	0.0	0.0	0.0	(104)

SOURCE: IHA, DRG Study, Provider Instruments, 1986, 1987. "Please tell me what policies, or interpretations of policy, have had the greatest impact on your agency in the past twelve months, and what that impact has been."

*Data are for the 1984–86 time period.

**Data are for the 1986–87 time period.

TABLE 2—Percentage Reporting They Have Experienced a Change in the Types of Clients Served

Agency Provider Type	1986*	N	1987**	N
	%		%	
Home Health Agencies	92.1	(165)	82.3	(164)
Hospice Programs	87.5	(24)	60.9	(23)
Nursing Homes	84.3	(166)	87.2	(164)
Discharge Planners	71.3	(174)	70.9	(175)
Adult Day Care	52.6	(38)	43.2	(37)
Community Mental Health	30.8	(91)	40.7	(91)
Senior Centers	***	***	34.0	(94)

SOURCE: IHA, DRG Study, Provider Instruments, 1986, 1987.

"As a result of Medicare DRG payment to hospitals in the area you serve, has your agency experienced any of the following: a) Changes in the types of clients you serve?"

*Data are for the 1984–86 time period.

**Data are for the 1986–87 time period.

***Question not asked of senior centers in 1986.

TABLE 3—Percentage of Providers Reporting Changes in Services as a Result of DRGs

Agency Provider Type	1986* %	N	1987** %	N
Home Health Agencies	75.5	(163)	63.4	(164)
Hospice Programs	75.0	(24)	56.5	(23)
Discharge Planners	68.9	(177)	*	*
Nursing Homes	55.2	(165)	55.2	(165)
Adult Day Care	36.8	(38)	34.2	(38)
Community Mental Health	33.3	(90)	27.5	(91)
Senior Centers	***	***	29.0	(100)

SOURCE: IHA, DRG Study, Provider Instruments, 1986, 1987.
"As a result of Medicare DRG payment to hospitals in the area you serve, has your agency experienced any of the following: b) Shifts in the kinds of services you provide?"
*Data are for the 1984–86 time period.
**Data are for the 1986–87 time period.
***Question not asked of that provider type that year.

providing increased information and referral services, and adult day care centers reported providing more direct supervision and more rehabilitative therapy. Community mental health centers reported case management was increased as were services for the chronically ill.

As an additional indicator of whether or not patients were being released with more medically related needs, a question was asked about changes in the units of service required as a result of DRGs. The majority of providers reported changes in the units of service now required by their clientele. For both years of the survey, the reported changes were in the direction of increased units of service required—either increases in the number of visits or increases in the length of time of care per patient.

- **Change in Referral Ability.** A substantial percentage of agencies reported change in their ability to refer clients to hospitals (Table 4). The most frequently reported change was an increased difficulty in referring clients from community service providers *to* hospitals due to stricter hospital admission policies making *both* admission and re-admission more difficult. This problem was reported more frequently in 1986 than in 1987, although a sizable proportion of providers were still reporting difficulty in 1987.

Not all providers were queried in both 1986 and 1987 about changes in their ability to refer to other community providers (all providers were asked in the 1987 survey). However, of the three types of agencies for which there are data for both 1986 and 1987, discharge planners and home health agencies were more likely to report changes in their ability to refer to other agencies in 1986 than in 1987. The proportion of adult day care centers remained virtually the same both years. The direction of the change reported was an increased difficulty in referring clients to other community agencies

Most disturbing is the number of discharge planners who reported changes in the direction of increased difficulty in referring their hospital patients to other agencies for both time periods of the survey. In 1986, 31 percent of all discharge planners interviewed (N=178) reported increased referral difficulty due to fewer resources and more difficulty in gaining admission to nursing homes. In 1987, that percentage increased slightly to 34 percent and was reported to be due to: more difficulty in obtaining homemaker/chore services, fewer resources available in general and longer agency waiting lists.

TABLE 4—PERCENTAGE OF AGENCIES REPORTING CHANGES IN ABILITY TO REFER TO HOSPITALS

Agency Provider Type	1986* %	N	1987** %	N
Hospice Programs	58.3	(24)	40.0	(20)
Community Mental Health	43.3	(90)	38.6	(88)
Home Health	35.7	(154)	25.5	(161)
Nursing Homes	30.5	(164)	25.9	(166)
Adult Day Care	20.6	(34)	18.8	(32)

SOURCE: IHA, DRG Study, Provider Instruments, 1986, 1987.
"As a result of Medicare DRG payment to hospitals in the area you serve, has your agency experienced any of the following: d) Changes in your ability to refer clients to hospitals?"
*Data are for the 1984–86 time period.
**Data are for the 1986–87 time period.

Discussion

Study data suggest that the community-based service system is becoming saturated with physically sicker, heavier-care clients filtering down through the post-hospital community care services, affecting the service packages of both medical and social service agencies. There are three potential implications for the clients of these provider agencies:

- If the situation continues to move in the direction begun with PPS, there is likely to be a trade-off between our society's ability to meet the needs of the post-acute elderly as opposed to needs of the chronically ill elderly who have not been recently hospitalized. This could create problems of access for the chronically ill elderly residing in the community.
- Medicare coverage for services outside of the acute care setting is extremely limited. An increased need for these services can be expected to increase the already high out-of-pocket health care expenses of the elderly, creating problems of access for older persons with limited financial capacity.
- In other sections of our survey not dealing with DRGs specifically, we found that agencies were depending more on family caregivers. Increasing attention is being given to informal caregiving as a means to control costs. Insofar as possible, we expect that the informal care sector represents the "third generation" of DRG effects, as responsibility for care of the disabled and chronically ill older adults continues to shift to the informal sector.

We have examined some of the short-term effects of DRGs on a number of community agencies but we do not know how long-term these effects may be nor can we generalize our findings beyond the agencies and SMSAs studied. Continuing and more detailed research on the services and clients of providers is needed to determine if beneficiary access to both post-acute care and chronic care is adequate and whether the shifts of the type we describe are occurring more generally. Our findings also need to be further validated and complemented by research on the case-mix and service-mix of organizations of the type studied here. Given the probability method of sampling and the high response rates within the sample SMSAs, however, we are confident in, and disturbed by, our results.

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The American Cancer Society is expanding its research program in psychosocial and behavioral aspects of cancer to include primary prevention and detection. Investigators are encouraged to submit research and clinical grant proposals, and requests for support of personnel in research (Postdoctoral Fellowships, Physician's Research Training Fellowships, Junior Faculty Research Awards, Faculty Research Awards, and Scholar Awards) in these areas.

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The Prevention of Intellectual Impairment in Children of Impoverished Families: Findings of a Randomized Trial of Educational Day Care

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Abstract: We estimated the effects of an experimental educational day care program on the intellectual development of preschoolers from 86 high-risk families in a randomly allocated longitudinal study. At six through 54 months of age, the IQs of experimental program children ranged from 7.9 to 20.1 points higher than those of control children when maternal mental retardation and home environment effects were controlled; at every age, a greater proportion of the experimental program children had normal range IQs (> 84). In 13 children with retarded mothers, none of six experimental program children, but six of seven control children, had IQ scores below normal. (*Am J Public Health* 1990; 80:844–847.)

Introduction

Mild mental retardation is more prevalent among lower socioeconomic groups, unlike moderate, severe, and profound mental retardation which are fairly evenly distributed across socioeconomic groups.[1] Family and social problems of many low income families (e.g., low education, unemployment) have been associated with impaired intellectual functioning in general, and mild mental retardation in particular.[2,3] These findings, in combination with those of national surveys showing that 25 percent of the nation's preschool age children live in poverty,[4] suggest that mild mental retardation should be an important public health concern.

Preschool programs designed to enhance the intellectual development of high-risk children have generally been associated with positive outcomes; however, the intellectual gains associated with the programs are often of limited duration.[5]

The goal of this report is to evaluate simultaneously the effects of an educational day care program (i.e., the Carolina Abecedarian Project) and familial factors, specifically maternal retardation and home environment, on the intellectual development of high-risk children throughout their first four and a half years of life. Previous investigation of this sample found that educational day care led to improved intellectual functioning;[6*] however, the impact of multiple potentially confounding factors was not evaluated.

Methods

Intervention Procedures

The Carolina Abecedarian Project is a randomly allocated, controlled, longitudinal investigation of the effectiveness of educational day care in enhancing the intellectual ability of children from impoverished multi-problem families. Children assigned to the experimental condition entered day care between 6 and 12 weeks of age, and remained in care for five days a week, 50 weeks a year. The program was designed to promote social and cognitive growth in an orderly, friendly environment.[7,8]

Sample Selection

A total of 120 pregnant women whose unborn children (the target children) were at high-risk for intellectual impairment were identified by public health agencies and hospitals. High-risk status was defined by an index score composed of 14 risk factors for intellectual impairment.[9] Of the 120 families randomly assigned to either the experimental or control group, 107 continued participation for at least 54 months.[6] This report focuses on 86 (80 percent) of these 107 families for whom complete follow-up information is available (41 experimentals and 45 controls). No statistically significant differences were detected between the 86 families with complete information and the 21 families with incomplete information in terms of condition assignment, maternal IQ, or maternal education.

Table 1 shows the sociodemographic comparability of the experimental and control groups at study enrollment. Study families were primarily Black, low-income, single parent families. Mothers tended to be young and to have low IQs and low education levels. Target children were predominantly firstborns.

Assessment Instruments

The intellectual development of the children was evaluated using three standardized developmental and IQ tests.

TABLE 1—Characteristics of the Families at Study Enrollment

Characteristics	Control Group (n = 45)	Experimental Group (n = 41)
	n (%)	*n (%)*
Race		
Black	45 (100)	39 (95)
White	0 (0)	2 (5)
Firstborn		
Yes	27 (60)	28 (68)
No	18 (40)	13 (32)
Marital Status		
Single	31 (69)	31 (76)
Other	14 (31)	10 (24)
	X̄ (SD)	*X̄ (SD)*
Maternal age (years)	21 (6)	20 (4)
Maternal IQ (mean)	84 (11)	85 (13)
Maternal education (years)	10 (2)	10 (2)

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Editor's Note: See also related editorial p 790 this issue.

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The Mental Development Index of the Bayley Scales of Infant Development[10] was used for assessments at 6, 12, and 18 months of age, the Stanford-Binet Intelligence Scale[11] for ages 24, 36, and 48 months, and the McCarthy Scales of Children's Ability[12] for ages 42 and 54 months.

For some analyses, children were classified into one of three groups on the basis of their IQ scores. Employing standard clinical cutpoints,[13,14] children with IQs of 70 or less were categorized within the mentally retarded range of intellectual ability, children with IQs between 71 and 84 fell within the borderline intellectual functioning range, and children who scored 85 or more fell within the normal range of intellectual ability; children could move across groups dependent upon their test scores at particular ages.

The Home Observation for Measurement of the Environment (HOME)[15] was employed to quantify the degree to which each child's home surroundings provided an age-appropriate, potentially stimulating environment. The 45-item infant version HOME was administered at 6, 18, and 30 months, and the 80-item child version at 42 and 54 months. The mean HOME scores of the experimental and control groups were comparable at each of the five assessments and evidenced little within-family variation over time.[16] Therefore, the five evaluations were summed to create a HOME-SUM score representative of each child's home environment throughout the follow-up. For purposes of analysis, the study homes were categorized into two groups: "more stimulating" homes (i.e., homes at or above this sample's mean HOME-SUM score), and "less stimulating" homes (i.e., homes below this sample's mean HOME-SUM score). Twenty-two (54 percent) of the experimental families were classified as having more stimulating homes, as were 19 (42 percent) of the control families.

Analyses

Descriptive statistics were calculated to compare the experimental and control children in terms of their mean IQ scores and the proportion of children who fell within the mentally retarded, borderline, and normal IQ ranges. A repeated measures multivariate analysis of variance procedure[17] was employed to evaluate the effects of the experimental day care on the IQ scores of the study children across time, while controlling for, and examining the impact of, familial factors. The children's IQs were modeled using the following predictor variables: experimental educational day care (yes or no); maternal mental retardation (yes or no); home environment (more or less stimulating); annual family income; maternal age; maternal education level; maternal marital status; and the number of siblings. Using a backward elimination modeling strategy,[18] the full model was reduced to a smaller final model containing educational day care, maternal retardation, and home environment. The Wilks Lambda criterion (with a type I error rate of .05) was used to evaluate all multivariate tests. Effect sizes were measured using estimated regression coefficients, and 95% confidence intervals were calculated.

Results

Table 2 shows that the mean IQ scores of the experimental program children were above the general population average of 100 at all but one assessment, unlike that of the controls. At all time points, a greater percentage of experimentals than controls fell within the normal range of intellectual ability. Furthermore, at all ages except 24 months, a greater percentage of controls fell within the mentally retarded range of intellectual ability. At 54 months, 93 percent of the experimentals vs 69 percent of the controls had IQs within the normal range.

The 13 children of mentally retarded mothers were examined in detail. At 6 and 12 months, the mean IQ scores of the six experimental program children of retarded mothers were comparable to those of the seven control children of retarded mothers, with all mean scores being slightly above 100. However, by the 54-month assessment, the mean IQ of the experimental program children of retarded mothers was 22 points higher than that of the control children of retarded mothers (95 vs 73). The IQ scores of six (86 percent) of the seven control children of retarded mothers fell below the normal IQ range at 54 months (three within the mentally retarded range and three within the borderline intellectual functioning range). However, the IQ scores of all of the six experimental program children of retarded mothers were within the normal IQ range at the final assessment.

The repeated measures multivariate analysis of the entire sample found that the experimental and control groups differed significantly in their overall level of IQ averaged over time when the effects of maternal retardation and home environment were controlled ($F_{(1,82)} = 49.12$, $p = .0001$), with the experimental group having a greater grand mean than the comparison group. The two groups also differed in their patterns of change in IQ across time ($F_{(7,76)} = 4.76$, $p = .0002$). Stepdown contrasts examining the differences in IQ between successive time periods found that the groups differed significantly on all comparisons except for those assessing change between the 6 and 12 month assessments and the 42 and 48 month assessments.

As shown in Table 3, positive educational day care effects were found at all ages, with the mean IQ of the experimental children averaging from 7.9 to 20.1 points

TABLE 2—Mean IQ, Standard Deviation, and the Number and Percentage of Children in the Mentally Retarded (IQ < 71), Borderline Intellectual Functioning (70 < IQ < 85), and Normal (IQ > 84) IQ Ranges at 6 through 54 Months of Age

	Control Group (n = 45)				Experimental Group (n = 41)			
Age (years)	Mean(SD)	Retarded(%)	Border(%)	Normal(%)	Mean(SD)	Retarded(%)	Border(%)	Normal(%)
6	101 (14)	1 (2)	2 (4)	42 (93)	110 (14)	0 (0)	0 (0)	41 (100)
12	105 (15)	1 (2)	3 (7)	41 (91)	114 (13)	0 (0)	0 (0)	41 (100)
18	90 (13)	4 (9)	6 (78)	35 (78)	111 (12)	0 (0)	0 (0)	41 (100)
24	85 (9)	0 (0)	24 (53)	21 (47)	98 (11)	0 (0)	3 (7)	38 (93)
36	84 (14)	6 (13)	17 (38)	22 (49)	104 (13)	0 (0)	2 (5)	39 (95)
42	93 (13)	2 (4)	10 (22)	33 (73)	104 (11)	0 (0)	2 (5)	39 (95)
48	89 (14)	6 (13)	13 (29)	26 (58)	103 (12)	1 (2)	1 (2)	39 (95)
54	91 (14)	4 (9)	10 (22)	31 (69)	101 (11)	1 (2)	2 (5)	38 (93)

TABLE 3—Estimated Regression Coefficients and Associated 95% Confidence Intervals

Age in months	Regression Coefficients (95% CI)		
	Educational Day Care	Maternal Retardation	Home Environment
6	8.2 (2.2,14.1)	1.7 (−6.9,10.3)	4.8 (−1.4,11.0)
12	7.9 (2.0,13.7)	1.0 (−7.4,9.4)	4.9 (−1.2,10.9)
18	20.1 (15.0,25.2)	−3.3 (−10.6,4.1)	6.6 (1.3,10.9)
24	12.7 (8.9,16.7)	−4.3 (−9.9,1.5)	5.1 (0.9,9.3)
36	18.6 (13.5,23.7)	−8.2 (−15.6,−0.8)	8.6 (3.2,13.9)
42	10.2 (5.8,14.7)	−12.2 (−18.6,−5.8)	5.3 (0.7,9.9)
48	13.2 (8.5,17.9)	−11.7 (−18.5,−4.9)	8.9 (4.0,13.8)
54	9.1 (4.5,13.8)	−11.8 (−18.5,−5.1)	7.9 (3.0,12.7)

Note that for the multivariate analysis: educational day care was coded as 1 if experimental, 0 if control; maternal retardation was coded as 1 if retarded (IQ < 71), 0 if not retarded (IQ > 70); and home environment was coded as 1 if more stimulating (HOME-SUM scores above the mean), 0 if less stimulating (HOME-SUM scores below the mean).

higher than that of the control children when the effects of maternal retardation and home environment were controlled. The estimated effects of maternal retardation vary with the age of the children, the coefficients being small and positive at 6 and 12 months, but negative and progressively larger from 18 months onward. By 54 months, children with retarded mothers had IQ scores which averaged 11.8 points lower than children with non-retarded mothers, when the effects of educational day care and home environment were controlled. The effects of a more stimulating home environment were positive at every time point and increased over time. By the final assessment, children with more stimulating homes had IQ scores which averaged 7.9 points higher than did the children with less stimulating homes, controlling for the effects of educational day care and maternal retardation.

Discussion

The findings suggest that educational day care, maternal intelligence, and home environment each contributes to the developmental course of children's intellectual abilities. However, some cautionary comments and explanations are in order.

First, since the IQ scores of the experimental program children were consistently greater than those of the controls, one may ask whether the intellectual advantage of the experimental group was the result of the educational day care or the initial allocation procedure (i.e., were the children most at risk for intellectual impairment disproportionately allocated to the control group?). The facts that random allocation procedures were employed to assign families to groups, that known risk factors were evenly distributed between the groups, and that the experimental children had been in day care for at least three months prior to the initial IQ assessment, argue for the IQ differences being attributable to the educational day care program. Furthermore, the initial similarity of the IQ scores of the experimental and control group children of retarded mothers (i.e., all scored within the normal IQ range at 6 and 12 months of age) and the later dissimilarity of their scores (i.e., only 14 percent of the controls were in the normal IQ range at 54 months of age, compared to all of the experimentals) supports the notion that the educational day care resulted in improved intellectual functioning.

Second, issues arise concerning the patterns of change in IQ seen in the experimental and control groups over time. The decline in IQ seen in both groups after 12 months of age may be the result of at least two factors. The norms for the 12-month Bayley may be outdated, resulting in over-estimation of the true abilities of some children.[19] If the assessments at 6 and 12 months are inflated estimates of the children's functioning, while the later assessments are more accurate estimates, one would expect to see the observed decline in both groups of children after the 12-month evaluation. The differential magnitude of the decline (i.e., the downturn in scores was greater for the controls than for the experimentals) may be because the 6- and 12-month assessments focused on motor behaviors, while the later assessments incorporate more verbal behaviors. Perhaps the deficits associated with an impoverished environment may be mostly cognitive and verbal, rather than motor. This also could explain why the greatest fall in mean IQ after the 12-month assessment occurred in the subgroup of control children of mentally retarded mothers.

Improvement in the control group's mean IQ after 36 months of age may be due to the beneficial impact of community day care programs since approximately half of the control families entered their children into licensed community day care programs when the children were around three years old. A recent study of the impact of community day care in this sample of children concluded that the controls benefited intellectually from the community day care experience, although not to the same degree as the experimentals benefited from the Abecedarian educational day care.[20]

The positive impact of educational day care was especially pronounced for the children with mentally retarded mothers. At 6 months of age, all experimental and control children of retarded mothers had IQ scores within the normal range; however, by 54 months, all experimental group children of retarded mothers, but only 14 percent of the control children of retarded mothers, had normal range IQ scores. Although the number of children with retarded mothers in this sample is small (n = 13), the strong beneficial impact of this intervention merits attention because these children are at extreme risk for intellectual impairment.[21,22]

Given the beneficial effect of early educational day care in this sample of children with retarded mothers, the question arises as to whether families such as these typically receive day care services for their children. Data from the US National Longitudinal Survey of Youth[23] suggest that this is not the case: only 6 percent of the lowest functioning mothers of preschoolers (i.e., those mothers who scored in the bottom 5 percent on a standardized achievement test) enrolled their children in day care, compared to 20 percent of the highest functioning mothers (i.e., those mothers who scored in the top 95 percent on the achievement test). Thus the children who may be in most need of educational day care do not tend to receive these services. Given the positive findings of the Carolina Abecedarian Project, the children of low functioning mothers appear to be a group which public health policies should target to receive high quality educational day care interventions.

In addition to the educational day care effect, this study found both maternal mental retardation and home environment to be independent predictors of children's IQ scores. By the final 54-month assessment, the IQ scores of the children of retarded mothers averaged 12 points less than those of the children of non-retarded mothers, and the IQ scores of the children from more stimulating homes averaged 8 points higher than those of the children from less stimulating homes, when the effect of the experimental day care was controlled.

Finally, one may ask whether educational day care programs for impoverished families are cost-effective. Although, to date, no specific cost-benefit analyses have been performed using the Abecedarian data, a study of the teenage mothers in the sample found that by the 54-month evaluation, mothers in the experimental group were significantly more likely to have graduated from high school and be self-supporting than were mothers in the control group.[24] Given the large numbers of teenage mothers and children living in poverty in the United States today, the provision of high quality educational day care services to impoverished families may be a feasible strategy aimed at the prevention of mild mental retardation.

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Yom Kippur Services Open to APHA Convention Attendees

Individuals planning to attend pre-convention activities related to the American Public Health Association's annual meeting this fall in New York City should note that Yom Kippur falls on Saturday, September 29. For those pre-annual meeting participants who wish to attend Yom Kippur services, two New York City educational institutions offer services open to the public.

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  (Student-led services.)

Risk Factors for Syphilis: Cocaine Use and Prostitution

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Abstract: In Philadelphia, a large increase in syphilis among minority group heterosexuals began in 1986 and preceded similar increases elsewhere in the United States. To determine reasons for this increase, we conducted a case-control study in the metropolitan sexually transmitted diseases clinic during 1987 and 1988. Cocaine use (odds ratio [OR] 3.1; 95% confidence interval [95% CI] = 1.5, 6.5 among men; OR 5.8; 95% CI = 1.5, 33 among women) and exchange of drugs for sex (OR 3.5; 95% CI = 1.4, 8.7 among men) were risk factors for syphilis. Although cocaine users reported more sexual partners and more frequently reported sex with prostitutes, cocaine use remained a risk factor after adjustment for these behaviors. These data suggest that sexual behavior or another factor, such as availability or utilization of health care, among cocaine users leads to increased risk of syphilis in this population. Increases in cocaine use may be partly responsible for recent increases in syphilis incidence in the United States. (*Am J Public Health* 1990; 80:853–857.)

Introduction

In 1986, the incidence of reported primary and secondary syphilis in Philadelphia, Pennsylvania was 38.0 cases per 100,000 persons, a 62 percent increase compared to 1985. This increase reversed a four-year period of declining syphilis incidence in Philadelphia. The increase continued during 1987 and 1988, reaching 71.9 cases/100,000 in 1988. This increase preceded increases in other areas, such as New York City, California, and Florida.[1] In Philadelphia, as in the rest of the United States, increases were greatest for Black and Hispanic heterosexuals.[2]

Reports by syphilis interviewers, and a review of interview records between 1985 and 1987,[3] suggested that an increasing proportion of syphilis patients were users of illegal drugs, especially of smoked or "crack" cocaine. In addition, use of smoked cocaine increased during this period in Philadelphia and in many other metropolitan areas experiencing increases in syphilis.[4,5] Authorities suggested that cocaine-addicted female prostitutes, exchanging sex for a dose of "crack" cocaine, were responsible for syphilis outbreaks in Philadelphia and elsewhere. To test this hypothesis and to explore the relation between cocaine use and syphilis, we conducted a prospective case-control study of risk factors for syphilis in the sexually transmitted disease (STD) clinic operated by the city of Philadelphia.

Methods

Subjects

Consecutive patients diagnosed with primary and secondary syphilis and latent syphilis of less than six months duration and interviewed for partner notification at the STD clinic were enrolled as cases. Interviews occurred between September 1987 and January 1988. We accepted a clinical diagnosis of syphilis if confirmed by review of test results by the syphilis interviewers. During the study period, 82 percent of reported syphilis cases were interviewed for partner notification. We excluded patients whose partner notification interview occurred outside of the STD clinic (approximately 30 percent of interviews) because information obtained in these settings might not be comparable to that obtained in the STD clinic.

Controls were recruited from among other patients seen in the STD clinic during the same time period. We enrolled as controls consecutive patients seen in the clinic during specified periods of the day and week who had not had syphilis during the past six months. Since anecdotal evidence suggested that gonorrhea was also linked to cocaine use, we also excluded patients with gonorrhea from the control group. All male controls with symptoms or signs of urethritis and all female controls had negative cultures for gonorrhea. All patients were tested serologically for syphilis.

Measurements

A standard questionnaire administered to all patients by STD interviewers recorded demographic data, STD history, reason for coming to the clinic, site at which medical care was initially sought, medical diagnoses and laboratory test results from that clinic visit, as well as specific questions about sexual and drug use behavior during the three-month period before the clinic visit. This time interval was chosen to be short enough for accurate recall but long enough to reflect behavior during the period immediately before infection for most patients. The questionnaire was administered before the usual partner notification interview for syphilis patients.

We abstracted results of partner notification efforts conducted on syphilis patients, after completion of the study. We recorded the number of exposed sexual partners identified and the outcome of partner notification efforts for each identified partner. The abstracter was blinded to the patients' questionnaire results.

Analysis

We calculated crude and adjusted odds ratios and 95% confidence intervals using standard methods.[6,7] We used chi-square analysis or Fisher's exact test to compare categorical variables and Student's t test and the Wilcoxon rank-sum test to compare continuous variables.

We used the Statistical Analysis System logistic regression package[8] for multivariate analyses. Separate logistic regression models were used for men and women to adjust for confounding variables. Variables examined in these analyses included: age, race, education, histories of syphilis and gonorrhea, reason for coming to clinic, location of residence

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within the city, and numbers of sexual partners in the last three months and last five years.

Results

We initially enrolled 170 syphilis patients (cases) and 166 controls in the study. Twenty percent of cases had primary syphilis, 36 percent had secondary syphilis, and 44 percent had latent syphilis. Sixty-nine percent of controls received a diagnosis associated with *Chlamydia trachomatis* (non-gonococcal urethritis, mucopurulent cervicitis, pelvic inflammatory disease), 10 percent were diagnosed with trichomoniasis, 5 percent with pubic lice, 5 percent with genital warts, 2 percent with genital herpes, and 1 percent with chancroid. Fourteen patients (9 percent) received more than one STD diagnosis. In 16 percent of control patients, no sexually transmitted infection was detected.

Nineteen percent of male cases reported sex with another man compared with 7 percent of controls. All men reporting sex with another man were excluded from the rest of these analyses, since the goal of this study was to determine reasons for the increase in syphilis among heterosexuals.

Among heterosexuals, cases were more likely than controls to be women and of Hispanic origin, and had completed fewer years of school (Table 1). The number of sexual partners reported in the last three months and the last five years were not significantly different between cases and controls. Controls were more likely to have come voluntarily to the clinic for care while over half of cases were detected by serologic screening or health department partner notification efforts. Controls were significantly more likely to have a past history of gonorrhea but history of syphilis was not different.

Use of cocaine during the three months before the interview was significantly more common among both male and female cases (Tables 2 and 3). Among men, having given a woman drugs in exchange for sex, having had sex with a woman met at a "crack house" or a place where drugs are sold or used ("crack house" sex), and sex with a woman on the same day she was met (first day sex) were also risk factors for syphilis. Twenty-six of 27 men, for whom the specific drug exchanged for sex was recorded, reported trading cocaine for sex. These associations persisted after adjustment for age, race, education, location of residence within the city, and whether the patient presented voluntarily to the city STD clinic for care, using logistic regression (Table 2). Adjustment for the number of sexual partners reported in the last three months and five years also did not change the risk estimates.

Among women, having accepted money or drugs for sex (prostitution) was more common among cases than controls (Table 3). "Crack house" sex and first day sex were uncommon among women and were not associated with syphilis. We could not obtain valid adjusted odds ratio estimates for women using logistic regression, because of the small number of female subjects. Odds ratio estimates were not reduced in any case, however.

When we examined route of cocaine use, the patterns of association were somewhat different for men and women, although the small number of women limits these comparisons. Among men, the strongest association was with intravenous cocaine use, although non-intravenous use was more commonly reported and was also a significant risk factor for syphilis. In contrast to men, non-intravenous cocaine use, primarily by the nasal route, was the strongest risk factor for syphilis among women. In this population, nearly all intravenous drug users reported using intravenous cocaine.

TABLE 1—Characteristics of Heterosexual Patients in Study, Philadelphia 1987–88

Characteristics	Cases (n = 147)	Controls (n = 157)	P value
Age (median)	27	25.5	p = .003[a]
Sex			
Men (%)	95 (65)	126 (80)	p = .002[b]
Women (%)	52	31	
Race			
White (%)	12 (8)	9 (6)	p = .002[b]
Black (%)	116 (80)	144 (92)	
Hispanic (%)	17 (12)	3 (2)	
Unknown	2	1	
Education			
< High school (%)	58 (43)	34 (23)	p < .001[b]
12 (%)	66 (49)	80 (53)	
> 12 (%)	11 (8)	37 (25)	
Unknown	12	6	
Sexual partners (mean [median])			
Last 3 months			
Men	2.9 [2]	3.8 [2]	N.S.[a]
Women	2.1 [1]	1.5 [1]	
Last 5 years			
Men	36.9 [10]	23.1 [10]	N.S.[a]
Women	7.5 [3]	8.4 [3]	
Reason for presentation			
Volunteer (%)	64 (44)	143 (92)	p < .00001[2]
Health department partner referral (%)	21 (15)	7 (4)	
Detected by serological screening (%)	59 (41)	6 (4)	
Unknown	3	1	
History of syphilis (%)	7 (5)	6 (4)	N.S.[2]
History of gonorrhea (%)	29 (20)	54 (34)	p = .004[2]

a) Wilcoxon rank-sum test
b) Chi-square test

TABLE 2—Risk Factors for Syphilis among Heterosexual Men in Philadelphia, 1987–88

Risk Behavior[a]	Cases (n = 95)		Controls (n = 126)		Odds Ratio (95% CI)	
	n	(%)	n	(%)	Crude	Adjusted[b]
No cocaine use	50	(53)	93	(74)	1.0	
Cocaine use	45	(47)	33	(26)	2.5 (1.4,4.5)	3.1 (1.5,6.5)
Cocaine, by route of use[c]						
Intravenous	10	(11)	1	(1)	18.6 (2.3, 150)	24.1 (2.0, 295)
Smoked	15	(16)	13	(10)	2.2 (0.9,5.3)	2.2 (0.8,6.3)
Nasal and unknown	20	(21)	19	(15)	2.0 (0.9,4.3)	2.7 (1.1,6.9)
Cocaine use, other than intravenous	35	(37)	32	(25)	2.0 (1.1,3.8)	2.5 (1.2,5.4)
Sex with prostitute	26	(27)	22	(17)	1.8 (0.9,4.7)	2.4 (1.1,5.4)
Sex with prostitute by what was exchanged[d]						
Drugs	21	(22)	14	(11)	2.3 (1.1,4.7)	3.5 (1.4,8.7)
Money	15	(16)	11	(9)	2.1 (0.9,4.7)	2.9 (1.03,8.5)
"Crack house" sex[e]	23	(25)	16	(13)	2.2 (1.1,4.5)	2.6 (1.1,6.2)
First day sex[f]	51	(54)	50	(40)	1.8 (1.03,3.0)	2.2 (1.1,4.4)

a) All behaviors refer to the 3 months prior to the interview.
b) Adjusted for age, race, education, geographic residence within the city, and whether patient presented voluntarily to STD clinic for care.
c) Route of cocaine use is classified hierarchically as follows: any intravenous, smoked but not intravenous, and nasal or unspecified route, but not intravenous or smoked.
d) Exchange of drugs and money are not mutually exclusive categories.
e) Sex with a woman met at a "crack house" or a place where drugs are sold or used.
f) Sex with a woman on the same day that woman was met.

TABLE 3—Risk Factors for Syphilis among Women in Philadelphia, 1987–88

Risk Behavior[a]	Cases (n = 52)		Controls (n = 31)		Odds Ratio (95% CI)
	n	(%)	n	(%)	
No cocaine use	33	(63)	28	(90)	1.0
Cocaine use	19	(37)	3	(10)	5.4 (1.4, 31)
Prostitution:	6	(12)	1	(3)	3.9 (0.4, 91)
"Crack house" sex[b]	2	(4)	3	(10)	0.4 (0.1,2.4)
First day sex[c]	8	(16)	6	(19)	0.8 (0.2,2.5)

a) All behaviors refer to the 3 months prior to the interview.
b) Sex with a man met at a "crack house" or a place where drugs are sold or used.
c) Sex with a man on the same day that man was met.

Cocaine use remained a significant risk factor after adjusting for sex with a prostitute among men (OR 2.4; 95% CI = 1.3, 4.4) and for prostitution among women (OR 4.6; 95% CI = 1.2, 31). Among men, "crack house" sex and having had sex with a prostitute appear to measure a common risk behavior. A large proportion of patients reporting "crack house" sex also reported having had sex with a prostitute, and no risk was attached to either risk factor when present alone, while risk was greatly increased when both behaviors were reported (OR, 6.2; 95% CI = 2.0, 19). Cocaine use remained a risk factor for syphilis after adjusting for "crack house" sex.

Cocaine users were more likely to report sex with a prostitute and other behaviors likely to increase syphilis transmission, such as greater number of sexual partners and first day sex (Table 4). Men who smoked cocaine reported the highest frequency of each of these behaviors. Women who used cocaine also reported more sexual partners in the last three months (mean, 3.7 compared to 1.2; $p < 0.01$) and were more likely to report prostitution (6/22 compared to 1/60; $p < 0.1$) than women who did not use cocaine.

Men with syphilis who reported IV drug use were significantly more likely to have been diagnosed with secondary syphilis (73 percent) than were men who did not report IV drug use (27 percent), and were somewhat less likely to have been diagnosed with primary syphilis (18 percent compared to 33 percent).

TABLE 4—Sexual Behavior Associated with Cocaine Use, Heterosexual Men, Philadelphia, 1987–88

Sexual Behavior[a]	Cocaine Use		No Cocaine Use		P value[b]
	n = 78	(%)	n = 143	(%)	
Exchanged money or drugs for sex	30	(38)	18	(13)	$p < .001$
Drugs for sex	28	(36)	7	(5)	$p < .001$
Sexual partners in last 3 months					
0–1	17	(22)	49	(35)	$p < .01$
2–3	33	(42)	69	(49)	
4–5	15	(19)	15	(11)	
6–10	8	(10)	4	(3)	
> 10	5	(6)	4	(3)	
First day sex	48	(62)	53	(37)	$p < .001$
"Crack house" sex	25	(32)	14	(10)	$p < .001$

a) All behaviors refer to the 3 month interval prior to interview.
b) All p values are by chi-square. Number of sexual partners in last 3 months yields a Wilcoxon rank-sum test p value < 0.001.

Partner notification results were available for 88 percent (130/147) of syphilis patients. While men who had sex with a prostitute and men who used cocaine reported significantly more sexual partners in the last three months than did men who reported neither risk behavior (means 4.3, 4.0, and 1.8, respectively; $p < 0.05$), the average number of exposed sexual partners identified for partner notification purposes was lower for men reporting sex with a prostitute and cocaine use than for men reporting neither behavior (0.7, 1.0, and 1.3, respectively; $p < 0.05$ for sex with a prostitute). Prostitution and cocaine use were not associated with partner notification effectiveness among women, but the number of women in each group was small.

Discussion

These data support the hypothesis that cocaine use and sexual behaviors related to it are risk factors for syphilis in Philadelphia. The association between syphilis and cocaine

use was not fully explained by contact with prostitutes among men or by prostitution among women.

This study has several potential limitations. Data were collected in a public STD clinic in Philadelphia; results may not be generalizable to other locations or other health care settings. Moreover, our control group was chosen to control for factors related to the tendency to seek care at a public STD clinic. If cocaine use is also important in the transmission of other sexually transmitted infections, associations between syphilis and cocaine use would be underestimated. We excluded gonorrhea patients from the control group because of the possible link between gonorrhea and cocaine use; gonorrhea incidence was increasing in Philadelphia when the study began. The information we collected on gonorrhea patients suggests our concern was valid. Gonorrhea patients reported cocaine use and sex with prostitutes nearly as frequently as did syphilis patients. In contrast, no consistent differences in the frequencies of these behaviors existed among controls with different diagnoses. The exclusion of patients with gonorrhea is further supported by a report linking illicit drug use with spread of penicillinase-producing *Neisseria gonorrhoeae* in King County, Washington.[9]

Selection bias[10] could occur if, among persons with sexually acquired infection, cocaine users are more or less likely than nonusers to be seen for care in the STD clinic. If cocaine users are less likely to seek care, active syphilis case detection (laboratory screening and partner notification) could artificially increase the proportion of syphilis patients who are cocaine users, producing a spurious association between syphilis and cocaine use. Patients who presented voluntarily to the clinic were as likely to report cocaine use and prostitution as were patients detected by health department partner notification efforts and screening, so this bias was probably absent.

High-risk sexual behaviors, such as sex with prostitutes, were common among cocaine users and are partly responsible for the increased risk of syphilis associated with cocaine use. However, cocaine use remained a risk factor after adjusting for differences in these sexual behaviors and is not merely a marker for differences in sexual behavior. Cocaine use may indicate a population with a greater prevalence of syphilis, resulting in increased risk of infection for any given behavior. An increased prevalence of syphilis among cocaine users could result from differences in sexual behavior, such as those described here. Alternatively, drug use may reduce health care access or utilization, resulting in longer duration of infectivity and therefore increased transmission.

While smoked cocaine or "crack" has received attention as a possible cause of increases in syphilis incidence,[11] cocaine use by all routes was associated with syphilis in this population. Especially among men, intravenous cocaine was most strongly associated with syphilis. "Crack" use, which has increased dramatically in the populations most affected by syphilis and was most strongly associated with high risk sexual behaviors among drug users in this population, may be a more important factor in the current syphilis epidemic. However, the strong association with intravenous cocaine use, not explained by differences in sexual behavior among drug users in this study, may suggest the importance of some other factor, such as health care behavior.

The strong association with intravenous drug use could also be due to nonsexual transmission due to needle sharing. *Treponema pallidum* has been transmitted by blood transfusion and by needle injury during surgery.[12,13] Patients with transfusion-acquired syphilis are usually diagnosed during the secondary stage,[12] since without cutaneous inoculation no primary lesion occurs. Intravenous drug users in this study were somewhat less likely to be diagnosed during the primary stage of syphilis. Further studies are warranted to determine if *T. pallidum* is transmitted by needle sharing.

Finding a history of gonorrhea more commonly reported by controls than by cases was unexpected. While gonorrhea incidence increased over this time period in Philadelphia, it decreased in the face of dramatic syphilis increases elsewhere in the United States. Cocaine-related behaviors which increased syphilis transmission would be expected to increase gonorrhea spread as well. Syphilis and gonorrhea may be distributed differently within the population,[14] providing one explanation for these otherwise surprising findings. Further study of differences between risk factors for syphilis and gonorrhea and of reasons for the divergence between trends in syphilis and gonorrhea incidence are needed.

Prostitution has increased syphilis transmission in the past,[15,16] although it has been overshadowed during the past two decades,[17,18] by the important role of homosexual and bisexual men in syphilis transmission during this time period.[19] Prostitution has also been linked to illegal drug use in the past,[20] often serving as a means of financing drug use. Our data suggest that trading sex for drugs, rather than for money, is an important risk factor for syphilis. However, an increased number of prostitutes and persons exposed to prostitutes, due to the greater availability and use of cocaine, is probably more important than whether drugs or money are exchanged.

Our results are consistent with data from Oregon which suggest that partner notification is less effective among persons trading sex for drugs.[21] Other tools of disease control, such as targeted screening and presumptive treatment,[22] may be useful in appropriately defined populations and settings. Since delay in seeking or gaining access to medical treatment may be important, barriers which prevent drug users from obtaining medical care should be removed.

HIV infection is more prevalent among heterosexual Blacks and Hispanics in inner cities than among heterosexuals in other settings, because of intravenous drug use-related transmission.[23] In addition to being important in needle use-related transmission,[24] cocaine use may also lead to increased sexual transmission of HIV. Because accumulating evidence suggests that genital ulceration is an important co-factor in HIV transmission,[25–27] the potential for sexual transmission within these settings is sizable.

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Workers Satisfied with Health Benefits, but Won't Pay More

Eighty-five percent of American workers are satisfied with their health care benefits, but they resist the idea of paying more for them or accepting less coverage, according to a poll by Northwestern National Life (NWNL) Insurance Company of Minneapolis. The survey involved interviews with 606 full-time employees whose health benefits are provided by private industry and state and local government.

Satisfaction with their benefits persisted whether they pay all, a portion, or none of the premium. However, many said they would contribute little, if anything, more to maintain current benefits: 39 percent said they would not pay even a dollar more; an additional 32 percent said they would agree to a monthly increase of less than $25.

A previous survey of 400 companies by NWNL last year had revealed that nine of 10 employers would soon ask workers to pay a much larger share of health care costs: 70 percent of employees felt the amount their employer now pays toward their health insurance is "about right." According to A. Foster Higgins and Company, employers contribute an average of $149 a month for each employee and his/her dependents for medical, dental, prescription drugs, and vision/hearing benefits.

Employees surveyed by NWNL this year indicated they view health benefits as an entitlement, and tend to be more satisfied with their plan when they have a choice of coverages and an understanding of their benefits. However, many employees are not aware of basic benefits information: 43 percent did not know the amount their employer pays as a premium; 53 percent did not know if their health plan covers catastrophic illness or injury which could run as high as $400,000 (most health plans do cover their expenses, the report noted). Asked about changes they would like, nearly one-third said they would like costs to be reduced; 25 percent would like broader coverage for routine doctor visits, dental care and prescriptions; 9 percent wanted more freedom to choose doctors and hospitals; some wanted fewer restrictions, such as the requirement for pre-authorized care. But 21 percent said they would not change anything.

The results of the report are in: Employee Satisfaction with Health Benefits, (conducted by Winona Market Research Bureau) NWNL, PO Box 20, Minneapolis, MN 55440. Tel: 612/342-7137.

The Increasing Frequency of Heterosexually Acquired AIDS in the United States, 1983–88

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Abstract: Of 88,510 cases of acquired immunodeficiency syndrome (AIDS) reported in adults in the United States from 1983 through 1988, the percentage attributed to reported heterosexual contact with persons known to be infected or at increased risk of infection with human immunodeficiency virus (HIV) has increased steadily from 0.9 percent in the first quarter of 1983 to 4.0 percent in the fourth quarter of 1988, from 0.1 (in 1983) to 1.4 percent (in 1988) among men, and from 13 (in 1983) to 28 percent (in 1988) among women. Among women, the cumulative incidence of AIDS attributable to heterosexual contact per million population is over 11 times greater for Blacks and Hispanics than for Whites. Among men, this incidence is over 10 times greater for Blacks and four times greater for Hispanics than for Whites. The pattern of distribution of heterosexually acquired AIDS parallels the distribution of other heterosexually transmitted diseases, which are also more frequent in Black and Hispanic inner-city populations. Drug use, exchange of sex for drugs or money, and early onset of sexual activity in adolescents are increasingly associated with heterosexually transmitted infections and are likely to be very important in heterosexual transmission of HIV in inner-city US populations. (*Am J Public Health* 1990; 80:858–862.)

Introduction

Early during the AIDS (acquired immunodeficiency syndrome) epidemic, there was considerable concern that AIDS would spread rapidly among heterosexuals in North America and Europe. These concerns were heightened by emerging evidence of heterosexual acquisition of AIDS in Africa. When the heterosexual AIDS epidemic did not rapidly materialize in industrialized countries, these concerns came to be regarded with skepticism. This paper provides an analysis of trends in the frequency and percentage of cases of AIDS in the United States thought to have been acquired heterosexually, together with a perspective on heterosexual transmission of HIV (human immunodeficiency virus) in the context of a general overview of heterosexually transmitted infections.

Methods

This report is based on the 88,510 adult (age at diagnosis at least 13 years) AIDS cases diagnosed in the United States between January 1, 1983 and December 31, 1988, and reported to the Centers for Disease Control (CDC) through June 30, 1989. AIDS case reporting is required in all states and territories, with individual city, state, and territorial health departments having the responsibility for surveillance. Each known case meeting a surveillance definition[1] is reported to CDC by the health department, with a likely mode of transmission based on a hierarchical definition (Appendix). If no risk factor for HIV infection is reported with the diagnosis, the health department contacts health care personnel and, if necessary, interviews the patient to obtain information concerning personal behavior, sexual contacts, and occupation that could result in exposure to HIV. A case is classified as "no identified risk" if the patient has died, is lost to follow-up, refused to be interviewed, could not be assigned to a risk group from the information obtained, or if information is still being sought.

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In this report, we separate heterosexual transmission cases into two groups, corresponding to different epidemiologic characteristics and different trends over time. One group consists of cases classified as likely due to heterosexual transmission only because they were born in certain African or Caribbean countries where heterosexual contact is believed to play a major role in the transmission of HIV; these countries are designated by the World Health Organization as "Pattern II."[2] We concentrate on analyzing the remaining 2,637 heterosexual transmission cases that we designate as "reported heterosexual contact" cases. Of these 2,637 cases, 33 (1 percent) were born in these African or Caribbean countries but report heterosexual contact with intravenous drug users (IVDUs) (27 men, five women) or bisexual men (one woman).

We consider only cases of AIDS diagnosed after 1982 because the first reports of apparent heterosexual transmission were in late 1982.[3] Of the 1,394 adult cases of AIDS diagnosed before 1983, and not considered further here, two of the 1,305 men and 12 of the 89 women are classified as cases with reported heterosexual contact, and an additional 67 men and 10 women are classified as heterosexual cases due to birth in a "Pattern II" country.

Results

Trends in Heterosexually Acquired AIDS in US

The percentage of cases classified as due to reported heterosexual contact (as defined in Methods) increased from approximately 1 percent in 1983 to over 4 percent by the second quarter of 1988, while the percentage of cases classified as "heterosexual" cases in patients from "Pattern II" countries declined from over 4 percent in the first half of 1983 to about 1 percent by the end of 1988 (Figure 1). Only by separating these two groups can the increasing importance of known heterosexual contact cases be appreciated.[4] When these groups are not separated, no temporal trend in the percentage of AIDS cases due to heterosexual transmission is found.[5]

Table 1 shows the number and percentage of adult US cases of AIDS attributed to reported heterosexual contact for men and women by year of diagnosis (1983–88). The increase in annual percentages is especially striking for men (within each race/ethnicity group) and for non-Hispanic White women, but in no group was this increase steady over time.

TABLE 1—Number and Percent[a] of Adult AIDS Cases in the United States Attributed to Reported Heterosexual Contact,[b] by Gender and Race or Ethnicity, Diagnosed during 1983–88.

Year of Diagnosis	Non-Hispanic White N (%)	Hispanic N (%)	Black N (%)	All[c] N (%)
Men				
1983	0 (0.0)	0 (0.0)	2 (0.3)	2 (0.1)
1984	6 (0.2)	1 (0.1)	12 (1.0)	19 (0.4)
1985	9 (0.1)	2 (0.1)	21 (0.9)	32 (0.3)
1986	22 (0.2)	11 (0.5)	42 (1.2)	75 (0.5)
1987	77 (0.6)	28 (0.9)	105 (2.0)	212 (0.9)
1988[d]	116 (0.8)	49 (1.2)	161 (2.6)	328 (1.4)
All	230 (0.5)	91 (0.8)	343 (1.8)	668 (0.8)
Women				
1983	5 (10)	8 (18)	12 (13)	25 (13)
1984	12 (13)	21 (27)	31 (16)	64 (18)
1985	43 (21)	52 (34)	84 (22)	179 (24)
1986	100 (26)	85 (34)	158 (23)	347 (26)
1987	164 (25)	141 (34)	264 (23)	573 (25)
1988[d]	216 (29)	210 (37)	342 (23)	781 (28)
All	540 (25)	517 (34)	891 (22)	1969 (25)

[a]Of all adult cases with the same race/ethnicity and gender.
[b]Excludes cases in people without other identified risk factors who were born in "Pattern II" countries (see Appendix), where heterosexual transmission is believed to play a major role in HIV transmission.
[c]Includes cases in other races. In men, 2 of 486 cases in Asians, 1 of 87 in American Indians, and 1 of 187 in persons with race unknown are attributed to heterosexual contact. In women, the corresponding numbers are 13 of 44, 6 of 17, and 2 of 17.
[d]Reporting of cases is incomplete, especially for cases diagnosed in 1988.

Persons born in Puerto Rico have been overrepresented in Hispanic heterosexual AIDS cases. Of the cases in men, 39 (43 percent) were known to be born in Puerto Rico; of those in women, 157 (30 percent) were born in Puerto Rico. In contrast, in 1980 less than 7 percent of all US Hispanics had been born in Puerto Rico.[6]

Although the rate of increase in the percentage of cases attributed to reported heterosexual contact has been greatest for men, the absolute percentage of cases attributed to reported heterosexual contact is much greater for women. For cases of AIDS diagnosed during 1988, the overall percentage of cases attributed to reported heterosexual contact is 20 times higher for women than for men, and the actual number of cases also was higher for women from 1983 through 1988. The female to male ratio of the number of heterosexually acquired cases for 1988 was 1.9:1 for Whites, 2.1:1 for Blacks, and 4.3:1 for Hispanics.

Although the percentage of reported cases of AIDS attributed to reported heterosexual contact does not differ greatly by race or ethnicity, the cumulative incidence of such cases (1983–88) was much higher for Blacks and substantially higher for Hispanics than for Whites (Table 2). The cumulative incidence of such cases per million adults, 1983–88, was over 10 times higher for Black men and four times higher for Hispanic men than for White men. Among women, the incidence was more than 11 times higher for Blacks and Hispanics than for Whites. As discussed below, this is similar to what is currently seen for certain other sexually transmitted diseases.

The temporal pattern of the percentage of cases attributed to heterosexual acquisition by women may also be somewhat different from that seen in men. In each racial group, there was an appreciable percentage of heterosexual cases in women as early as 1982 (at least 6 percent in each group, data not shown). The percentage has increased most rapidly among White women, from 10 percent in 1983 to 29 percent during 1988.

TABLE 2—Cumulative Incidence[a] (1983–88) of AIDS Cases Attributed to Reported Heterosexual Contact, 1983–88, by Race, Ethnicity, and Gender

Race/Ethnicity	Men		Women	
	Cumulative Incidence	Ratio vs. whites	Cumulative Incidence	Ratio vs. whites
White	3.0	1.0	6.6	1.0
Black	32.6	10.7	73.6	11.1
Hispanic	13.2	4.4	74.1	11.2

[a]Per million persons, ages ≥13. The estimated population sizes, in thousands, for July 1988, were 81,563 for White women; 12,106 for Black women; 6,975 for Hispanic women; 75,868 for White men; 10,535 for Black men; and 6,869 for Hispanic men.

Source of Heterosexually Acquired AIDS

Table 3 summarizes the distribution of the sex partner's risk group for cases of AIDS attributed to reported heterosexual contact, by race and gender of the index person with HIV infection. IVDUs comprised a substantially lower percentage of the male sex partners for White than for Black or Hispanic women. Bisexual men comprised a much larger percentage of the male sex partners for White women with AIDS than for Black or Hispanic women with AIDS.

The racial distribution of female heterosexual AIDS cases stating that their contact was an IVDU (20 percent White, 31 percent Hispanic, 48 percent Black) was nearly identical to the corresponding racial distribution of male IVDU AIDS cases diagnosed during 1983–88 (19, 33, and 48 percent, respectively). Whites were overrepresented and

TABLE 3—Distribution of Partner's Risk Group for Reported Heterosexual Contact AIDS Cases,[a] by Race, Ethnicity, and Gender of Index AIDS Case, for Cases Diagnosed during 1983–88

Partner's Risk Group	Non-Hispanic White N (%)	Hispanic N (%)	Black N (%)	All[b] N (%)
Men				
Intravenous drug user	148 (64)	67 (74)	270 (79)	487 (73)
Pattern II[c]	17 (7)	3 (3)	17 (5)	37 (6)
Blood product transfusion	8 (3)	0 (0)	4 (1)	14 (2)
Other[d]	57 (25)	21 (23)	52 (15)	130 (19)
All	230 (100)	91 (100)	343 (100)	668 (100)
Women				
Intravenous drug user	273 (51)	439 (85)	679 (76)	1400 (71)
Bisexual	138 (26)	35 (7)	86 (10)	262 (13)
Pattern II[c]	0 (0)	0 (0)	24 (3)	24 (1)
Blood product transfusion	37 (7)	5 (1)	3 (0)	46 (2)
Hemophiliac	27 (5)	1 (0)	2 (0)	31 (2)
Other[d]	65 (12)	37 (7)	97 (11)	206 (10)
All	540 (100)	517 (100)	891 (100)	1969 (100)

[a]Excludes cases in people without other identified risk factors who were born in Pattern II countries (see Appendix), where heterosexual transmission is believed to play the major role in HIV transmission.
[b]Includes 4 men and 21 women of other races (see Table 1).
[c]21 of 37 men and 23 of 24 women were Haitians; the remaining 17 cases were born in sub-Saharan Africa.
[d]101 men and 179 women with heterosexual partner with HIV infection or AIDS but risk group not specified; 17 men and 14 women with heterosexual partner with uncertain HIV status and no indicated risk group; and 3 men with infected female partners who received clotting factors.

Hispanics were somewhat underrepresented in the male heterosexual AIDS cases stating contact with an IVDU compared to the racial distribution of female IVDU AIDS cases diagnosed 1983–88; the distributions were 30 percent White, 14 percent Hispanic, and 55 percent Black for male heterosexual cases reporting contact with an IVDU vs 22, 20, and 58 percent, respectively, for female IVDU cases.

Bias in Attribution

A possible source of bias is denial of other risk factors, such as IVDU and homosexual contact, perhaps as a result of an increasing awareness of heterosexual transmission. We examined the diseases present at diagnosis (Appendix). Of the 13,927 AIDS cases diagnosed in men during 1981–88 which included a report of Kaposi's sarcoma (KS), 13,177 (95 percent) were in homosexual or bisexual men (including 1,217 who were also IVDU). If the increase in the proportion of male cases attributed to heterosexual contact was primarily due to denial of homosexual contact, we would expect the percentage of reported heterosexual contact cases who had KS to change during 1984–88 to resemble the corresponding percentage in homosexual transmission cases more than the percentages in IVDUs and transfusion recipients.

We examined the annual percentages of male AIDS cases with KS during 1984–88 in these risk groups (Table 4). The percentage of AIDS cases in homosexual or bisexual men with KS declined during this period from 34 to 14 percent. The percentage of male AIDS cases attributed to heterosexual contact in 1988 was more than three times that in 1984. If this increase was due only to misclassification of homosexual contact AIDS cases, then at least two-thirds of the male heterosexual contact cases diagnosed in 1988 actually were due to homosexual transmission, so we would expect a diagnosis of KS in at least two-thirds of 14 percent, or about 10 percent, of the male AIDS cases attributed to heterosexual contact diagnosed during 1988. In fact, the percentage of heterosexual contact AIDS cases with KS remained at 5 to 6 percent for cases diagnosed during 1984–87, and was less than 4 percent for cases diagnosed during 1988. Thus KS in men with AIDS attributed to heterosexual transmission resembles the pattern of KS in male IVDUs and male transfusion recipients with AIDS (Table 4) much more than the pattern of KS in male homosexual transmission AIDS cases.

Discussion

Table 1 and Figure 1 might actually understate somewhat the true proportion of AIDS cases resulting from heterosexual transmission in the United States. When AIDS cases initially reported with no identified risk were investigated (including patient reinterviews) by Castro, *et al*, approximately one-fifth were reclassified as attributable to heterosexual transmission.[7] Of cases that remained in this category after follow-up investigations were completed, 38 percent reported a history of sexually transmitted disease, and 34 percent of the men reported contact with female prostitutes. Over 4 percent of AIDS cases diagnosed in 1988 currently are classified in the no identifiable risk category. Thus, heterosexual transmission of HIV probably accounts for close to 5 percent of AIDS cases now being diagnosed in the United States. Furthermore, the hierarchical system of classifying cases may misclassify some heterosexually acquired cases among persons with multiple risk factors for HIV exposure into other risk groups. Extrapolation of present trends suggests that at least 7 percent of adult AIDS cases diagnosed in late 1990 will be classified as reported heterosexual transmission cases (J Karon, unpublished).

On the other hand, several possible sources of bias could inflate the percentages of cases attributed to reported heterosexual contact, particularly in men. One possible explanation is that the observed trend may be due to a decreasing percentage of cases in homosexual and bisexual men. However, even after excluding all AIDS cases attributed to sexual contact between men (with or without IVDU), the percentage of cases in men attributed to reported heterosexual contact increased from 1.6 percent of the cases diagnosed in 1984–85 to 4.7 percent of the cases diagnosed in 1988. Although male AIDS patients with KS might be questioned more carefully than male AIDS patients without KS about same sex contacts, the data in Table 4 are not consistent with the hypothesis that the increase in male AIDS cases attributed to reported heterosexual contact is due *primarily* to misclassification of homosexual or bisexual men with AIDS. Nevertheless, as heterosexual transmission of HIV has become more widely acknowledged recently, it may have become easier for persons with AIDS to claim heterosexual exposure instead of homosexual contact or IVDU. This might especially be a problem outside the original epicenters. Decreasing likelihood of investigation (because of rising case loads) and recruitment of less experienced interviewers could also

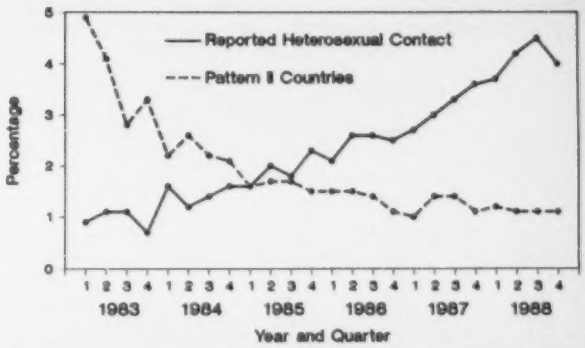

FIGURE 1—Trends in the Percentage of US Cases of AIDS Classified as Heterosexually Acquired, by Quarter of Diagnosis, 1983–88.
Declining trends in the percentage of cases born in WHO Pattern II countries (where heterosexual transmission predominates, such as Haiti and countries in sub-Saharan Africa) are offset by steadily increasing trends in the percentage of remaining US cases of AIDS attributed to reported heterosexual contact with an individual in another risk group for HIV infection or with someone known to have AIDS or HIV infection.

TABLE 4—Percentage of Men with AIDS with a Diagnosis of Kaposi's Sarcoma: Comparison of Reported Heterosexual Contact Cases, and Cases in Other Risk Groups, for Cases Diagnosed during 1984–88

Year of Diagnosis	Percent with Kaposi's Sarcoma			
	Homosexual or Bisexual[a]	Heterosexual IVDU	Transfusion Recipients	Reported Heterosexual Contact
1984	34.4	4.1	4.9	5.3
1985	28.4	2.9	5.7	6.2
1986	23.0	3.3	4.0	5.3
1987	18.1	2.4	3.6	5.7
1988	14.1	2.6	2.7	3.7

[a]Not also IVDU

account for some of the temporal increase in AIDS cases attributed to heterosexual contact.

Because the source of the HIV infection in most cases attributed to heterosexual contact is reported as an IVDU (Table 3), another indirect assessment of misclassification of these cases is a comparison of the geographic distributions of cases attributed to heterosexual contact and of those attributed to IVDU. The geographic distribution of heterosexual contact cases in women is similar to that of cases in male IVDUs, but there are substantial differences between these distributions for male heterosexual contact cases and female IVDU cases.[8] The ratio of male heterosexual contact cases to female IVDU cases is unusually low in New York and unusually high in Florida. Thus, the direction of any misclassification bias is uncertain. Geographic differences in follow-up of men claiming heterosexual contact as their only risk factor probably causes some of these geographic differences.

The change in the criteria for a reportable AIDS case in September 1987 may affect subsequent trends in the percent of cases attributed to reported heterosexual contact. The criteria were expanded to include both additional diseases and presumptive diagnoses of some opportunistic infections in persons known to be HIV-seropositive.[1] This change affected the percentages of cases in some risk groups, especially homosexual/bisexual men not using IV drugs (a sharp decrease) and heterosexual IVDU (a sharp increase).[9] These changes roughly offset each other in men, resulting in a modest increase in the proportion of cases attributed to reported heterosexual contact: for cases diagnosed during September 1987 through December 1988, this proportion is 1.2 percent (355 of 28,686 cases) diagnosed under the pre-1987 definition and 1.8 percent (199 of 10,894 cases) diagnosed under the expanded criteria. There is a marked effect in the opposite direction among women, with corresponding proportions 30 percent (840 of 2,756 cases diagnosed under the pre-1987 definition) and 22 percent (412 of 1,835 cases diagnosed under the expanded criteria), due to the increase in cases in women using IV drugs reported under the expanded criteria.

The present analysis shows that for AIDS cases reported from 1983 through 1988 and attributed to reported heterosexual contact, the cumulative incidence was much higher in Blacks and Hispanics than in Whites (Table 2). By comparison, for all cases of AIDS reported to CDC through January 18, 1988, Selik, *et al*, reported that the incidence was 2.8 times higher for Black men and 2.7 higher for Hispanic men than for White men; the incidence was 13.2 times higher for Black women and 8.1 times higher for Hispanic women than for White women.[10,11] Among Hispanics, incidence has been higher in those born in Puerto Rico than elsewhere, especially for IVDUs.[12] Thus, among men, the incidence of AIDS attributable to reported heterosexual contact for Blacks and Hispanics, relative to Whites, is higher than the overall risk of AIDS for Blacks and Hispanics, relative to Whites. The racial and ethnic differences in the incidence of AIDS attributed to reported heterosexual contact mirror the racial differences in incidence of AIDS associated with IVDU,[13] as well as differences in incidence of other sexually transmitted diseases (STD) as discussed below.

Further evidence for the importance of heterosexual acquisition of HIV among women comes from an analysis of the mother's exposure category for AIDS cases in children (age < 13 years at diagnosis). Of the 1,176 pediatric AIDS cases diagnosed through 1988 and reported to CDC through June 1989 that were attributable to perinatal transmission of HIV from a mother not born in a Pattern II country, 350 (30 percent) involved children born to mothers whose sex partners were in a risk group for HIV infection or had known HIV infection. Trends in this proportion should be further examined to see if the proportion is increasing over time.

Because of the delay of several years from acquisition of HIV until onset of AIDS, the increasing percentage of cases of AIDS seen now that are attributable to reported heterosexual contact actually reflects increasing trends in heterosexual transmission of HIV that were occurring several years ago. Data are not yet available concerning the current percentage of new HIV infections in the United States that are attributable to heterosexual acquisition, but there is a growing body of information about the prevalence of HIV in various nonrandom samples of populations that were not selected for specific high-risk behaviors.

For example, among US military applicants the overall prevalence of HIV seropositivity from October 1985 through March 1988 was 1.2 per 1,000, and was related to age, sex, race, and geographic area.[14,15] Male sex, Black race, and residence in a metropolitan area with a high incidence of AIDS were associated with the highest rates of HIV seropositivity. Seroprevalence rates exceeding 1 percent were observed in six counties in New York, New Jersey, California, and the District of Columbia. In future surveys, to assess further the role of heterosexual transmission in comparison with the role of other risk factors, it will be important to attempt representative sampling, and to conduct reinterviews of seropositive heterosexual persons.

So far, prostitution and sex with prostitutes have been implicated as important in the heterosexual transmission of HIV mainly in Pattern II countries.[2,16–19] Nonetheless, the history of prostitute contact among 34 percent of US male patients classified as having no identified risk[7] dictates that this issue be examined closely in the future. Other specific risk factors for heterosexual transmission include receptive anal intercourse for women[20] and immunosuppression in the source contact.[21] In Africa, genital ulcer disease[22–25] and lack of male circumcision[23,25] have been repeatedly implicated as cofactors associated with heterosexual transmission. Other sexually transmitted infections such as cervical chlamydial infection,[24] vaginal trichomoniasis,[26] and current oral contraceptive use[24] have been implicated as risk factors for heterosexual acquisition of HIV in single studies and require confirmation in other studies. Condom use has been associated with reduced risk of heterosexual transmission of HIV.[27–29]

Much remains to be learned about the epidemiology of heterosexually transmitted HIV infection in industrialized countries. However, the fact that HIV seropositivity among heterosexuals in the United States is associated with urban residence and certain racial minorities should not be automatically attributed solely to patterns of distribution of IVDU, since these associations also parallel the epidemiology of other heterosexually transmitted infections in North America. In the United States the reported incidence of other sexually transmitted diseases, such as gonorrhea, like the incidence of heterosexually acquired AIDS, is highest for persons of lowest socioeconomic status, higher for urban dwellers than for those living in the suburbs or rural areas, and much higher for Blacks and Hispanics than for Whites and Asians.[30] For example, during 1988 in the United States, the incidence of reported cases of gonorrhea was approximately 21 times higher in Black than in White women.[30]

Reporting of syphilis is considered to be more complete than reporting of gonorrhea. During 1987, the incidence rates of reported early syphilis in the United States were 25 times higher in Black men and 12 times higher in Hispanic men than in White men; the incidence was 31-fold higher in Black women and 8-fold higher in Hispanic women than in White women.[31] Chancroid, another sexually transmitted disease that has been increasing recently in incidence in the United States, has also occurred predominantly among Blacks and Hispanics, often in association with prostitution.[32]

It is evident that at least four phenomena in inner-city indigent populations could interact to promote heterosexual spread of HIV in the United States.[30] These include: 1) the pattern of sexual behavior, including early onset of sexual intercourse, prostitution, and sex with prostitutes; 2) epidemic spread in these populations of sexually transmitted diseases, including genital ulcer disease, which themselves may promote heterosexual spread of HIV; 3) transmission of HIV by IVDU; and 4) increasing use of drugs such as crack cocaine, which promotes high-risk sexual behavior, including traditional forms of prostitution and exchange of sex for drugs—a form of prostitution often involving teen-agers. Promotion of "safe sex" practices will be especially difficult in this group.

Culturally sensitive, age-specific social and health educational programs, endorsed by community leaders and beginning in early adolescence, if not sooner, are urgently needed to interrupt the heterosexual spread of HIV. These should be accompanied by programs to better control other STDs as risk factors for HIV transmission, and by programs to prevent and curtail illicit drug use.

APPENDIX

Risk Groups for AIDS/HIV Transmission

In the CDC hierarchical definition of risk group, a person with AIDS who reports sexual contact with a member of the same sex (if male), receipt of clotting factors, or IVDU will not be classified as having AIDS due to heterosexual transmission. In the absence of these risk behaviors, a person classified as having AIDS due to heterosexual transmission must either report heterosexual contact with a person known to be infected or at risk for infection (i.e., someone in one of the above risk groups, an HIV-infected transfusion recipient, or a person born in a Caribbean or African country designated by the World Health Organization as "Pattern II"). Nearly all people in the United States with reported AIDS attributed to heterosexual transmission who were born in these countries were born in Haiti (1,233 of 1,253). A person with AIDS reporting heterosexual contact with a prostitute but no other risk factor is classified as having no identified risk, not as a heterosexual transmission case.

As a result of the hierarchical classification, some cases attributed to other exposures (especially IVDU) could actually have resulted from heterosexual contact. Follow-up has shown that some cases initially classified as having no identified risk actually resulted from heterosexual exposure.[7] Of course, some cases designated as "reported heterosexual contact" may have other risk factors, such as unreported homosexual contact or IVDU.

For each AIDS case, the surveillance data include the diseases recorded at the time of diagnosis, but additional diseases that develop later are not necessarily reported. This could affect time trends of the percent of cases with a disease, such as Kaposi's sarcoma (KS). For example, if wasting syndrome tends to develop earlier in the course of HIV disease than KS, the addition of wasting syndrome to the AIDS surveillance definition in 1987 would tend to decrease the percent of reported cases with KS.

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New NCHS Report Summarizes 1987 Divorce Statistics

The National Center for Health Statistics has issued a new report giving the final United States divorce data for 1987, and showing that the divorce rate (for 1,166,000 divorces) dropped 1 percent from the previous year, and was down 4 percent from the record high of 1,213,000 reached in 1981. Several measures of divorce have declined to levels of the 1970s:

- The divorce rate for 1987 was 4.8 per 1,000 population; provisional data for 1989 show a further decline to 4.7.
- The number of children involved in divorce was 1,038,000 in 1987, fewer than in any year since 1973 and reflecting both the declining number of divorces and the shrinking size of American families.

In other findings, the study reported that:

- 52 percent of divorcing couples had children under 18 years of age.
- 12 percent of those married less than a year had children, as did 26 percent of those married 1–2 years; 42 percent of those married 3–4 years; 60 percent of those married 5–9 years, and 75 percent of those married 10–14 years.
- Divorcing men and women who were college graduates had fewer minor children than couples with less education.
- Husbands and wives divorcing in 1987 were older than their counterparts in any year since 1970, reflecting longer durations of first marriages and marrying at older ages.
- For both sexes, divorce in 1987 is concentrated between 20 and 45 years of age: 81 percent of husbands and 87 percent of wives were under age 45; only 3 percent of husbands and 2 percent of wives were age 60 or over.
- In 1987, 72 percent of divorces were ending first marriages, 21 percent ending second marriages, and 7 percent ending third or higher order marriages. The average age of men ending first marriages was 34.6 years. Typically, women were 2–4 years younger, depending on the number of times they were married.

The summary report is published by NCHS as *Advance report of final divorce statistics, 1987,* in Monthly Vital Statistics Report 1990;38(12), suppl 2. Hyattsville, MD, USPHS. The full report will be published in *Vital Statistics of the United States, Vol III—Marriage and Divorce*. For further information, contact Sandra Smith, Public Affairs Officer, NCHS, (301) 436-7135, or Barbara Foley Wilson, Demographer, (301) 436-8977.

Public Health Briefs

Patient 'Dumping' Post-COBRA

ARTHUR L. KELLERMANN, MD, MPH, AND BELA B. HACKMAN, MD

Abstract: To gauge the impact of the new federal patient transfer provisions following the federal Combined Budget Reconciliation Act of 1985 (COBRA), we monitored all emergency interhospital transfers to a public hospital emergency department in the Memphis, Tennessee area during three identical time periods: June 1 to August 31 of 1986, 1987, and 1988. A high number of transfers in the summer of 1986 diminished only slightly in summer 1987 (following implementation of COBRA). Far greater reductions occurred in summer 1988, when overcrowding forced our hospital to refuse most transfers. In contrast to changes in hospital policy, COBRA alone had little effect in this area. (*Am J Public Health* 1990; 80:864–867.)

Introduction

On August 1, 1986 the patient transfer provisions of the federal Combined Budget Reconciliation Act of 1985 (COBRA) became law.[1] Under COBRA, hospitals receiving Medicare funds are required to take certain steps prior to transferring unstable patients or women in active labor. Although COBRA is primarily intended to stop the inappropriate transfer of unstable patients for economic reasons (a practice known as patient "dumping"), the impact of this law is unclear.[2] To study the effect of COBRA in our community, we identified all patients transferred to our public hospital emergency department during the summer months of 1986, 1987, and 1988.

Methods

Shelby County, Tennessee contains the city of Memphis (1985 population 635,000) and is surrounded by rural west Tennessee, northern Mississippi, and eastern Arkansas. The county's population is 54.5 percent White and 44.5 percent Black, with small Asian and Hispanic minorities. One of 10 general hospitals in the county, the Regional Medical Center at Memphis receives an annual operating subsidy from Shelby County government to treat county residents who otherwise cannot afford care. No other hospital for adults in Shelby County receives county funds.[3]

During each of three study intervals, June 1–August 31 of 1986, 1987, and 1988, we identified all patients transferred to the Medical Center Emergency Department from area hospital emergency departments or their affiliated free-standing emergency centers. Patients sent directly to one of the Medical Center's special care units (Level 1 Trauma Center, Burn Unit, and High-Risk Obstetric Service) were assumed to represent tertiary care referrals and were excluded.

Transfers were considered "authorized" if the sending facility contacted a physician at the Medical Center who accepted the patient prior to transfer. Patients sent without prior notification, patients sent despite the Medical Center's refusal to accept the transfer, and patients in route prior to the transfer request were considered to be "unauthorized." Transfers were judged to have been made for primarily economic reasons when "no money" or "Shelby County indigent" was explicitly identified by the sending physician as the primary reason for transfer or no specific reason for transfer was recorded but the patient lacked private insurance, Medicare, or Medicaid. Unstable patients were identified by three independent reviewers who compared each patient's chart to a previously published[3] list of explicit criteria (see Appendix). Only patients meeting one or more criteria in the judgment of all three reviewers were considered to be unstable at the time of transfer.

COBRA became law on August 1, 1986 but no immediate changes in local hospital policy occurred at that time. However, in October 1986, local news stories[4–6] and subsequent state[7] and federal[8] hearings focused increasing attention on patient "dumping." Telephone screening of transfer requests was also intensified, but our long-standing practice of accepting indigent patients deemed "stable" for transfer did not change. Although hospital over-crowding intermittently required us to restrict interhospital transfers during 1986 and 1987, these intervals were generally brief. By the summer of 1988, however, overcrowding became so severe that the Medical Center Emergency Department (ED) was repeatedly forced to restrict authorization of interhospital transfers for days at a time. No other major changes in health policy occurred in Memphis during this time (Table 1).

TABLE 1—Study Interval Characteristics

	Summer 1986	Summer 1987	Summer 1988
Time Interval	92 days	92 days	92 days
Total ED Visits	15,617	17,355	18,220
COBRA in Effect?	No*	Yes	Yes
Hospital Crowding	Moderate	Moderate	Severe
Total Days Transfers to Medical Center ED Restricted**	31 days	36 days	65 days

*COBRA implemented 8/1/86; law not widely recognized until fall of 1986.
**Authorizations restricted all or part of a calendar day.

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Results

During summer 1986, 266 patients were transferred to the Medical Center ED, 243 of which (91 percent) were sent for primarily economic reasons. Somewhat fewer patients (N = 226) were transferred during summer 1987, (when COBRA was in full effect), but the proportion sent for primarily economic reasons did not change. Under the severely overcrowded conditions of summer 1988, total interhospital transfers declined precipitously (N = 85), but the proportion sent for primarily economic reasons remained the same (92 percent).

More than half of patients transferred during the summer of 1986 were unauthorized, including four sent despite refusal by the Medical Center. During summer of 1987 (post COBRA) unauthorized transfers declined by only 18 percent, but 10 patients were sent despite refusal by the Medical Center. During summer 1988, unauthorized transfers declined by fully 61 percent compared to 1986; five transfers arrived despite prior refusal by the Medical Center (Figure 1).

COBRA is intended to stop the inappropriate transfer of unstable patients, particularly when transfer is being accomplished for primarily economic reasons.[2] Based on explicit criteria, 64 of 243 patients (26 percent) transferred to the Medical Center primarily for economic reasons during the summer of 1986 were determined to be unstable. Two of every three had been accepted as "stable for transfer" on the basis of telephone assurances by the sending physician.[3] Unstable economic transfers fell modestly in the summer of 1987 (N = 46) and declined sharply in summer 1988 (N = 13) but the proportion sent without advance authorization did not change. Virtually identical numbers of economic transfers required emergency hospitalization during the summers of 1986 and 1987, and the number requiring emergency surgery and/or intensive care actually increased. Both total admissions and total critical care cases fell sharply during the summer of 1988 (Figure 2). Seven transferred patients died

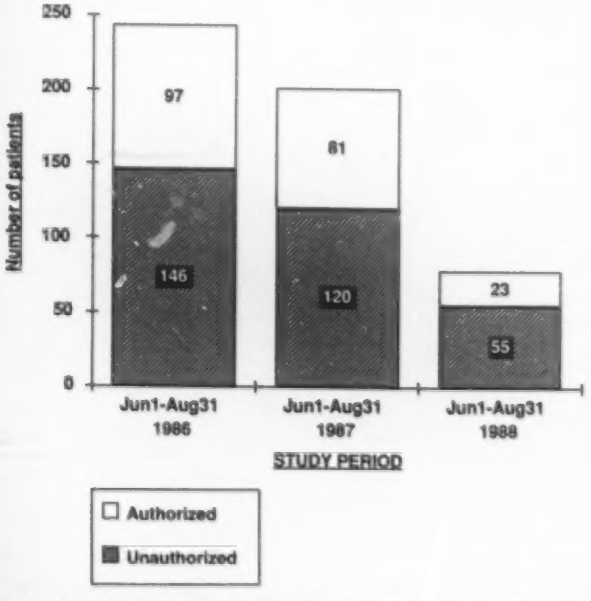

FIGURE 1—Authorized vs Unauthorized Economic Transfers to Memphis Medical Center, Summer 1986, 1987, 1988

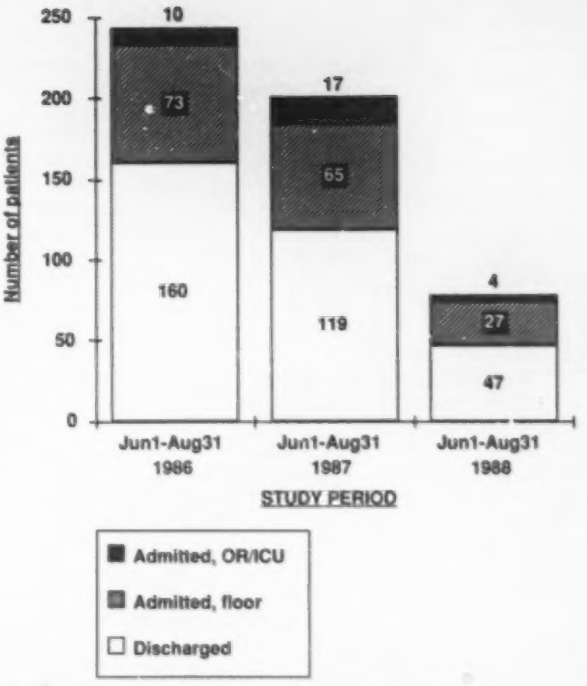

FIGURE 2—Disposition of Economic Transfers Made to Memphis Medical Center Emergency Department, Summer 1986, 1987, 1988

prior to hospital discharge during one of the three study periods: three in summer 1986, three in summer 1987, and one in summer 1988. None of these had been sent without advance authorization by the Medical Center.

Discussion

In 1988, an audit by the Office of Inspector General for the Health Care Financing Administration found that record keeping at most sending hospitals is inadequate to monitor patient transfers in general, much less to screen for "dumping," and that few receiving hospitals have a procedure for identifying potential violations of COBRA. With no effective mechanism for monitoring transfers, the report concluded that ". . . the ability of HHS (Department of Health and Human Services) to monitor and oversee this (dumping) problem is jeopardized. . ."[9]

By documenting interhospital transfers to a single public hospital, we were able to study community practice before and after implementation of COBRA. Explicit criteria were devised and consistently applied throughout each of three identical study intervals. Definitions of "unstable cases" and "economic transfers" were narrowly drawn. As a result, our case totals are almost certainly conservative. For example, all obstetric, burn, and trauma center transfers were presumed to represent tertiary care referrals, even when the patient was indigent and his or her problem could have been easily managed at the original hospital.

Four limitations to our study warrant comment. First, given the case mix of our emergency department, we cannot address COBRA's impact on transfers of children or of women in labor. Second, our data are based on temporal associations between changes in national (and local) health

care policy and changes in the number and types of patients transferred to our emergency department. Although no major changes to Shelby County's health care system occurred during the time period studied, we cannot exclude possible effects of other, unidentified factors. Third, without access to the charts of indigent patients seen at other hospitals, we cannot judge whether they were better or worse off when transfers to our hospital were restricted. We believe that most who would otherwise have been transferred to the Medical Center were either admitted or adequately treated at the original hospital, since few found their way to our emergency department the following day. Finally, our conclusions are based on transfers to a single public hospital. They may not reflect COBRA's effect on patient "dumping" in other cities or states.

In an era of intense health care competition, the pressure on private hospitals to transfer non-paying patients to public facilities is great. We found that COBRA alone had little impact on the magnitude or nature of patient "dumping" in Memphis. Substantial reductions in total economic transfers did not occur until the Medical Center began to sharply restrict authorizations due to overcrowding. The role COBRA may have played in limiting the number of unauthorized transfers during this latter time period is unclear.

Caught between concerns about patient dumping and the need to maintain cordial multi-institutional relationships, public hospital officials are reluctant to address potential violations of COBRA.[9,10] Recently HCFA proposed stiff penalties for receiving hospitals that fail to report suspected cases of dumping.[11] Our data suggest such measures may be needed. As currently written, the impact of COBRA appears to be slight.

APPENDIX
Severity of Illness Criteria
(noted at time of arrival in receiving emergency department)

Vital Signs

a) Heart rate >140 or <50 beats per minute
b) Respiratory rate >28 or <8 breaths per minute
c) BP Diastolic <40 or >130 mm Hg
d) Temperature <94 or >102 degrees fahrenheit

Cardiovascular

a) Chest pain with suspicion of ischemia
b) Rhythm: supraventricular tachycardia with heart rate ≥149 heart block (second degree mobitz II or greater), ventricular tachycardia or high grade ectopy
c) IV antiarrhythmics or IV pressors (eg, dopamine, dobutamine, norepinephrine, lidocaine, procainamide, bretyllium) during transport

Respiratory

a) Hypoxia (PO2 ≤ 60 mm Hg regardless of FIO2)
b) Intubated prior to transfer
c) Noted to be in "respiratory distress" or "respiratory failure"
d) Airway obstruction with stridor
e) Respiratory acidosis (pH <7.30 with pCO2 >50 mg Hg)

Infections

a) Meningitis (suspected or diagnosed)
b) Sepsis (suspected or diagnosed)
c) Active infection in an immunocompromised hosts. (cancer patients, leukemia, AIDS)

Shock

a) BP <100, with tachycardia (P≥100), and/or other findings of hypoperfusion: (i.e., decreased mentation, cool extremities, dusky extremities, evidence of organ dysfunction)

Metabolic

a) Diabetic ketroacidosis (pH <7.30, HCO3 <14 mmol/l with glucose >200 mg/dl and ketonuria)
b) Hypoglycemia (BG <40 mg/dl with mental status change)
c) Hyperglycemia: (BG >800 mg/dl)
d) Potassium ≤3.0 or ≥6.0 meq/L
e) Acidosis (pH <7.30) (any cause)
f) Drug toxicity (Digoxin level >2.0 ng/ml, theophylline >20.0 mg/ml Phenytoin level >30.0 mg/ml, Lithium >2.0 mmol/l, drug screen positive of tricyclic antidepressants with suspicion of overdose)
g) Sodium >150 meq/l or <125 meq/l

Neurologic

a) Altered mental status (lethargic, confused, comatose, unable to answer questions)
b) New focal neurologic deficit(s) (all causes)

Ob/Gyn

a) Suspected ectopic pregnancy with pain and/or bleeding

Hematology

a) Severe anemia (hematocrit <25% with evidence of acute blood loss per nasogastric tube or hematemesis, rectal bleeding, and/or vaginal bleeding)
b) Required blood transfusion during or prior to transport
c) Active blood loss upon arrival of >500 cc with evidence of shock regardless of hematocrit

Gastroenterology

a) Active upper/lower GI bleeding
b) Possible acute abdomen (abdominal tenderness, with signs of acute peritonitis) or pancreatitis

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Serious Poisonings among Older Adults: A Study of Hospitalization and Mortality Rates in Massachusetts 1983–85

ALAN WOOLF, MD, MPH, SUSAN FISH, PHARMD, CAREY AZZARA, MA, AND DEBORAH DEAN, MSPH

Abstract: We analyzed poisoning-related hospitalization and mortality rates among older adults living in Massachusetts during 1983–85. While poisoning-related hospitalization rates of individuals ≥60 years were near the state average, death rates for men ≥70 years and for women over 60 were higher than those of younger individuals. Medications and carbon monoxide were commonly implicated agents in these poisoning deaths. Older adults are more vulnerable to death from a poisoning than are younger age groups. (*Am J Public Health* 1990; 80:867–869.)

Introduction

The incidence of serious poisonings among older individuals is poorly documented. The purpose of this study was to determine poisoning hospitalization and death rates among older people in Massachusetts, and to compare such rates with those of younger age groups in order to assess relative risk.

Methods

The Massachusetts Uniform Hospital Discharge Data Set (UHDDS) contains all hospital discharges recorded by 108 acute care institutions within the state. Data from 1983–85 were scanned for principal discharge diagnoses recorded as poisonings or adverse effects of chemicals or drugs. The International Classification of Diseases, 9th edition, Clinical Modification (ICD-9-CM) diagnosis codes used in the analysis are shown in the Appendix. Data were aggregated by five-year age intervals and sex. Death certificates for 1983–85 constituted our second data set. A death was counted as due to poisoning if either the underlying cause of death or any mentioned cause was listed as a poisoning or adverse effect of a drug or chemical. Rates were calculated based on population estimates for 1985 made by the Massachusetts Institute for Social and Economic Research.[1] Hospitalizations and deaths due to the acute or chronic effects of alcoholism were specifically excluded. Age- and sex-specific poisoning-related hospitalization rates and death rates were calculated and compared with mean statewide rates.

Results

Hospitalization

There were 17,805 hospitalizations in Massachusetts during 1983–85 in which a poisoning or the toxic effect of a drug or chemical was the principal discharge diagnosis; 2,200 of these involved persons 60 or more years of age. Almost 81 percent (N = 1778) of the hospitalizations of persons 60 or more years of age were associated with exposures to medications; household products were implicated in 301 (13.7 percent); plant exposures, animal stings or envenomations were implicated in 121 (5.5 percent). The poisoning hospitalization rates and 95% confidence intervals for both males and females in all age groups between 60–79 years were below the state average for persons under age 60 (Table 1). The poisoning hospitalization rates for males age 80 and older were higher than the state average for males under age 60.

Deaths

There were 1,116 deaths attributable to poisonings or toxic effects recorded in Massachusetts during the years 1983–85, including 136 male and 139 female deaths of persons 60 or more years of age. Death rates from poisoning for males 70 or more years of age and for females 60 or more years of age were substantially higher than the sex-specific state rates

TABLE 1—Annualized Poisoning Hospitalization Rates and Relative Risks,* Massachusetts 1983–85

Age Groups (years)	Males (Hosp/10^5/Yr)	RR* (95% CI)	Females (Hosp/10^5/Yr)	RR* (95% CI)
60–64	44	.47 (.40, .55)	58	.47 (.42, .53)
65–69	57	.61 (.52, .70)	64	.52 (.46, .58)
70–74	58	.62 (.52, .73)	74	.60 (.52, .67)
75–79	64	.68 (.56, .83)	91	.73 (.65, .83)
80–84	116	1.23 (1.01, 1.50)	102	.82 (.72, .95)
85+	112	1.19 (.94, 1.51)	96	.77 (.67, .89)
State Average (all ages)	89		114	
State Average (persons less than 60 years old)	94		124	

*RR = Relative Risk (Age Specific Rate/State Average Rate [<60 yr]). 95% confidence intervals in parentheses.

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for persons under 60 years of age with relative risks increasing directly with age (Table 2).

For 152 of the original 275 poisoning-related deaths of persons 60 or more years of age, E-coded data were available enabling assessment of intentionality (Table 3). For both males and females, drugs were the most important agent of accidental (unintentional) death. Carbon monoxide was a common cause of intentional (suicidal) poisoning-related death in males but drugs predominated among females.

Discussion

Seniors might be considered to be at a higher risk for a serious poisoning incident than adults in younger age groups for a variety of reasons. While the elderly comprise just 12 percent of the total population, they consume 31 percent of all prescription medications.[2] It is common for elders to be taking multiple medications simultaneously.[3–6] Many have underlying heart, liver, kidney, or lung diseases, any of which might complicate an overdose situation or affect the pharmacokinetics of a drug or toxin.[7–9] Also, 2–4 million older Americans suffer from dementia[10]; such patients may be confused and have difficulty complying with complicated dosage regimens. Failing eyesight and memory may be responsible for medication errors or for mistaking household products as potables. Finally, the elderly are at risk for depression[11] and may take overdoses in suicide attempts.

Previous analyses of calls to Poison Control have concluded that the poisoned elderly are more likely to be admitted to the hospital and are more likely to die from the poisoning than are younger age groups.[12,13] However small numbers of subjects, a lack of population-based denominators, and an acknowledged reporting bias raise questions regarding the validity of such findings. The current study suggests that hospitalization rates for poisoning among older adults are not very different from those of younger age groups. However, our results support the conclusion that both elderly males and females are at greater risk of poisoning-related death than younger persons. That older individuals are more vulnerable to a terminal outcome from a poisoning episode reinforces the speculation that they are more fragile to the toxic effects of drugs and poisons than are younger individuals. Also, E-coding analysis suggests suicide to be a prominent cause of poisoning death among the elderly. These two factors, vulnerability and intentionality, are important considerations for both clinicians and researchers studying poisoning in this age group.

TABLE 2—Annualized Poisoning-Related Death Rates and Relative Risks,* Massachusetts 1983–85

Age Group (years)	Males (Deaths/ 10^5/Yr)	RR* (95% CI)	Females (Deaths/ 10^5/Yr)	RR* (95% CI)
60–64	8.34	1.04 (.73, 1.48)	6.62	1.74 (1.20, 2.22)
65–69	7.45	0.93 (.62, 1.40)	7.04	1.85 (1.26, 2.70)
70–74	14.43	1.80 (1.27, 2.55)	5.68	1.49 (.95, 2.34)
75–79	13.07	1.63 (1.04, 2.55)	7.79	2.04 (1.32, 3.15)
80–84	14.00	1.75 (.99, 3.09)	6.07	1.59 (.89, 2.84)
85+	20.54	2.56 (1.48, 4.44)	13.07	3.43 (2.28, 5.16)
State Average (all ages)	9.37		5.16	
State Average (persons less than 60 years old)	8.02		3.81	

*RR = Relative Risk (Age Specific Rate/State Average [<60 yr]). 95% confidence intervals in parentheses.

TABLE 3—Intentionality and Agent of Poisoning Deaths in Massachusetts Adults 60 Years and Older, 1983–85*

Circumstance/Intentionality	Sex: Male N = 74	Sex: Female N = 78
Accidental/Unintentional		
Drugs	20	26
Carbon Monoxide	5	5
Isopropanol	1	0
Benzene	1	0
Subtotal	27	31
Indeterminate		
Drugs	3	9
Suicide/Intentional		
Drugs	9	32
Carbon Monoxide	31	6
Corrosive	1	0
Carbon Tetrachloride	1	0
Other Agents	2	0
Subtotal	44	38

*Table includes only those deaths for which E-codes were available (152 out of 275 total deaths).

ACKNOWLEDGMENTS

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APPENDIX

ICD 9-CM Diagnosis Codes Used in Dataset Analysis

960-979.9	Poisonings by drugs, medicinal and biological substances
980-989.9	Adverse effects of drugs
E850-E858.9	Accidental poisoning by drugs, medicinal substances, and biologicals
E860-E869.9	Accidental poisonings by other solid and liquid substances, gases, and vapors
E950-E952.9	Suicide and self-inflicted poisoning
E962.0	Assault by poisoning
E980-E980.5	Injury undetermined whether accidentally or purposefully inflicted by poisoning

(E coding refers to the circumstances under which poisoning or injury occurs)

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Changes in Ambulatory Surgery Utilization 1983–88: A Community-Based Analysis

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Abstract: Changes in ambulatory surgery utilization in the metropolitan area of Syracuse, New York 1983–88 were identified. Total ambulatory surgery utilization rose from 12,479 to 24,643 procedures. Approximately 69 percent of the increase was generated by freestanding surgery centers. Operations responsible for the largest additional volumes included lens procedures, certain cervical procedures, hernias, and excision of skin and breast lesions. (*Am J Public Health* 1990; 80:869–871.)

Introduction

Ambulatory surgery is usually defined as procedures which are more complicated than those carried out in physicians' offices but less complex than those which necessitate inpatient hospital stays.[1] In the United States, the availability and utilization of ambulatory surgery have increased considerably during the past two decades.[2] By 1988, 964 freestanding surgery centers were operating in the nation, and hundreds of hospitals performed selected procedures on an outpatient basis.[3,4] Between 1983 and 1988, ambulatory surgery utilization nationwide increased from approximately 5,000,000 to about 13,735,000 procedures.[4,5] It has been reported that physicians no longer have to be convinced to use ambulatory surgery, they insist on it.[6]

Changes in the health care marketplace have also encouraged the growth of ambulatory surgery. The introduction of prospective payment approaches by Medicare and other payors has focused on reduction of hospital stays. Medicare now requires that certain eye procedures be performed in outpatient settings. Reductions in inpatient utilization, brought about by these mechanisms, have encouraged hospitals to compete in a variety of settings, including ambulatory care.[2,7,8]

Evaluation of the growth of ambulatory surgery has been made difficult by the lack of consistent, longitudinal data concerning this modality.[9] The present study evaluated developments in ambulatory surgery in the metropolitan area of Syracuse, New York between 1983 and 1988 to follow up earlier reports.[10,11]

The health care system of Syracuse, New York has made extensive use of ambulatory surgery because of the limited supply of acute care beds in the area (2.9 per 1,000 population compared with 4.1/1,000 population nationally).[12,13] During 1983, four ambulatory surgery programs existed in Syracuse. One of these programs was freestanding operated by a hospital (including four operating rooms), the others were hospital-integrated (using existing institutional operating rooms and support services). Between 1983 and 1988, two additional freestanding programs were opened in Syracuse. One of these was an additional program operated by Crouse Irving Memorial Hospital, the other was established by a group of local physicians.

Methods

This study identified changes in ambulatory surgery utilization in Syracuse between 1983 and 1988 at the aggregate and procedure-specific levels. Aggregate data for ambulatory surgery were obtained from the hospitals. Procedure-specific data were collected from two hospitals, Crouse Irving Memorial Hospital and St. Joseph's Hospital Health Center, which maintained such information for both years. Inpatient data for the same procedures were obtained from New York's Statewide Planning and Research Cooperative System.

Results

Between 1980 and 1987, the rate of inpatient surgical operations per 1,000 population in the United States declined from 78.1 to 76.4.[14] The data in Table 1 demonstrate that, between 1983 and 1987, discharges of surgical patients per 1,000 population declined in all six of New York State's major metropolitan areas. The largest decline (16.0 discharges per 1,000 population or 25.4 percent of the 1983 level) was produced in Binghamton. Syracuse, which in 1983 had a rate lower than all of the other areas, was able to reduce that rate by 10 percent within a five-year period.

The data in Table 2 indicate that the total procedures performed in ambulatory surgery programs in Syracuse increased by 97.5 percent during the five-year period. This was paralleled by an estimated 150 percent increase at the national level.[8,4] The total ambulatory procedures performed in Syracuse during 1988 (24,643) exceeded the number of

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TABLE 1—Resident Surgical Discharges per 1,000 Population, New York State Metropolitan Areas, 1983–87

Metropolitan Area	Surgical Discharges per 1,000 Population	
	1983	1987
Albany (Albany County)	56.3	41.6
Binghamton (Broome County)	62.9	46.9
Buffalo (Erie County)	67.1	52.9
New York City	60.1	51.9
Rochester (Monroe County)	48.1	43.5
Syracuse (Onondaga County)	45.4	40.5

Sources: New York State Department of Commerce, Economic Development Board, Population Projections—February 14, 1983 (identified through linear interpolation of 1980 U.S. Census data and April 1985 New York State Department of Commerce Population Projections); New York Statewide Planning and Research Cooperative System.

TABLE 2—Ambulatory Surgery Utilization, Syracuse, New York, 1983–88

Facility	Ambulatory Surgery Procedures		
	1983	1988	Percentage Increase
Community-General Hospital	1,519	3,223	112.2
Crouse Irving Memorial Hospital Total	8,104	12,699	56.7
(Irving Avenue Site)	(8,104)	(8,646)	
(Madison-Irving Site)	(0)	(4,053)	
St. Joseph's Hospital Health Center	2,118	2,895	36.7
University Hospital of the SUNY Health Science Center at Syracuse	738	2,061	182.0
Harrison Center	0	3,746	—
Total	12,479	24,644	97.5

principal inpatient operations performed in hospitals (19,075).

Increased use of ambulatory surgery in Syracuse reflected developments at most programs in the community. Freestanding centers operated by Crouse Irving Memorial Hospital and Harrison Center accounted for 68.6 percent of the total increase of 12,164 procedures. Utilization of the hospital integrated programs operated by Community-General Hospital, St. Joseph's Hospital Health Center, and University Hospital also increased, but absolute changes in volume at these facilities were smaller.

Detailed diagnostic data are available for 1983 and 1988 only for procedures performed at the freestanding program operated by Crouse Irving Memorial Hospital and the hospital integrated program managed by St. Joseph's Hospital Health Center.

The freestanding model at Crouse Irving Memorial Hospital was able to generate greater volumes of individual ambulatory surgery procedures. Most of the specific procedures carried out at this hospital were performed in the freestanding model rather than on an inpatient basis (data available on request to authors). With the exception of rhinoplasties and inguinal hernia repairs, more than 90 percent of all of the procedures identified in Table 3 were performed at Crouse Irving Memorial Hospital on an outpatient basis. It was also notable that the percentage of rhinoplasties carried out on an outpatient basis increased from 28 to 81 and the percent of inguinal hernia repairs rose from 49 to 68. These procedures have proven to be useful indicators of the extent of ambulatory surgery utilization.

The hospital-integrated program at St. Joseph's was also able to increase ambulatory surgery volumes but, at the absolute level, these increases were considerably smaller than those which occurred in the freestanding program. By comparison, in 1988, approximately 80 percent of rhinoplasties and 12 percent of inguinal hernia repairs at this hospital were being performed on an outpatient basis. These trends have caused St. Joseph's Hospital Health Center to plan the development of its own freestanding program.

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TABLE 3—Ambulatory Surgery Utilization* for Selected Procedures, Crouse Irving Memorial Hospital, St. Joseph's Hospital Health Center, 1983–88

Procedure	Crouse Irving Memorial Hospital		St. Joseph's Hospital Health Center	
	1983	1988	1983	1988
Neurological, Eye, Otolaryngologic				
Carpal Tunnel Release (04.43)	194	262	70	170
Lens Extraction (13.1–13.6)	207	2,238	78	355
Rhinoplasty (21.8)	34	90	79	38
Tonsillectomy, Adenoidectomy (28.2–28.3)	495	582	262	137
Gynecological				
Bilateral Destruction of Fallopian Tubes (66.2)	489	489	0	0
D&C other than Termination of Pregnancy (69.02–69.09)	1,309	1,738	214	273
Excision of Lesion of Vulva, Perineum (71.0–71.11, 71.2–71.4)	65	150	11	20
Other Procedures				
Repair of Inguinal Hernia (53.0–53.1)	240	421	26	35
Incision and Division of Muscle, Tendon, Fascia, Bursa (82.0, 83.0–83.1)	100	78	73	18
Local Excision or Destruction of Breast Lesion (85.11–85.12, 85.20–85.23)	114	474	55	218
Local Excision or Destruction of Lesion Skin, Subcutaneous Tissue (86.11, 86.3)	402	608	78	120
Laparoscopy Except for Destruction, Fallopian Tubes (54.21)	112	597	28	23

*Indicates percent of hospital total (inpatient and outpatient) performed on an outpatient basis.

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Hip Fracture Incidence among the Old and Very Old: A Population-Based Study of 745,435 Cases

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Abstract: Data were obtained from the Health Care Financing Administration and the Department of Veterans Affairs (formerly called Veterans Administration) on all hospital discharges among the elderly population from 1984 through 1987 and combined with census estimates to calculate incidence rates of hip fracture for the elderly population of the United States. Rates for White women were the highest, reaching 35.4 per 1,000 per year among 95 year-olds. Comparably, White men, Black women, and Black men experienced similar age-related increases in risk, although of less magnitude and relatively less rate of change, respectively. (*Am J Public Health* 1990; 80:871–873.)

Introduction

Hip fractures are an important cause of the morbidity and mortality experienced by the elderly population in the United States.[1] Despite this, there have been relatively few national population-based studies of the incidence of hip fracture in this country. Of those previous studies, most have produced unstable estimates of incidence rates for non-Whites (due to small sample sizes)[2,3] or the oldest old (over 85 years of age).[2–7] The present study uses newly available data from the Health Care Financing Administration (HCFA) and the Department of Veterans Affairs (VA) to provide detailed estimates of the incidence of hip fracture in the elderly by age, race, and sex.

Address reprint requests to Steven J. Jacobsen, PhD, Division of Biostatistics/Clinical Epidemiology, Medical College of Wisconsin, P.O. Box 26509, Milwaukee, WI 53226. This work was done while Dr. Jacobsen (currently a third-year medical student at Medical College of Wisconsin) was completing the doctoral requirements in the Epidemiology Program, School of Public Health, University of Illinois at Chicago. Drs. Goldberg and Miles are both with the Epidemiology/Biostatistics Program there, and Dr. Brody is Dean of the School of Public Health, University of Illinois, Chicago; Dr. Stiers is with Health Services Research and Development, VA Hospital, Hines, IL; Dr. Rimm is Professor and Head, Division of Biostatistics/Clinical Epidemiology, Medical College of Wisconsin, Milwaukee. This paper, submitted to the Journal June 19, 1989, was revised and accepted for publication October 17, 1989.

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Methods

Data were obtained from HCFA on all 40 million short-stay hospital discharges for the calendar years 1984 through 1987 and 4 million discharges from VA hospitals. HCFA requires all institutional providers to submit a uniform billing form that contains the following data: age, race, sex, a maximum of five ICD9-CM diagnostic categories, date of admission and discharge, and a unique patient identifier. VA maintains a similar database, keyed by the same patient identifier.

Cases of hip fracture were identified by scanning the 44 million discharge records for a discharge diagnosis of fracture of the hip (ICD9-CM 820.0 through 820.9) in any of the five diagnostic fields. Patients were excluded if: 1) age at admission was less than 65 years; 2) the fracture could have been attributed to a neoplastic process; 3) the patient had been discharged previously for fracture of the hip in the four-year period; 4) the first discharge diagnosis was for late effects of fracture; or 5) the patient resided outside of Puerto Rico or the United States proper. The remaining cases were considered to be first fractures of the hip and thus comprised the numerator for the calculation of rates.

Denominator information was obtained from the Bureau of the Census for 1985. Incidence rates were calculated as the average annual age-, race-, and sex-specific number of cases divided by the population at risk. Rates were adjusted for one-year age groups using the direct method, with the entire United States population aged 65 years and older serving as the standard.[8]

Results

During the period of 1984 through 1987, 810,949 hospital discharges for fracture of the hip were recorded by HCFA and 11,435 were recorded by VA for persons over 65 years of age. Of these, 54,055 were second fractures or rehospitalizations for a previous hip fracture. An additional 19,899 cases were persons under the age of 65 from the HCFA database, and 4,504 cases were discharged for late effects or attributable to neoplastic processes. The final number of eligible incident cases of first hip fracture was 745,435. (Some cases were excluded for more than one reason.) Women accounted

for 580,129 of these fractures (79 percent) whereas men accounted for only 165,306 (21 percent) (Table 1). The great majority of the cases were among Whites (93 percent), while Blacks and those of other or unknown racial background each accounted for 3 percent.

The incidence of hip fracture among the elderly for this time period was 6.63 fractures per 1,000 per year. Adjusting incidence rates for age, White women experienced the highest incidence rates at 8.07 per 1,000, followed by White men at 4.28/1,000, Black women at 3.06/1,000, and Black men at 2.38/1,000. Examining incidence rates by one year age groups (Figure 1) shows that rates for White women increased exponentially from 1.63/1,000 in 65 year-olds, to 35.4/1,000 in women aged 95 years. A similar exponential pattern of increase was observed for White males, with rates increasing from 0.9/1,000 for 65-year-olds to 26.0/1,000 for 96-year-olds. The age-associated increase in incidence persisted in Blacks, but was less than exponential in women and nearly linear in men.

Discussion

The risk of fracture of the hip increased with age well into the tenth decade of life for women and men, and Blacks and Whites alike. Incidence rates for White women between the ages of 85 and 95 were especially high, with more than 3 percent of this population suffering a fracture of the hip. White men, Black women, and Black men also suffered from high rates of injury, with rates reaching nearly 3, 2, and 1 percent of the nonagenarians, respectively.

The rates for White men and women calculated in this study resemble those reported from the Scandinavian countries,[9–11] except that rates in the oldest women appear to be higher in Sweden.[11] Estimates from Rochester, Minnesota appear to underestimate rates for both men and women over the age of 80 years,[5–7] but this may be due to small sample sizes. It is noteworthy that our results substantiate prior national estimates using data from the National Hospital Discharge Survey for all race and age groups,[2,4,12] despite the previously noted problems with similar data.[13]

TABLE 1—Demographic Characteristics of Elderly Hip Fracture Patients Medicare and VA Population: 1984–87

	Sex		
	Women N (%)	Men N (%)	Total N (%)
All Races	580,129	165,306	745,435
Blacks	17,143	7,809	24,952
65–74	3,424 (20.0)*	2,697 (34.5)*	6,121 (24.5)†
75–84	6,819 (39.8)	3,063 (39.2)	9,882 (39.6)
85–94	5,966 (34.8)	1,820 (23.3)	7,786 (31.2)
95+	934 (5.4)	229 (2.9)	1,163 (4.7)
Whites	544,100	151,872	695,972
65–74	103,150 (19.0)	40,323 (26.6)	143,473 (20.6)
75–84	233,140 (42.8)	64,344 (42.4)	297,484 (42.7)
85–94	189,548 (34.8)	42,978 (28.3)	232,526 (33.4)
95+	18,262 (3.4)	4,227 (2.8)	22,489 (3.2)
Other	3,740	1,801	5,541
65–74	1,131 (30.2)	548 (30.4)	1,679 (30.3)
75–84	1,319 (35.3)	725 (40.3)	2,044 (36.9)
85–94	1,118 (29.9)	473 (26.3)	1,591 (28.7)
95+	172 (4.6)	55 (3.1)	227 (5.2)
Unknown	15,146	3,824	18,970
65–74	3,375 (22.3)	1,211 (31.7)	4,586 (24.2)
75–84	6,272 (41.4)	1,766 (46.2)	8,038 (42.4)
85–94	4,560 (30.1)	736 (19.3)	5,296 (27.9)
95+	939 (6.2)	111 (2.9)	1,050 (5.5)

*Percent of Race- and Sex-Specific Category.
†Percent of Race-Specific Category.

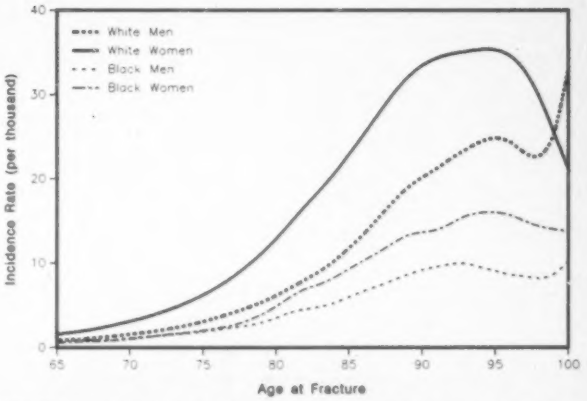

FIGURE 1—Annual Age-Specific Incidence of Hip Fracture among the Elderly: Population-based Rates by Race and Sex for 1984–87, United States
Lines represent a smoothed curve based on the calculated age-, race-, and sex-specific incidence rates.
SOURCES: Health Care Financing Administration, Department of Veterans Affairs, US Bureau of Census.

There are several limitations in these data. The assumption that all fractures of the hip necessarily result in hospitalization is probably valid, because hip fractures are associated with a great deal of pain and incapacity; but persons who did not survive long enough to be hospitalized would be missed. A second assumption is that persons hospitalized in this age range are covered by the Medicare insurance program or cared for in VA hospitals. According to the Health Care Financing Administration,[14] 97 percent of the United States population ages 65 and older is enrolled in the Medicare program. The number of cases treated in VA hospitals appears to account for the vast majority of the remaining 3 percent, suggesting these rates reflect true population rates.

These data may over-estimate the risk of an incident first fracture since data are only available for up to four years of follow-up. The rates presented herein may represent up to a 6 percent over-estimate of the risk of a first fracture. Likewise, the use of these data to estimate the prevalence of fracture would result in an underestimate for the same reason.

The apparent decline in fracture incidence after age 95 among white women is intriguing. Possibly, women who are at risk of fracture have been removed from the population through fracture-related mortality or competing causes. It is noteworthy that this decline persists when all fractures during the four years of follow-up are included in the numerator. Alternatively, this result may reflect some degree of non-sampling error inherent to the data. Only more detailed surveillance of an elderly population will provide the opportunity to ascertain the meaning of the decline.

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Swaddling and Acute Respiratory Infections

KADRIYE YURDAKOK, MD, TUNA YAVUZ, MD, AND CARL E. TAYLOR, MD, DRPH

Abstract: In Turkey and China the ancient practice of swaddling is still commonly practiced. Both countries have extremely high rates of pneumonia, especially during the neonatal period. Preliminary evidence on the possibility that swaddling may interfere with normal respiratory function and thereby predispose to pneumonia was gathered in a teaching health center in Ankara. Babies who had been swaddled for at least three months were four times more likely to have developed pneumonia (confirmed radiologically) and upper respiratory infections than babies who were unswaddled. These preliminary findings were highly significant and are being followed up by further studies. (*Am J Public Health* 1990; 80:873–875.)

Introduction

The ancient practice of swaddling has almost disappeared in most countries of the world. A national sample survey in Turkey, however, showed that 93 percent of mothers swaddle their children.[1] In China also, most babies are tightly swaddled from birth through the first several months of life.[2] Since these two countries include over one-fifth of the children of the world, the number of swaddled babies is substantial.

In both countries pneumonia is the first cause of death among children, with particularly high incidence among neonates. In Turkey about 50,000 infant deaths occur annually due to pneumonia.[3] In China over 300,000 child deaths per year are attributed to pneumonia with a child mortality rate twice as high as the second highest cause of death.[4] Forty percent of these deaths are under one month of age.

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In trying to explain this extremely high incidence of pneumonia we focused on the possibility that swaddling might interfere with normal respiratory function and lung expansion. No studies have been published of possible relationships between swaddling and acute respiratory infections or pneumonia.

The common cultural practice is that immediately after birth babies are tightly bound in layers of cloth. Complete swaddling immobilizes the baby from the neck to the feet. The legs are pressed firmly together with the knees straight and the arms are bound to the sides or slightly to the front of the body. The layers of cloth are not only pulled tightly but they are also securely tied to minimize body movement. In partial swaddling cloth is wrapped around the legs and torso up to the armpits, but the arms are free. In both types of swaddling the child may also be covered with netting or a blanket to prevent exposure to flies, drafts or cold air. Babies are almost always laid on their backs and kept in a dark room to induce sleep.[5] Swaddled babies seldom cry and respirations seem shallow to an observer, raising the question of whether full expansion of the lungs occurs. A variety of devices are used to dispose of excreta in Turkey but in the urban area where this study was done families use cloth diapers.

Methods

The records from Gulveren Health Center in a suburb of Ankara were examined to look for associations between swaddling and selected health problems. This teaching health center of Hacettepe Medical School has high standards of follow-up and care of all the children in the health center area. Records were available on 186 infants, of whom 94 had been unswaddled, 29 had been partially swaddled, and 63 had been completely swaddled. The rate of swaddling is lower than the national figure because this study included only those babies who were swaddled for at least three months.

All infants were examined and detailed histories were taken. The ages of the children at the time of this cross-sectional study ranged from three to 12 months, with a mean of 6.8 months. Sex ratios were similar in all groups as shown

in Table 1. The mothers ranged in age between 21 and 25 years and literacy levels in the three groups were 84, 88, and 89 percent. The average number of people in each household was 5.0 in the unswaddled group, 4.81 in partially swaddled, and 5.47 in the completely swaddled group. Families were all from the same socioeconomic levels with most fathers employed in industrial plants in Ankara and approximately 11 percent unemployed in each group.

Body weights and developmental stages were similar in all groups. The history included careful questioning about respiratory infections; having more than two infections during the period up to examination was classified as frequent upper respiratory infections. Physical examination included observation for signs of rickets and congenital dislocation of the hip.

All children had been followed at the health center by midwives with high compliance for regularly scheduled clinic and home visits. If illness occurred, babies were usually brought to the health center. At the time of regularly scheduled visits, parents were also asked about illnesses treated elsewhere. All of this information was filled in on follow-up records. If the baby had special tests at a hospital, the laboratory and x-ray records were obtained and transferred to the follow-up cards. History of respiratory infections was included in this analysis only if the diagnosis had been recorded on the baby's follow-up records. A diagnosis of pneumonia was made only when clinical records showed cough, fever, and crepitations. All pneumonia cases had positive x-rays at the time of illness which were reviewed again as part of this study. Some had respiratory distress but none required hospitalization.

Results

Table 1 shows the frequency of respiratory illnesses identified in these infants. Pneumonia and a history of at least two upper respiratory infections were each about four times more common in swaddled than unswaddled babies. The association with partial swaddling was as strong as with complete swaddling and these groups were combined for statistical testing.

Discussion

Few studies have been done of the effects of swaddling. Lipton,[6] *et al*, reported that swaddling reduces motor activity and encourages the baby to sleep. Chisholm[7] confirmed these findings about motor activity and sleep and also reported less response to stimulation and less variation in heart rate. A report from Beijing[8] compared transcutaneous oxygen tension in 40 neonates with moderate pneumonia, confirmed radiologically, who were observed for periods when they were swaddled and unswaddled and placed in prone and supine positions. Measurements were made 30 minutes after feeding when the infant was asleep. Unswaddled babies in the prone position had oxygen tension 18 percent higher than swaddled supine babies and 12 percent higher than unswaddled supine babies. Several studies have shown that babies in the prone position have improved respiratory function when compared with babies in the supine position.[9,10] It may be that a combination of swaddling and the supine position produces the greatest effect on respiratory ventilation.

Rickets was more common in swaddled babies than in unswaddled babies presumably because of lack of exposure to sunlight. In Israel, Block reported an association between swaddling and congenital dislocation of the hip[11] and similar results were reported among Navajo babies bound to cradle boards.[12] No such association was found in this series of cases.

The extremely high mortality and morbidity from neonatal and infant pneumonia in China and Turkey is being further investigated by prospective studies which will provide more precise information on time relationships between swaddling and pneumonia. If a causal relationship can be defined, educational efforts can try to modify this traditional practice. The possible advantages of swaddling should also be studied since parents say that a swaddled baby child is easy to care for because it is quiet and sleeping most of the time.

TABLE 1—Swaddling, Pneumonia and Respiratory Infections in Turkey

	Unswaddled (n:94)	Partially Swaddled (n:29)	Completely Swaddled (n:63)
Age (months)	7.9 ± 2.3	8.1 ± 3.3	7.4 ± 2.1
Male/Female	1.08	1.07	1.11
Number in Household	5.0	4.81	5.47
% Literacy of Mothers	84	88	89
	n %	*n* %	*n* %
Pneumonia*	3 3.2	7 24.1	8 11.6
Frequent URI*	3 3.2	4 13.8	10 14.5

*Fisher's exact test used to compare unswaddled with both groups of swaddled babies added together: for pneumonia p = .002 and for frequent upper respiratory infections p = .004.

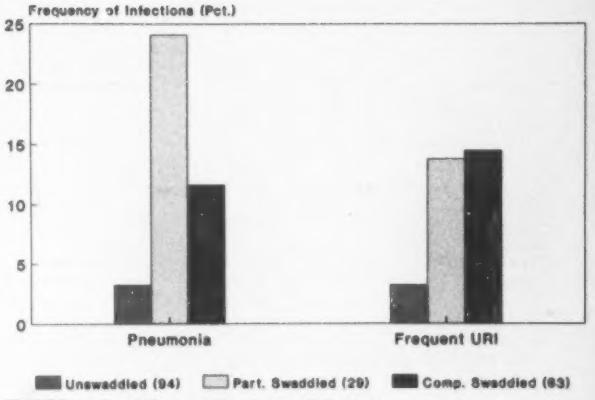

FIGURE 1—Swaddling and Respiratory Infections.

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Assessment of AIDS Knowledge, Attitudes, Behaviors, and Risk Level of Northwestern American Indians

ROBERTA L. HALL, PHD, DONI WILDER, BS, PAMELA BODENROEDER, BA, AND MICHAEL HESS, DDS

Abstract: A survey was made of 710 American Indians of Oregon, Washington, and Idaho to assess the population's knowledge, attitudes, and behaviors in respect to acquired immunodeficiency syndrome (AIDS), to estimate the population's risk, and to plan strategies to reduce it. In contrast to 3 percent of the general population, this study found 10.6 percent of male and 6.4 percent of female Pacific Northwestern American Indians in groups considered at high risk for AIDS. (*Am J Public Health* 1990; 80:875–877.)

Introduction

Assessment of the degree to which any population is at risk for infection with the human immunodeficiency virus (HIV) is difficult. The American Indian/Alaskan Native population presents a special challenge because of its heterogenous cultural roots and its dispersion within the general population. Few Native Americans who live on reservations have been included in the National Health Interview Survey (NHIS), which studies the public's knowledge and attitudes toward acquired immunodeficiency syndrome (AIDS) (Deborah Dawson, National Center for Health Statistics, personal communication, July 14, 1989).

No census exists of persons with Native American ancestry; many in rural or reservation areas do not have telephones, and are not represented in telephone surveys. Unpublished AIDS knowledge, attitudes and behavior studies include data from women clients in a WIC (Women, Infants and Children) program; a survey of health care workers in Arizona; a survey of junior college students who primarily have Indian ancestry; and a survey of health service workers who work primarily with American Indians in the San Diego, California area (Steven Helgerson, Indian Health Service, personal communication, November 7, 1989).

Current literature reports a low rate of known AIDS cases in the American Indian population, especially in comparison to other minority groups,[1–3] but data on HIV infection do not exist and current information could understate risk in this relatively small and vulnerable population. In order to assess risk and recommend appropriate educational and preventive initiatives, the Northwest Portland Area Indian Health Board conducted an extensive survey of AIDS knowledge, attitudes, and behaviors among American Indians who live in the Pacific Northwest states of Oregon, Washington, and Idaho.

Methods

The questionnaire* was adapted from an Indian Health Service form, which included many of the items on the NHIS.[4] Questions probed respondents' knowledge about AIDS, attitudes toward people with AIDS, and behaviors related to HIV transmission. The questionnaire included a number of questions on sexual behavior (number and sex of partners, degree of acquaintance with partners, age at first sexual intercourse, etc.) and on the use of various licit and illicit drugs, including alcohol. Demographic questions asked respondents to identify their age, sex, ethnic affiliation, and education, and to categorize their residence by on or off reservation, community type and size, and state and county.

The survey was administered from September 1988 through March 1989 at 24 Indian centers in Oregon, Idaho, and Washington either by a member of the staff of the Northwest Portland Area Indian Health Board or by staff at the participating agency. Sixty-six percent of respondents were surveyed at health clinics or health stations, 17 percent at tribal offices, and 17 percent at Indian educational or community agencies. Respondents include employees, persons conducting tribal business, persons seeking health care, and family members accompanying them. All respondents were asked to complete the questionnaire; anonymity was guaranteed and individuals were assured that they would not be denied any tribal service if they chose not to answer the survey, or any part of the survey.

The sample consisted of 710 persons from ages 12 to 78 with a median age of 33 years; 237, 283, and 190 respondents were from Oregon, Washington and Idaho, respectively. Women represented 73 percent of the sample (Table 1). Idaho had the largest percentage resident on a reservation (85 percent), Washington was intermediate (69 percent), and Oregon was lowest (43 percent). This percentage did not differ by sex, but fewer of the age group under age 30 resided on reservations (59 percent) than in the age group over 50 (71 percent), while the age group from 30 to 49 was intermediate (65 percent).

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*For a copy of the questionnaire or a tabulation of responses, please write to the NW Portland Area Indian Health board, 520 SW Harrison, Suite 440, Portland, OR 97201.

TABLE 1—Sample Distribution by Age, Sex, and Risk Group*

	Age Groups (years)			
	12–17	18–29	30–49	50–78
Sex	N (%)	N (%)	N (%)	N (%)
Male	21 (41.2)	49 (23.9)	73 (24.7)	33 (31.1)
Female	30 (58.8)	156 (76.1)	222 (75.3)	73 (68.9)
Total	51 (7.8)	205 (31.2)	295 (44.9)	106 (16.1)
Risk Groups	8 (22.9)	93 (48.7)	187 (66.1)	93 (90.3)
Middle	25 (71.4)	78 (40.8)	74 (26.1)	8 (7.8)
High	2 (5.7)	20 (10.5)	22 (7.8)	2 (1.9)
Total	35 (5.7)	191 (31.2)	283 (46.2)	103 (16.8)

*Fifty-three persons (7%) did not provide their age; of these, 17 also did not provide information on sexual behaviors; an additional 45 (9% altogether) indicated their age but not their sexual behavior. Percentages are based on those responding to specific questions.

Since there was no selection among persons who visit Indian agencies to use tribal services, the sample is believed to represent that population well; those who do not use these services, such as homeless individuals and those on Skid Road, may be underrepresented. The relatively large number of women in the sample is probably due to their greater use of tribal services during business hours. To control for sex and age and to allow comparison among states and with sex and age groups surveyed in the NHIS, all responses were tabulated by sex, age, and state.[5]

Six males who had sexual relations with other males and 12 males and 30 females who have taken drugs intravenously were designated as members of the high-risk group, while middle-risk group members consisted of those who had two or more sexual partners in the past year or who had been involved with a person he/she did not know well. The low-risk group consisted of all others after exclusion of 62 persons who did not answer any of the questions about sexual behavior.

Results

Of the classifiable respondents, 7 percent were in the high-risk group, 30 percent were in the middle-risk group, and 63 percent were in the low-risk group (Table 1). Even if all 62 persons who did not answer sexual behavior/drug use questions were considered in the low-risk category, the proportion of high-risk respondents still would be considerably higher than that in other samples, i.e., 6.8 percent in this sample compared to 2 percent in the Hispanic population,[6] 4 percent in the Black population,[7] and 3 percent in the general population.[4]

The knowledge level of the sample does not differ markedly from that of the general population as reported by the NHIS for December 1988,[4] (Table 2). Other unpublished surveys in selected Indian populations have produced similar results in terms of knowledge (Steven Helgerson, personal communication, November 7, 1989). As has been found in other samples, respondents' knowledge is better about specific clinical properties of the disease than knowledge concerning transmission.[1,8]

Discussion

The American Indian population of the Pacific Northwest is at potential risk for high rates of HIV infection not only because of the relatively large numbers in high-risk groups but because of the large numbers of youth in the middle-risk group and the small size of many of its 39 tribes. The middle-risk group is predominantly young and male (42.9 percent of males as opposed to 25.3 percent of females). Use of alcohol and illicit drugs is much higher among middle-risk

TABLE 2—Percentage of Correct Answers to Selected Knowledge Questions by NHIS and Northwest Indian Samples

	NHIS December 1988[4]		Northwest Indian Sample				
Questions	Male	Female	Male	Female	Low-Risk	Medium-Risk	High-Risk
1. AIDS is a disease caused by a virus[a]	85	79	83	78	78	80	77
2. Any person with the AIDS virus can pass it on to someone else during sexual intercourse.[a]	95	96	95	96	97	91	98
3. A pregnant woman who has AIDS can give AIDS to her baby.[a]	94	95	89	94	94	91	90
4. How likely do you think it is that a person will get AIDS or the AIDS virus from the following?							
A. Eating in a restaurant where the cook has AIDS.[b]	45	48	45	46	45	45	34
B. Working near someone with AIDS.[b]	67	66	66	65	66	62	60
C. Being coughed or sneezed on by someone with AIDS.[b]	42	43	43	37	40	35	34

[a]Definitely true or probably true were scored as correct.
[b]Very unlikely or definitely not possible were scored as correct.

group members than among those in the low-risk group.[5] Middle-risk group members reported their first sexual experience at a younger age than those in either the high- or low-risk group, and women in the middle- and high-risk group who have had pregnancies were younger at the time of their first pregnancy than those in the low-risk group.[5] Finally, over half of the middle-risk group, compared to 13 percent of low-risk and 41 percent of high-risk groups, reported having sexual partners from both reservation and non-reservation communities. If introduced into one of the small reservation communities, HIV could decimate the population.

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Steady Increase Reported in Health Personnel; Shortages Persist among Nurses, Minorities

Despite recent declines in the numbers of health professions school graduates, the United States supply of active health personnel has risen steadily since 1970, and ratios of health personnel to population are currently at their highest levels, according to a recent report from the Health Resources and Services Administration (HRSA). In submitting the report to the Congress, Health and Human Services Secretary Louis W. Sullivan warned that, "While the numbers generally show that we have a strong foundation to meet personnel requirements through the end of the century, they also reveal problems that will continue to demand our attention." Highlights of the report include the following:

- Black and Hispanic physicians constitute only 3 percent each of the total physician supply, while Blacks account for 12 percent of the total US population and Hispanics for 7 percent. First-year and total enrollments of minorities in health professions schools have not changed significantly in recent years, and Black enrollment has shown less increase than other minority groups.
- The smaller graduating classes and continuing exits from the health work force are expected to impact on the field between the years 2000 and 2020. Growth in the supply of many health professions, such as nursing and dentistry, is expected to slow.
- A national shortage of registered nurses exists despite growth in the supply from 750,000 in 1970 to more than 1.6 million in 1988. Although the active supply will continue to increase over the next 15 years, the present shortage evokes concern about projections of a smaller supply of nurses in the future.
- More primary care practitioners are needed to provide services for patients in their initial contact with the health care system, and to address rural and inner-city health problems as well as promote health enhancement and illness prevention.
- Recent studies and surveys also reveal shortages of personnel in public health, allied health, geriatrics and gerontology, the report noted.

More information on the report, *Seventh Report to the President and Congress of Health Personnel in the United States*, is availabie from the Office of Data Analysis and Management, Bureau of Health Professions, Room 8-47, HRSA, 5600 Fishers Lane, Rockville, MD 20857. Tel: 301/443-6936.

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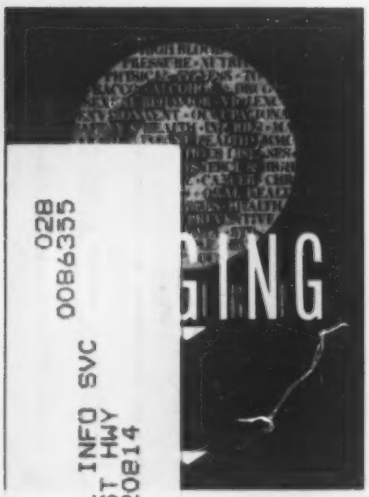

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The Federal Environmental Health Workforce in the United States

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Abstract: This paper summarizes existing data on the size and composition of the federal environmental health workforce, delineates the major categories of activities carried out by its members, identifies current and emerging issues that are likely to affect workforce activities, and makes qualitative inferences about future trends and directions. Findings suggest that there is a current and future need for more and better qualified professionals in the federal environmental health workforce. (*Am J Public Health* 1990; 80:913–920.)

Introduction

The United States federal government has responsibility for protecting the public from the adverse consequences of exposure to potentially harmful environmental agents. The importance of the nation's environmental health has been recognized during the past 20 years, as evidenced by the passage of more than a dozen major environmental laws and subsequent amendments, including the: Clear Air Act; Safe Drinking Water Act; Toxic Substances Control Act; Federal Insecticide, Fungicide, and Rodenticide Act; Resource Conservation and Recovery Act; and Comprehensive Environmental Response and Compensation Act ("Superfund").

The federal government is the single largest employer of environmental health professionals in the United States. Although the Environmental Protection Agency (EPA) has the lead role in administering and enforcing many environmental laws, other federal agencies, such as the Public Health Service (PHS) and the Occupational Safety and Health Administration (OSHA), also have direct statutory authority or involvement in environmental health issues. Moreover, many environmental health professionals are employed by federal agencies whose primary mission is not related directly to the protection of environmental health (e.g., the Department of Defense). Major Executive Branch agencies with environmental responsibilities are shown in Figure 1.

Attempts to evaluate the environmental health workforce, federal and otherwise, are a relatively recent development.[1–6] Owing to a variety of factors, there is little consensus about its size and composition. Among the problems are the absence of a generally accepted definition for environmental health, disagreement about the discrete specialties that comprise the workforce, the diversity of job titles within the field, the wide variety of responsibilities and programs in environmental health, differing employers, and the varied professional/technical/administrative skills required in different environmental health settings.[1–2]

Our objective is to examine the federal environmental health workforce by using available information and to draw qualitative inferences about current and future directions. It is important to note that we specifically exclude members of the Armed Services from our analysis. Although there are certainly a substantial number of environmental health professionals contained therein, obtaining objective data on their number and function is problematic.

The Existing Skills Mix

The federal environmental health workforce is defined broadly to include all civilian members of the federal government and the Commissioned Corps of the Public Health Service, whose primary duties and responsibilities are the identification, assessment, management, communication, or remediation of human health risks that result from environmental factors. This definition encompasses a diverse group of professionals, trained in a multitude of academic disciplines, and focusing on a wide variety of issues. Among the myriad of problems addressed by the environmental health workforce are air quality, water quality, food protection, vector control, waste management and control, consumer protection, noise control, and general environmental health issues, including direct and indirect effects on human health from ecological changes.

Within the federal government, all civilian employees who are not members of the Senior Executive Service (SES) are assigned to a particular civil service job classification series. Many of the job classifications are broadly defined. Thus, a person classified as a Public Health Program Specialist in one agency may have vastly different training, educational background, work experience, and duties and responsibilities than a person with the same classification in another agency, or even in another division within the same agency. The Environmental Protection Specialist classification, for example, which is used to the greatest extent by EPA, is defined so broadly that almost any environmental health professional, and some non-health related professionals, can be so classified.

Toxicology and epidemiology are two academic disciplines that are traditionally considered to be an integral part of the environmental health workforce. Yet until recently, neither specialty was classified as a separate job category within the federal government. Although EPA and other agencies are beginning to reclassify toxicologists into the newly created toxicology series, many are still categorized as Medical Officers, Chemists, Mathematical Statisticians, Environmental Engineers, or General Biological Scientists. Epidemiologists still do not have a separate job title and are classified in a wide variety of series, which vary by agency.

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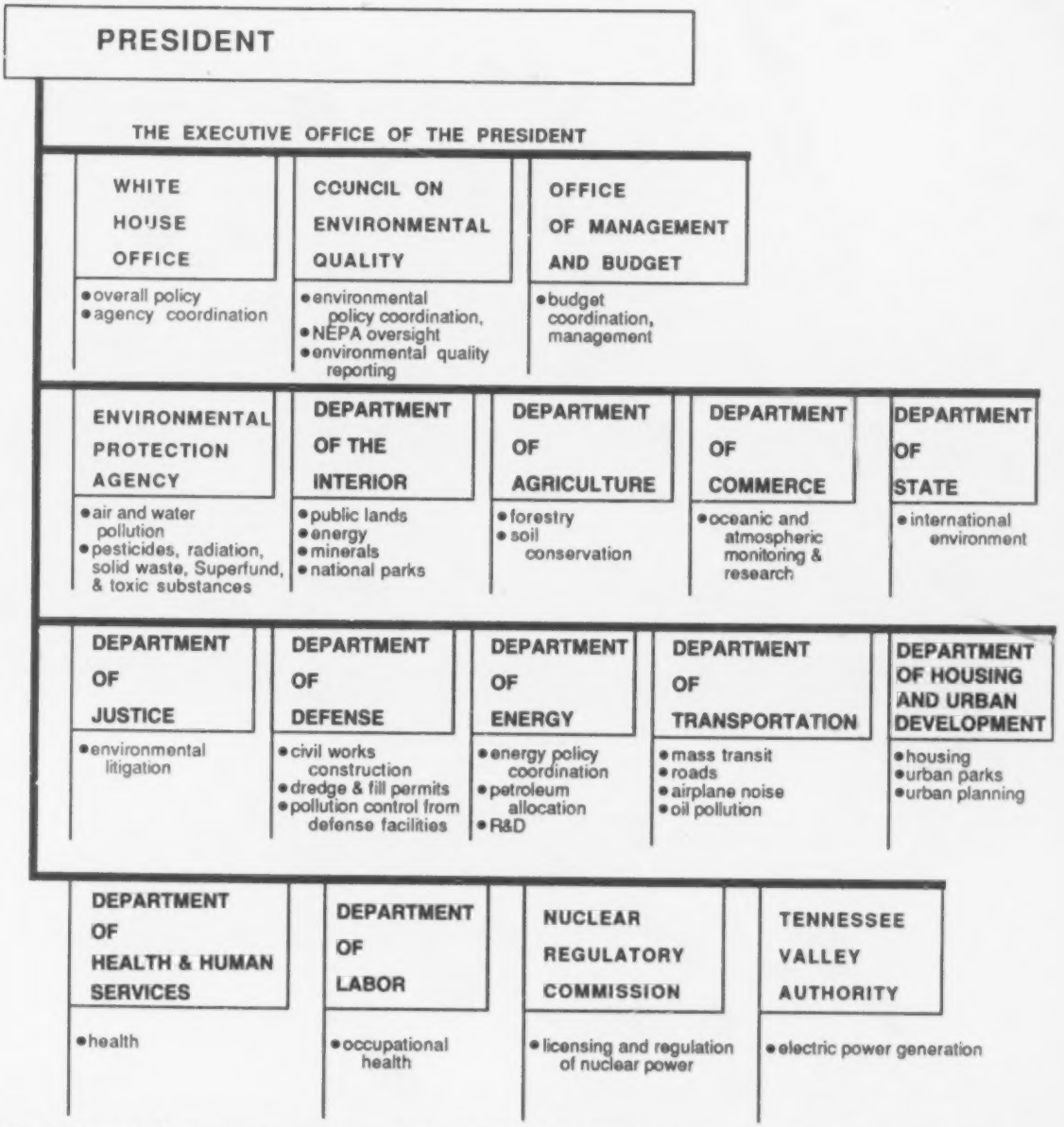

FIGURE 1—Major Executive Branch Agencies with Environmental Responsibilities, United States (adapted from ref. 16).

Keeping these caveats in mind, we used the Civil Service job classifications to examine the current federal environmental health workforce. Data given in Table 1 provide a summary for eight executive branch agencies of the number of civilian employees who are listed in job classifications that we believe are most directly related to environmental health. An indication of their collective contribution to the total number of employees in the selected job categories from across the entire federal government can be inferred from percentage figures in the last column of the table.

These data suggest that there are a substantial number of environmental health jobs in federal agencies whose primary mission is not protection of public health (e.g., Department of Agriculture, Department of Defense), and that, of the 27 job categories listed, most employees (75 percent) fall into one of eight job classifications: Medical Officer, Biological Technician, General Biological Science, Safety Management, Veterinary Medical Science, Environmental Engineer, Microbiology, and Environmental Protection Specialist.

In the entire federal government, the total number of full-time, white collar, civilian workers (excluding the Postal Service) was 1,602,438 as of October 1985. In the same year, the total for EPA, OSHA, and the PHS (three agencies directly involved with environmental health issues) was 60,298. The total number of employees in the 27 environmental health-related job classifications was 28,919 for the

TABLE 1—Workforce Statistics for Environmental Health-Related Job Classifications in Eight US Federal Agencies

Civil Service Job Classification	Number of Employees[a]									
	EPA	Defense[b]	HHS[c]	USDA	Interior	Energy	Labor	State	Total[d]	(%)[e]
Safety Management	12	1975	41	41	113	53	840	4	3079	(87)
Safety Technician	2	123	6	10	14	1	0	0	156	(76)
Environmental Protection Specialist	1162	161	0	12	183	75	0	0	1593	(88)
Environmental Protection Assistant	171	29	0	0	5	0	0	0	205	(97)
General Biological Science	574	799	879	607	1328	27	0	2	4216	(89)
Microbiology	79	290	885	313	20	1	0	0	1588	(86)
Biological Technician	56	545	700	2231	742	3	0	0	4277	(74)
Pharmacology	44	57	280	12	1	0	0	0	394	(90)
Physiology	10	128	95	72	16	0	0	0	321	(71)
Toxicology[f]	0	0	0	0	0	0	0	0	0[f]	—
Genetics	6	2	51	208	2	1	0	0	270	(96)
Biological Science Student Trainee	5	28	10	300	44	0	0	0	387	(97)
General Health Science	58	61	721	0	0	2	15	0	857	(50)
Medical Officer	6	761	1542[g]	2	1	2	9	58	2381	(25)
Public Health Program Specialist	0	0	1060	0	0	0	0	0	1060	(90)
Sanitarian	0	10	36	0	3	0	0	0	49	(98)
Industrial Hygiene	8	390	47	3	6	14	473	0	941	(93)
Consumer Safety	32	0	1483	0	0	0	0	0	1515	(100)
Environmental Health Technician	0	92	46	4	2	0	0	0	144	(96)
Veterinary Medical Science	5	14	93	1948	7	0	0	0	2067	(99)
Safety Engineering	1	358	8	0	17	24	85	0	493	(80)
Environmental Engineering	1614	694	16	20	30	29	0	0	2403	(97)
Biomedical Engineering	1	30	40	2	1	0	0	0	74	(30)
Health Physics	22	188	22	3	2	50	0	0	287	(50)
Public Health Educator	0	2	29	0	0	0	0	0	31	(91)
Consumer Safety Inspection	2	0	101	0	0	0	0	0	103	(99)
Public Health Quarantine Inspection	0	0	28	0	0	0	0	0	28	(100)
Total for Job Classifications Listed	3870	6737	8219	5766	2537	282	1422	64	28,919	(70)
Total Workforce[h]	11,822	650,517	115,872	98,282	58,135	14,392	17,504	14,936	979,460	(61)

a) All data are current as of 10/31/85 and were provided by the Federal Office of Personnel Management. All figures are based on full-time, white collar, federal civilian workers, excluding the Postal Service.
b) Defense includes only civilian employees of army, navy, air force.
c) Health and Human Services (HHS) has several offices and administrations, including the: Public Health Service (See footnote b, Table 2), Social Security Administration, Office of Human Development Services, US Office of Consumer Affairs, Health Care Financing Administration, and the Family Support Administration.
d) Total employees in each job classification for the eight agencies listed. Figures do not include Commissioned Corps of the Public Health Service.
e) Percentage of the entire federal government workforce for the specific job category, based on full-time, white collar civilian employees, excluding the Postal Service. For example, the 3,079 employees in the eight agencies who are classified in safety management represent 87% of the people in that job series across the whole federal government.
f) Toxicology is a new job series and there were no employees listed in this category as of 10/31/85. The process of reclassifying toxicologists is currently underway in several agencies.
g) The majority of these medical officers are in the Public Health Service, with approximately half directly involved in patient care and half involved in medical research.
h) Total workforce includes all full-time, white collar federal civilian workers for each listed Agency. This includes the workforce for the 27 job classifications listed, plus all other full-time employees, regardless of whether or not they are involved in environmental health-related jobs. This includes all support staff, such as secretaries, budget and personnel staff, and all managers.

eight selected agencies, that is 70 percent of all employees so classified in the federal government as a whole (Table 1).

In Table 1 we did not include more generic classifications, such as chemist, physical scientist, or statistician, because we were less certain that individuals in these categories would be involved in environmental health activities for all of the agencies listed. Nevertheless, individuals so classified and who work in a federal agency that deals primarily with environmental health issues are an important part of the environmental health workforce. Information for 1988 on the number and types of environmental health practitioners (including chemists, physical scientists and statisticians) in eight of the federal agencies most directly responsible for environmental health issues is given in Table 2. For EPA, the addition of six of these generic job categories increased the identified workforce by about 20 percent.

Because of EPA's lead role in many environmental health issues and owing to the relatively large number of environmental health professionals employed therein, Table 3 provides a more detailed description of EPA's environmental health workforce. Considering full-time, part-time, and intermittent employees, about 16 percent are involved directly with research and development; 27 percent are in regulatory offices in Washington, DC where they have responsibility for developing and carrying out environmental laws and regulations; 50 percent are employed in the 10 EPA regional offices where they oversee implementation and enforcement of regulations; and 6 percent provide necessary technical and administrative support.

Current Federal Environmental Health Activities

We categorized all environmental health activities into one or more of four general categories: research and development; risk assessment (including hazard identification); risk management/reduction; and risk communication. A description of these four categories with examples of related activities is provided in Table 4.

Many of the environmental health activities in the federal government are focused on risk assessment and risk management. To aid decision-making, agencies such as EPA, OSHA, and the Food and Drug Administration have developed formal risk assessment procedures for identifying health

TABLE 2—Workforce Statistics for Environmental Health-Related Job Classifications in Selected US Federal Agencies that are Primarily Responsible for Environmental Health Issues

	Number of Employees[a]							
			Public Health Service[b]					
Civil Service Job Classification	EPA[c]	OSHA[d]	FDA[e]	Commissioned Corps[f]	CDC[g]	NIEHS[h]	NIOSH[i]	ATSDR[j]
Job classifications likely to be involved with environmental health activities regardless of agency mission								
Safety Management	8	630	4	3	11	1	5	0
Safety Technician	1	1	3	0	0	0	0	0
Environmental Protection Specialist	1456	0	0	0	0	0	0	0
Environmental protection Assistant	200	0	0	0	0	0	0	0
General Biological Science	570	0	239	18	141	139	10	4
Microbiology	91	0	383	44	244	11	2	0
Biological Technician	45	0	133	0	93	136	8	0
Pharmacology	25	0	154	11	8	13	8	0
Physiology	10	0	26	8	8	9	7	0
Toxicology	72	0	0	0	1	8	0	1
Genetics	6	0	3	0	4	22	1	0
Biological Science Student Trainee	2	0	4	0	1	0	0	0
General Health Science	87	13	47	67	66	17	22	12
Medical Officer	8	2	575	1651	29	11	4	2
Public Health Program Specialist	0	0	20	107	578	0	2	19
Sanitarian	1	0	26	156	0	0	0	0
Industrial Hygiene	15	502	2	86	16	6	27	0
Consumer Safety	18	0	1486	59	0	0	0	0
Environmental Health Technician	0	0	0	0	1	0	1	0
Veterinary Medical Science	6	0	86	87	1	16	0	0
Safety Engineering	1	83	2	1	4	0	4	0
Sanitary Engineering	1918	0	20	367	10	0	0	8
Biomedical Engineering	1	0	52	10	0	1	0	0
Health Physics	40	0	48	63	2	4	0	0
Public Health Educator	1	0	0	19	5	0	1	0
Consumer Safety Inspection	0	0	133	0	0	0	0	0
Public Health Quarantine Inspection	0	0	0	0	31	0	0	0
Job classifications likely to be involved with environmental health activities in agencies responsible for environmental health issues								
Physical Science Technician	69	3	93	0	14	5	14	0
Chemist	638	57	1042	39	151	94	55	0
Operations Research	32	0	17	0	0	0	0	0
Mathematical Statistician	25	0	83	12	63	8	3	1
Statistician	43	4	31	56	203	3	16	0
Statistical Assistant	3	1	12	0	77	0	12	0
Total for Listed Job Classifications	5396	1496	4724	2864	1782	504	202	47
Total Workforce[k]	13,601	2360	6721	5600	4600	556	800	150

a) With the exception of the Public Health Service (PHS), all figures are based on white collar civil service employees, excluding the Commissioned Corps.
b) The *Public Health Service* is an operating division within HHS and includes the following agencies: ATSDR, FDA; NIH (including NIEHS); Alcohol, Drug Abuse and Mental Health Administration; Health Resources and Service Administration; Indian Health Service; Office of the Assistant Secretary of Health; and CDC, which includes the National Institute for Occupational Safety and Health (NIOSH), Center for Environmental Health and Injury Control, Center for Infectious Diseases, Center for Prevention Services, National Center for Health Statistics, Center for Health Promotion and Education, Training and Laboratory Program Office, International Health Program Office, Epidemiology Program Office, and the Office of Program Support.
c) EPA (Environmental Protection Agency), data current as of 12/1/88, provided by the EPA Office of Administration and Resources Management, and represent permanent, full-time, civil service.
d) OSHA (Occupational Safety and Health Administration), data were provided by the OSHA Office of Personnel and are current as of 7/30/89.
e) FDA (Food and Drug Administration), data current as of 4/1/88, provided by the Department of Health and Human Services, Office of the Assistant Secretary for Health.
f) Commissioned Corps of the Public Health Service, irrespective of agency, data current as of 4/1/88 and provided by the Division of Commissioned Personnel.
g) CDC (Centers for Disease Control), data current as of 5/31/88, provided by Personnel Management Office of CDC.
h) NIEHS (National Institute of Environmental Health Sciences), data current as of 5/21/88, provided by personnel office, NIEHS, Research Triangle Park, NC.
i) NIOSH (National Institute for Occupational Safety and Health), data are current as of June 1988, and were provided by the Personnel Management Office of the Centers for Disease Control.
j) ATSDR (Agency for Toxic Substances and Disease Registry), data are current as of June 1988, and were provided by the Personnel Management Office of the Centers for Disease Control.
k) Total workforce refers to all full-time, white collar federal civilian workers for each listed agency, including those people in the 23 job classifications listed, and all other full-time employees including all support staff such as secretaries, budget and personnel staff, and all managers. The only exceptions is PHS, which includes only the Commissioned Corps, and has only degreed scientists, engineers, doctors, etc.

hazards and for estimating the risks to human health posed by products and activities.[7–9] Risk management procedures are employed for weighing policy alternatives and for selecting the most appropriate action by integrating the results of risk assessment with engineering data and with social, economic, and political concerns.[10] The interrelationships among research and development, risk assessment, risk management, risk reduction, and risk communication are depicted schematically in Figure 2.

As shown in Table 3, most of the EPA workforce is involved in the application of scientific data to the resolution of environmental health problems (e.g., risk management and risk reduction).[11] A relatively smaller number is involved directly with data generation (research and development), while a steadily increasing fraction of the workforce is involved in risk assessment and risk communication, a trend that is expected to continue into the foreseeable future.[12–14]

Emerging Environmental Health Issues

Advances in the field of environmental health sciences, introduction of innovative technologies, and promulgation of new environmental regulations are examples of the kinds of activities that can have significant impacts on the future size, composition, and activities of the environmental health workforce. For example, increasing awareness of the potential health hazards associated with municipal and hazardous wastes led to passage of the Resource Conservation and Recovery Act which, in turn, created an expanded need for environmental health practitioners in all aspects of waste management, including collection, transport, storage, treatment, and disposal, as well as stimulating research and development on related health and risk reduction issues.

Attempts have been made to examine the environmental health issues that are likely to be most significant over the next decade or more.[15–25] Some of the more important issues are discussed briefly below.

Detection of Health Effects

Just as environmental monitoring has progressed so that it is now possible to measure pollutant concentrations at the part-per-trillion level, so too must our capabilities to ascertain target tissue concentrations, and to detect effects at the cellular and molecular level be enhanced. Coupled with similar improvements in our understanding of the basic biological mechanisms that underlie health effects, this will allow for greater certainty and accuracy in assessment of environmental health risks.

Molecular epidemiology, the use of biologic indicators to link individual exposure with health outcome, is gradually becoming a reality. Use of these techniques within the context of epidemiologic investigations offers a means to assess health risks more directly and thereby reduce uncertainties.[26,27]

Non-Cancer Health Effects

Current and future research efforts are likely to focus on several non-cancer effects of environmental agents (e.g., pulmonary, cardiovascular, reproductive, developmental, neurologic, immunologic, hepatic, and dermal) to determine which health outcomes are important and in what kinds of situations.[28]

High-Risk Groups

Advances in our understanding of biological predisposition to a particular health effect (e.g., by virtue of age or genetic susceptibility),[29] as well as in our knowledge of human activities and lifestyles that give rise to elevated exposures (e.g., time spent inside a building with air pollution problems)[30,31] will continue to help focus research and regulatory efforts on the appropriate population subgroups. More precise techniques to identify and study populations

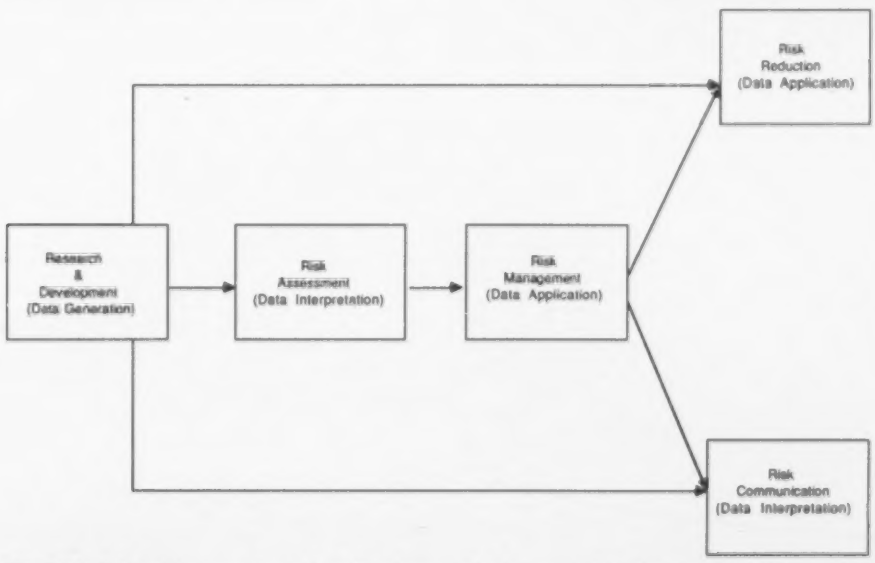

FIGURE 2—Relationships among Research and Development, Risk Assessment, Risk Management, Risk Communication, and Risk Reduction

TABLE 3—The Environmental Health Workforce Arranged According to Functional Categories within the US Environmental Protection Agency

Civil Service Job Classification	Number of Employees in Four Major EPA Functional Categories[a]							
	Research	Regulatory Offices						
	Office of Research & Development[b]	OPTS[c]	OSWER[d]	OW[e]	OAR[f]	Regions[g]	Other[h]	Total
Job classifications likely to be involved with environmental health activities regardless of agency mission								
Safety Management	0	0	1	0	0	7	0	8
Safety Technician	0	0	0	0	1	1	0	2
Environmental Protection Specialist	22	199	192	117	94	846	79	1549
Environmental Protection Assistant	17	10	1	1	4	183	4	220
General Biological Science	220	170	10	32	5	182	92	711
Microbiology	60	28	0	2	0	11	2	103
Biological Technician	111	3	0	0	0	3	0	117
Pharmacology	10	19	0	1	0	1	3	36
Physiology	10	2	0	0	0	0	0	12
Toxicology	23	38	0	4	0	14	2	81
Genetics	4	2	0	0	0	0	0	6
Biological Science Student Trainee	0	0	0	0	0	2	0	2
General Health Science	49	13	8	1	8	10	19	108
Medical Officer	5	4	0	0	0	1	19	29
Public Health Program Specialist	0	0	0	0	0	0	0	0
Sanitarian	0	0	0	0	1	0	0	1
Industrial Hygiene	2	1	0	0	0	5	9	17
Consumer Safety	0	0	0	0	0	18	0	18
Environmental Health Technician	0	0	0	0	0	0	0	0
Veterinary Medical Science	5	1	0	0	1	0	3	10
Safety Engineering	0	0	0	0	1	0	0	1
Environmental Engineering	115	3	62	93	169	1513	41	2007
Biomedical Engineering	1	0	0	0	0	0	0	1
Health Physics	13	0	1	0	18	11	3	47
Public Health Educator	0	0	0	0	1	0	0	1
Consumer Safety Inspection	0	0	0	0	0	0	0	0
Public Health Quarantine Inspection	0	0	0	0	0	0	0	0
Job classifications likely to be involved with environmental health activities in agencies responsible for environmental health issues								
Physical Science Technician	75	8	0	4	12	22	10	141
Chemist	239	167	23	21	13	176	62	701
Operations Research	4	6	2	6	5	0	9	32
Mathematical Statistician	14	1	1	0	7	1	3	27
Statistician	12	17	0	4	4	1	17	55
Statistical Assistant	1	0	0	2	0	0	0	3
Total of Listed Job Classifications	1012	692	301	288	344	3008	379	6046
Total for Whole Office	1996	1240	750	677	917	7208	3011	15,799

a) Figures provided by EPA Office of Administration and Resources Management and are current as of December 1, 1988. Figures represent civil service permanent employees, including full-time, part-time, intermittent, and temporary employees. Full-time employees work 80 hour/2 weeks; part-time employees work less than 80 hour/2 weeks. Intermittent employees work no more than 130 days/year and are primarily advisory board members and consultants. Some consultants are also in the full-time, temporary category.
b) ORD, the Office of Research and Development, is the primary research arm of EPA.
c) OPTS, the Office of Pesticides and Toxic Substances, has primary responsibility for regulating pesticides (under FIFRA, the Federal Insecticide, Fungicide, and Rodenticide Act) and other toxic chemicals (under TSCA, the Toxic Substances Control Act).
d) OSWER, the Office of Solid Waste and Emergency Response, has primary responsibility for administering programs that address hazardous and solid wastes, including the Superfund program, underground storage tanks and active Resource Conservation and Recovery Act (RCRA) facilities.
e) OW, the Office of Water, has primary responsibility for regulating contaminants in drinking water, ground water, and surface water.
f) OAR, the Office of Air and Radiation, has primary responsibility for regulating air emissions under the Clean Air Act and for administering the agency's radiation program.
g) Regions, the 10 regional offices have the primary responsibility of carrying out the various regulations in different areas of the country.
h) Other, includes the Office of the Administrator, and the following offices: Enforcement and Compliance Monitoring, General Counsel, Inspector General, External Affairs, Administration and Resources Management, and Policy, Planning and Evaluation.

that are at higher risk because of either increased susceptibility or elevated exposures are needed.

Health Hazard Identification

Currently, there are several issues and/or technologies for which the hazard identification process is underway, including: human exposure to non-ionizing radiation (particularly Extremely Low Frequency radiation inside residences)[32,33]; non-asbestiform fibers used to replace asbestos in buildings[34]; methanol used as an alternative fuel in motor vehicles[35]; and the release of genetically engineered and naturally occurring microorganisms in conjunction with

TABLE 4—General Categories of Activities Carried out by the US Environmental Health Workforce

Activity Category	Description of Activities	Example Activities
Research and Development	Scientific research and development to generate appropriate data on relevant environmental health issues. The goal is to improve the quantity and quality of the existing data base, as well as to enhance our understanding of basic underlying mechanisms.	Animal toxicology Epidemiology Human clinical research Environmental monitoring
Risk Assessment	Assessment and evaluation of available environmental health data to determine their appropriateness and adequacy for incorporation into decisions about safeguarding public health. The goal is often to develop a quantitative health risk assessment in order to identify and prioritize environmental hazards.	Evaluation of data validity and completeness Hazard identification Risk characterization Development of guidelines (e.g., risk assessment, toxicity testing)
Risk Management and Risk Reduction	The use of environmental health data to make, implement, and enforce policy decisions about protection of public health. The goal is to improve decisions that affect environmental health, reduce public health risks, facilitate implementation of decisions, and enhance enforcement activities.	Policy and regulatory decisions Pollution prevention, control, and remediation Enforcement and compliance activities Promulgation of testing guidelines
Risk Communication	Communication of the risks associated with exposures to environmental agents, as well as the scientific basis for risk management decisions. The goal is to provide environmental health practitioners and the public with usable and understandable information about environmental health issues.	Technology and information transfer Explanation of scientific basis for decisions Implementation of "Public Right-to-Know" laws Communication of the health significance of environmental exposures

the burgeoning biotechnology industry.[36,37] Both research and regulatory decisions will be affected by the outcome of these and other hazard assessment activities.

Pollution Prevention

Historically, EPA has emphasized the use of pollution control equipment to reduce health and environmental risks (e.g., stack scrubbers for SO_x, tailpipe catalysts for automotive emissions). Informally known as "end-of-pipe" controls, these measures focus on reducing pollutant emissions after they have been generated. As we move into the 1990s, however, it is becoming increasingly apparent that many environmental health problems are not amenable to this approach. Indoor air pollution, global warming, and runoff from farms and construction sites are examples of problems that are not readily solved by these traditional approaches. Thus, it is imperative that EPA and other environmental health agencies develop strategies that prevent or reduce pollution before it is generated. In the future, increasing emphasis will be placed on innovative approaches to pollution control, which include minimizing the amount of wastes generated as well as recycling or reusing those that are produced.[22]

Future Outlook

Many factors suggest an expanding requirement for environmental health professionals: the potential health hazards associated with commercialization of new technologies (e.g. superconductors); continued efforts to evaluate and control previously identified health hazards (e.g., asbestos, radon, lead, ozone, and dioxin); ongoing attempts to characterize and mitigate the health risks associated with tens of thousands of chemicals in our environment; expanding knowledge about the subtle health effects resulting from exposures to the complex mixture of pollutants in our daily environment; and increasing concerns about the potential for adverse health effects from environmental changes, such as global warming and stratospheric ozone depletion.[15–22] Studies indicate that there is already an inadequate supply of people with appropriate education and training.[1–6,22] It seems highly probable that the shortage will become even more acute unless adequate steps are taken to rectify the situation. Despite the fact that detailed analyses are precluded by a lack of objective data, certain trends in educational and training needs seem readily apparent.

- Regardless of their specific job, members of the environmental health workforce need a firm grounding in the general principles of public health, as well as an appreciation of the rudiments of health and ecological sciences. In addition, familiarity with risk assessment and risk management procedures is increasingly important.
- It has become clear that many environmental health problems are not limited to a single medium (e.g., air, waste, soil) and therefore require multimedia and multidisciplinary approaches to understand and reduce associated risks. Thus, there will be a premium on individuals who appreciate and understand the complex interactions underlying environmental hazards, and who have the interpersonal skills to function effectively as part of a multidisciplinary team.
- The requirement for professional risk assessors and risk managers will become more acute as environmental problems escalate from local concerns to national and international issues. The economic and social ramifications of addressing these issues demand that we apply informed decision-making based on adequate scientific information about the relationships between human exposures and associated health effects.

- There is an obvious need to train medical doctors so they can effectively recognize and treat environmentally induced disease (e.g., chemical sensitivity).
- A large growth in demand is likely for qualified environmental health experts who can effectively communicate the risks of environmental exposures, as well as the associated uncertainties, to nonscientists.
- There will be a continuing and probably expanding requirement for qualified environmental health professionals capable of performing relevant research and development activities. Among the professionals who are likely to be in greatest demand are epidemiologists, biostatisticians, environmental engineers and chemists, toxicologists, and mathematical modelers (e.g., environmental fate and transport models, pharmacokinetic models).
- A general knowledge of the history of environmental health, particularly in the 20th century, as well as familiarity with the major environmental laws is becoming a requirement for many environmental health professionals working in federal agencies.

In summary, there is a current shortage and a projected demand for more and better qualified professionals in the federal environmental health workforce. These needs can only be addressed if we acknowledge the problems and take appropriate, timely steps to educate, train, recruit, and retain environmental health professionals in the federal government.

DISCLAIMER

The information presented has not been subjected to the Environmental Protection Agency's peer and administrative review. Therefore, the conclusions and opinions drawn are solely those of the authors and should not necessarily be construed to reflect the views of the agency.

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Environmental and Biological Monitoring for Lead Exposure in California Workplaces

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Abstract: Patterns of environmental and biological monitoring for lead exposure were surveyed in lead-using industries in California. Employer self-reporting indicates a large proportion of potentially lead-exposed workers have never participated in a monitoring program. Only 2.6 percent of facilities have done environmental monitoring for lead, and only 1.4 percent have routine biological monitoring programs. Monitoring practices vary by size of facility, with higher proportions in industries in which larger facilities predominate. Almost 80 percent of battery manufacturing employees work in job classifications which have been monitored, versus only 1 percent of radiator-repair workers. These findings suggest that laboratory-based surveillance for occupational lead poisoning may seriously underestimate the true number of lead poisoned workers and raise serious questions regarding compliance with key elements of the OSHA Lead Standard. (*Am J Public Health* 1990; 80:921–925.)

Introduction

The United States Public Health Service goals for 1990 include the virtual elimination of occupational heavy metal poisoning.[1] Ability to assess the extent to which occupational health programs are effective in achieving this goal rests on the existence of an adequate surveillance system.[2] Adult lead poisoning is virtually always associated with work and is due to a single causative agent. Because early lead toxicity is reversible, and patients may be asymptomatic or have vague symptoms, biological monitoring for lead by analysis of blood lead levels is particularly useful.[3] Biological and environmental monitoring for lead are well-established, and required by the California and federal Occupational Safety and Health Administration (OSHA) Lead Standards.[4] California now requires laboratories to report elevated blood lead levels to the State Department of Health Services.[5]

However, little is known regarding what proportion of workers at risk of lead exposure participate in a biological monitoring program that would allow them to be identified through a laboratory-based surveillance system. Although index cases could be identified if even a small proportion of workers were monitored, a laboratory-based system with low sensitivity would reduce the ability to evaluate the true magnitude of the problem, identify trends, and/or properly target or evaluate interventions. An estimate of the sensitivity—or ability to identify workers with elevated blood levels—of such a laboratory-based system is important, if registry data are to be used for monitoring trends or targeting interventions.

The goals of the survey herein reported were to assess monitoring practices for lead exposure in California work sites, and to evaluate the extent to which a laboratory-based occupational lead poisoning surveillance system would fulfill surveillance functions.

Methods

Selection of Sample

Four pre-existing data bases were used to identify 505 unique four-digit Standard Industrial Codes (S/C) with potential for lead use: the National Institute for Occupational Safety and Health (NIOSH) National Occupational Hazard Survey 1972–74 (NOHS)[6]; federal OSHA inspections, 1979–82, provided by Dr. John Froines (UCLA) from federal OSHA Management Information Service Data; Cal/OSHA Lead Standard violators, 1979–85, provided by the California Department of Industrial Relations; and the New York Heavy Metals Registry, 1981–85.[7] A list of employers in California in these SIC codes was obtained from the California State Employment Development Department[8]; these 236,564 reporting facilities, employing 5,837,156 individuals, comprised the sampling frame for the survey.

A weighted sampling scheme, designed to be free of systematic bias, was used to enrich the survey sample with lead-using industries[9]: seven SIC codes were selected as "special interest" industries based on past evidence of high levels of ambient lead and high proportions of workers exposed. The remaining codes were categorized into 165 "high" and 333 "low" exposure groups, based on either federal OSHA inspection data, or judgment of experienced industrial hygienists. Facilities were stratified by total number of employees. The predicted number of lead-exposed workers at each facility was estimated, based on NOHS data on the proportion of exposed workers in each SIC code. Sampling with probability proportional to size[10] was conducted, using predicted numbers of lead-exposed workers as the measure of size.

Survey of Employers

Information was obtained from employers through two questionnaires administered by mail and telephone interview. The initial questionnaire was designed to identify the presence or absence of lead use, the total number of employees at the facility, and a contact name for the second questionnaire. The second questionnaire was administered only to those employers reporting lead use, and requested a list of all processes involving lead use at the facility, the number of workers involved in each process, and information about environmental and biological monitoring practices at the facility.

Categorizing Survey Responses

Lead-using processes described by employers were categorized into 12 specific processes, and three more general exposure categories (high/medium/low) based on anticipated level and frequency of exposure as judged by eight experienced industrial hygienists. It should be noted that the exposure categories utilized in the analysis were *independent*

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of the SIC categories in the initial sampling scheme. Responses were also categorized by facility size and unionization.

Analysis

The statistical analysis incorporated information on the weighted sampling scheme to produce valid and unbiased statewide estimates of numbers and percentages.[11] The standard errors for each estimate reflect the variability that arose from taking a sample; estimation of small numbers frequently leads to large relative standard errors.

Results

Response Rates

Of the 1,165 unique facilities in the initial sample, 1,089 (93.5 percent) completed the first questionnaire. Of these, 539 employers reported using lead or lead-containing materials and 521 (96.7 percent) of this group completed the second questionnaire.

Occupational Lead Exposure

In California, 52,700 facilities (7.7 percent of all facilities) were estimated to use lead. An estimated 230,000 employees work in positions in which their employers report a lead-using process. This represents almost 2 percent of California's total work force.

Environmental Monitoring

Only 2.6 percent of facilities engaged in lead-using processes were estimated to have ever done any environmental monitoring for lead. These facilities employed an estimated 205,000 workers, 10.7 percent of California workers exposed to lead. The proportion of facilities with environmental monitoring varies directly with the number of employees (Table 1).

The percent of potentially exposed employees in positions which have had environmental monitoring performed varies according to lead-using process (Table 2). For example, less than 1 percent of workers soldering pipes or painting with lead-based paints worked in monitored positions, in contrast to 80 percent of battery manufacturing workers.

In general, the proportions of employees in monitored positions are higher in unionized than non-unionized facilities; this was not true in the 100–499 employee size category, perhaps due to the large number of monitored non-unionized electronics industry solderers in this size category (data available on request to author).

TABLE 1—Estimated Number of Facilities with Lead Use in California, and Estimated Number with any Environmental Monitoring of Exposure to Lead, by Facility Size

Facility Size	Facilities with lead use N	Facilities with any environmental monitoring N	Facilities with any environmental monitoring % (SE)
1–19 employees	33,410	32	0.10 (0.04)
20–99 employees	12,753	292	2.29 (1.23)
100–499 employees	4,112	405	9.85 (4.10)
500 or more employees	2,402	630	26.22 (7.12)
Total	52,677	1,359	2.58

Of those workers with high/daily exposures, 37 percent work in positions which had been monitored, versus only 2.6 percent in positions with high/weekly exposures. Radiator repair was the most commonly reported process with high/daily exposures for which environmental monitoring had never been performed.

The characteristics of process, size, and unionization appear to be interrelated. For example, an estimated 88 percent of radiator repair employees work in small (1–19 employees) facilities, while 87 percent of battery manufacture workers are employed in larger (100–499 employees) facilities. Less than .6 percent of the smallest facilities' workers are unionized, versus nearly 50 percent of those at the largest size facilities. A significant proportion of environmental monitoring appears to be done by company-employed industrial hygienists. At large facilities, this was true for 80 percent of monitored workers. For the smallest companies, about 57 percent of monitoring was conducted by private contractor, 19.7 percent by insurance companies, and 23.2 percent by Cal-OSHA (compliance or consultation). Less than one-half of monitored positions had been evaluated in the year preceding the survey; in over 17 percent, monitoring was most recently performed in 1980.

Biological Monitoring

Only 1.4 percent of facilities engaged in lead-using processes, and about 2.6 percent of potentially lead-exposed workers were estimated to have routine biological monitoring programs. Marked variation in biological monitoring practices was noted among the seven "special interest" SIC groups (Table 3). Fifty-three percent of the blood lead analyses performed as part of routine biological monitoring programs are performed by California laboratories, with the rest being done by out-of-state laboratories. The proportion of employees receiving biological monitoring varies with size of facility and potential for high lead exposures (Table 4).

Discussion

There are several possible limitations in the methodology employed in this survey. Results were based exclusively on data from employer self-reporting of lead use, number of workers exposed, and monitoring practices. No attempts were made to validate the information received from employers.

It is possible that some employers did not report actual use of lead on the first survey, and were subsequently excluded; this should not have led to an underestimate of the proportion of workers monitored.

Workers conducting different job activities may have been lumped together as "monitored", even if only one activity in an area has actually been assessed. Also, full- and part-time workers may have been lumped together. Some battery manufacturers included clerical workers among those potentially exposed. In several facilities, these employees received biological monitoring, but none of their positions were environmentally monitored. This may explain discrepancies between environmental and biological monitoring prevalence (e.g., 79 percent of battery workers are in environmentally monitored positions, while 95 percent are biologically monitored).

In the absence of actual exposure data for each work site, the extent to which monitoring actually should be conducted in the sampled facilities cannot be determined. It is possible that monitoring was not done because exposures are below those levels that warrant it. Although our survey

TABLE 2—Estimated Number of Lead-exposed Employees, by Occurrence of Environmental Monitoring at any Time in the Past by Type of Lead-using Process

Lead-using Process	Employee working in position where environmental monitoring ever done? Yes N	Yes % (SE)	No N	No % (SE)	Total employees exposed to lead N
Soldering, except pipes and sheets	18,931	16.59 (3.98)	95,169	83.41 (3.98)	114,100
Dispensing leaded gasoline	129	0.42 (.42)	30,829	99.58 (0.43)	30,958
Soldering pipes and sheets	116	0.52 (0.31)	22,234	99.48 (0.31)	22,350
Cable cutting and splicing	374	2.72 (3.52)	13,393	97.28 (3.52)	13,767
Painting with lead-based paint	92	0.72 (0.53)	12,601	99.28 (0.53)	12,693
Painting with lead-based inks	0	0	7,496	100.00	7,496
Casting or melting lead or jointing	975	21.71 (7.86)	3,517	78.29 (7.86)	4,492
Radiator repair[a]	49	1.14 (0.79)	4,271	98.86 (0.79)	4,320
Welding metal alloys or surfaces painted with lead based paints	280	6.79 (2.55)	3,844	93.21 (2.55)	4,124
Machining, grinding or sanding surfaces painted with leaded paints	254	7.92 (7.23)	2,954	92.08 (7.23)	3,208
Battery manufacture	1,477	79.62 (8.65)	378	20.38 (8.65)	1,855
Other process with lead use	1,814	18.01 (6.41)	8,258	81.99 (6.41)	10,072
Total	24,491	10.67	204,944	89.33	229,435

[a]Includes workers in SICs 7528 and 7539

instrument did not directly assess this possibility, it seems unlikely that this would be true in the "high" exposure categories. There is no evidence that workers in smaller facilities are likely to have lower exposure than those in larger plants, suggesting that the lack of monitoring in small facilities is due to factors other than lack of exposure.

Few other data on biological and environmental monitoring are available. Ratcliff, Halperin, *et al.*[12] utilized data from NOHS to demonstrate that there is no consistent evidence that workers potentially exposed to chemical hazards are more likely to receive screening than other workers, but that such screening is more likely in large facilities. This is consistent with the results of the current survey.

However, the NIOSH study estimated that almost 25 percent of lead-exposed workers receive periodic monitoring, and that 18 percent receive blood tests—a substantially higher proportion than seen in this survey. The NOHS survey was done prior to promulgation of the OSHA Lead Standard; it asks about any blood test, without reference to specific biological monitoring for a particular substance. Also, the NOHS appears to have counted all employees as receiving blood tests if any did.[6] In contrast, the current study asked about monitoring in workers specifically identified as working in lead-using jobs. Therefore, the results reported here are likely to constitute a more accurate assessment of monitoring for lead in lead-exposed workers.

The lack of environmental or biological monitoring in a majority of potentially lead-exposed workers may result in inability to identify workers or work sites where interventions could prevent cases of occupational lead poisoning. The widespread lack of monitoring makes surveillance for trends, assessment of the true magnitude of the problem, or evaluation of the efficacy of interventions virtually impossible. A laboratory-based surveillance system for lead over-exposure would be limited to the identification of two categories of workers: those with overt clinical signs of lead poisoning for whom an alert clinician has ordered a blood lead test, and the small minority of workers who participate in a routine monitoring program. These categories are likely to represent two extremes: the very worst cases, and those who are likely to have more access to medical and industrial hygiene services by virtue of being enrolled in a monitoring program. The large majority of lead-exposed workers remain excluded, and thus inaccessible to the surveillance system.

These limitations do not undercut the ability of a laboratory-based surveillance system to identify important index cases of lead poisoning.[13]

Our data imply a significant lack of compliance with the monitoring provisions of the OSHA Lead Standard, which were constructed specifically to facilitate timely interventions. The relatively high prevalence of monitoring in battery manufacture may be attributable to past California OSHA compliance efforts specifically directed at biological monitoring activities in this industry.* Implementation of recommendations[14] that would require employers to report results of medical examinations and exposure monitoring to OSHA could complement laboratory-based surveillance and assist OSHA in identifying industries and employers who appear to be out of compliance with monitoring requirement.

*Personal communication, J. Simonowitz, RN, Cal/OSHA Medical Unit.

TABLE 3—Estimated Number of Employees Who are Receiving Routine Biological Monitoring by SIC Code

Lead-using SIC Codes	Employees potentially exposed to lead[a] N	Employees receiving routine biological monitoring N	% of exposed (SE)
Estimated exposure below action level[b]	107,322	997	0.93 (0.46)
Estimated exposure above action level, excl. below[b]	88,262	2,475	2.80 (1.08)
1711: Plumbing, heating and air conditioning	22,742	0	0
3341: Secondary smelting non-ferrous metals	393	132	33.59 (12.84)
3691: Storage battery manufacture	1,850	1,762	95.25 (3.68)
3731: Shipbuilding and repair	1,089	477	(43.79) (28.07)
7538: Automotive repair shops	6,561	1	0.02 (0.02)
7539: Radiator repair shops[c]	903	70	7.95 (3.95)
7692: Welding repair	312	0	0
Total	229,434	59,142	2.58

[a]Based on employer survey report.
[b]Based on industrial measurements from fed/OHSA or judgment of industrial hygienist.[9]
[c]Companies in this SIC without "radiator" in the name excluded.

Approximately 12 percent of potentially lead-exposed individuals are in construction jobs which are not covered by the Lead Standard.** In this segment, monitoring is extremely limited—for example, no plumbing employees were reported to have biological monitoring for lead, and less than 1 percent of those painting with lead-based paints worked in environmentally monitored positions. Yet reports indicate a serious continued potential for lead poisoning in the construction industry.[13]

Our findings suggest a need for new strategies for enforcement and enhanced compliance with the existing occupational lead standard, as well as a need for expansion of the standard to cover construction workers. Increased employer and worker education regarding lead and the lead standard are also warranted. Education and targeted compliance may be particularly important in small and non-unionized shops.

In conclusion, only a small proportion of the large number of potentially lead-exposed workers in California work in positions which have ever had industrial hygiene monitoring; an even smaller proportion participate in a routine biological monitoring program. Workers in small and non-unionized facilities are especially unlikely to participate in any type of monitoring program. The failure of employers to establish legally mandated environmental and biological monitoring programs severely limits the ability of laboratory-based surveillance programs to adequately fulfill critical functions of occupational disease or exposure surveillance. This, in turn, severely impedes our progress in the prevention and ultimate elimination of occupational lead poisoning.

TABLE 4—Estimated Percent of Lead-exposed Employees Receiving Routine Biological Monitoring by Highest Level of Potential Exposure Reported at Their Workplace and by Facility Size

Level of exposure	Number of employees at facility 1–19	20–99	100–499	>500	All facilities
High	0.52	9.90	48.99	15.90	16.34
Moderate	0	0	8.38	1.01	1.1€
Low	0	0	0.24	0.10	0.05
All facilities	0.06	0.55	11.27	4.09	2.58

**The construction industry is required to meet other OSHA standards limiting lead exposure to 200μg/m³, but is not required to provide air or blood monitoring.

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Accelerated Nursing Program at JHU Quadruples Enrollment in 2nd Year

The shortage of nurses in the United States will be eased by former park rangers, Peace Corps volunteers, teachers, flight attendants, and even an ex-circus clown, according to word received from the Johns Hopkins Medical Institutions. Enrollment in an accelerated nursing program there has quadrupled during its second year.

The program, initiated last year in response to the nationwide need for high quality nurses, allows individuals who already hold a bachelor's degree in some other field to earn a bachelor's in nursing in just 15 months—nearly half the time it ordinarily takes to become a registered nurse.

The accelerated program was established to attract a new pool of bright students into the nursing profession by providing an intensified curriculum that responds to their academic and clinical needs. While 13 in the current class have just graduated from college, the majority of students are choosing nursing after working at a previous career, such as those listed above.

The current class of 60 future nurses at JHU includes 10 men. Students range in age from 20–51 years, come from 17 different states, and hold degrees from Stanford, Duke, Antioch, Smith, the University of Virginia and the University of Chicago, among other institutions. Ten members of the new class hold master's degrees.

More than one in every 10 budgeted positions for registered nurses in US hospitals is vacant at the present time, and as many as one in five budgeted nursing home positions is empty. It has been estimated that it would take about 130,000 full-time registered nurses to fill vacant positions in hospitals and nursing homes across the nation.

For further information, contact Johns Hopkins Medical Institutions, Office of Public Affairs, 550 North Broadway, Suite 1100, Baltimore, MD 21205. Tel: (301) 955-6680.

Field Comparison of Several Commercially Available Radon Detectors

R. William Field, MS, and Burton C. Kross, PhD

Abstract: To determine the accuracy and precision of commercially available radon detectors in a field setting, 15 detectors from six companies were exposed to radon and compared to a reference radon level. The detectors from companies that had already passed National Radon Measurement Proficiency Program testing had better precision and accuracy than those detectors awaiting proficiency testing. Charcoal adsorption detectors and diffusion barrier charcoal adsorption detectors performed very well, and the latter detectors displayed excellent time averaging ability. Alternatively, charcoal liquid scintillation detectors exhibited acceptable accuracy but poor precision, and bare alpha registration detectors showed both poor accuracy and precision. The mean radon level reported by the bare alpha registration detectors was 68 percent lower than the radon reference level. (*Am J Public Health* 1990; 80:926–930.)

Introduction

Radon provides the primary source of ionizing radiation exposure in man, imparting a greater effective dose equivalent to the average individual than other natural and man-made radiation sources combined.[1] Although radon has always existed in the environment, it did not become a major public health concern until 1984, when high radon levels were discovered in eastern Pennsylvania homes.[2] In 1986, the United States Environmental Protection Agency (EPA) estimated that up to 20,000 lung cancer deaths per year in the US may be due to exposure to radon.[3] The news coverage, public information campaigns, and reported health consequences that followed these discoveries initiated the cascade of public interest, awareness, and anxiety that surrounds radon today.

Public concern over radon in individual dwellings has resulted in a rise in the number of primary companies that provide radon measurement services to over 700 companies nationwide. Although the US does not have a national program to certify radon measurement companies, the National Radon Measurement Proficiency (RMP) Program within the Radon Division of the US EPA Office of Radiation Programs determines the accuracy of radon detectors on a voluntary basis and informs the public of these results upon request.[4] The majority of states have no ongoing quality assurance program for radon testing enterprises operating within their borders. Iowa has recently initiated a certification program to set minimum requirements for radon testing and analysis,[5] but will still rely on the federal government to actually test the radon measuring devices. In most cases, before Iowa certifies companies for radon testing, the State Department of Public Health requires that companies pass National RMP Program proficiency testing. Nevertheless, if companies enroll in the National RMP Program for the first time, then certification can be granted, pending review of their application and proficiency testing which allows some companies to market their radon detecting devices for up to a year prior to completion of proficiency testing.

After a company enrolls in the proficiency program, it must submit a specified number of detectors (e.g. five charcoal canisters) to the EPA for testing. These detectors are exposed in a calibration chamber to known levels of radon and radon decay products and returned to the company for analysis. The radon level in the chamber is not revealed to the company. A company is judged proficient if the mean of the absolute value of the relative measurement errors (MARE) of all exposed detectors for a particular method (activated charcoal adsorption, alpha track detection, etc.) is less than or equal to 0.250. In addition to the above proficiency testing, the EPA performs intermittent blind testing of commercially purchased detectors. A company that fails to pass either the announced or blind testing will not pass proficiency for that round. However, the company may participate in the next round of testing which occurs at intervals of up to a year or longer.

The majority of radon detectors purchased in the US are used for performing an initial radon screening measurement, which can quickly and inexpensively determine if elevated radon levels exist in a home. If screening is performed in adherence to EPA radon measurement protocols,[6,7] then this initial measurement will usually represent the maximum radon concentration to which the occupants of the home may be exposed. It is crucial that the initial screening measurement be accurate, because this result dictates whether additional testing is necessary.

The National RMP Program uses only a controlled calibration chamber to test radon-detecting devices, which may not reflect conditions in a dwelling. For example, the accuracy and precision of the radon measurement in a dwelling may be affected by temporal radon variations, changes in particulate (dust) concentration and size, air movement, humidity, temperature, and degree of radon progeny equilibrium, whereas in a calibration chamber the radon concentration and environmental parameters are highly controlled. Because of these potential differences, it is not known whether the accuracy measured in a calibration chamber can be extrapolated to the field.

This study determines the accuracy and precision of several commercially available radon detectors in a field setting. The accuracy and precision of detectors that have passed National RMP Program testing are compared to those that are awaiting proficiency testing.

Methods

The study site was a basement bedroom (see Appendix A) located in a ranch style home in southeastern Iowa. This house was selected based on the following criteria:

- Its observed radon concentration reflects the mean radon level of 10.5 pCi/L found in a recent screening

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study of 600 Iowa homes.* Prior radon sampling at the experimental site had shown that the basement bedroom radon concentration averaged 11 pCi/L during the winter months.

- The air in the home was conditioned with a humidified central forced-air heating system which maintained the relative humidity and temperature at 38–47 percent and 20°C, respectively.
- None of the four occupants (two adults and two children) smoked.
- Closed house conditions as described by EPA protocols could be monitored.

Radon detectors from six companies were chosen for this study (Appendix B). Criteria for detector selection included: large sales volume in Iowa, state certification or pending state certification, and ease of availability to consumers. In addition, detectors from two other sources not readily available to shoppers were chosen based on either ability to produce a true integrated average radon measurement (in the case of the electret ion chamber from Rad Elec, Inc.) or, widespread use in radon screening (in the case of the EPA's charcoal adsorption canister).

Radon detectors from each company listed in Appendix B were purchased from various southeast Iowa commercial sources, including hardware stores, discount stores, pharmacies, grocery stores, and a heating supply company. Placement of the radon detectors was performed in accordance with EPA protocols for screening measurements.[6,7] All the detectors were placed a minimum of 10.2 cm away from other objects, including other radon detectors. Except for normal entering and exiting, all windows and exterior doors in the home were closed 12 hours prior to and during the testing period; fans and ventilation systems that bring in outside air or exhaust house air were not operated. An occupant activity log was maintained to document maintenance of closed house conditions.

*Kross BC, Vust LJ, Field RW: Factors associated with elevated radon levels in rural areas. Manuscript in preparation.

A femto-Tech continuous radon monitor (CRM) was positioned in the center of a 4 m^2 platform. The platform, located on a queen size bed, was 0.9 m above floor level. Radon detectors from all but one company were evenly distributed around the CRM. As per the accompanying instructions, the detectors from Air Check, Inc. were suspended from the ceiling approximately 30 cm above the other detectors.

The femto-Tech radon monitor was calibrated before the study period commenced against a femto-Tech "master" unit. The "master" unit was calibrated in the EG&G Mound Technologies, Inc. Radon Chamber which participated in the cross comparison network of US Department of Energy and US EPA radon chamber facilities. The accuracy of the femto-Tech CRM is estimated to be within 10 percent. The CRM provided hourly printouts of the radon concentrations in the room and was operating 24 hours prior to and during the entire seven day study period which initiated at 1100 hrs on March 18, 1989 and terminated at 1100 hrs on March 25, 1989.

The radon measurement periods and numbers of detectors exposed from each company are listed on Table 1. With the exception of the electret ion chambers from Rad Elec, Inc., these measurement periods were based on the optimal exposure duration, as indicated in the instructions that accompanied each detector. Five electret ion chambers were exposed for each of the three exposure periods in order to evaluate the homogeneity of the bedroom radon concentration. Although there were three different radon measurement initiation dates, all measurements were terminated at 1100 hrs on March 25, 1989. One detector from each company and three detectors from Rad Elec, Inc. were used as field control detectors which remained sealed and stored in a low radon environment during the course of the study. The controls were labeled in identical fashion to the exposed detectors to ensure identical processing and mailed back to the companies with the other detectors on March 28, 1989. The measurement results for each type of detector were compared to the reference values established for the various exposure periods by the CRM.

TABLE 1—Precision and Accuracy of Radon Detectors

Company	Number of Detectors	Exposure (days)	Radon Level Mean ± S.D. (pCi/L)	*Coefficient of Variation (%)	**Mare	Reference Radon Concentration Mean (pCi/L)
Rad Elec, Inc.	5	7	10.2 ± 0.4	3.9	0.045	10.6
American Radon Services	15	7	10.8 ± 0.8	7.5	0.057	10.6
Air Check, Inc.	15	7	11.4 ± 1.1	9.6	0.098	10.6
The Radon Project	15	7	10.5 ± 2.7	25.9	0.169	10.6
Rad Elec, Inc.	5	5	10.1 ± 1.2	11.8	0.091	10.6
Terradex	15	5	3.4 ± 1.7	50.6	0.679	10.6
Rad Elec, Inc.	5	2	9.6 ± 0.5	5.1	0.052	9.2
Ryan Nuclear Labs	15	2	11.0 ± 0.8	7.7	0.198	9.2
Key Technology	15	2	10.3 ± 0.8	7.5	0.136	9.2
EPA	15	2	11.6 ± 0.5	4.4	0.264	9.2

*The coefficient of variation was calculated using a standard deviation carried out to three decimal places.
**The mean of the absolute values of the relative errors (MARE) must be ≤ 0.250 to pass proficiency. Its calculation is shown below.

$$MARE = \frac{\sum_{i=1}^{n} \frac{|M_i - T_i|}{T_i}}{n}$$

n = number of detectors exposed
M_i = measured value for detector i
T_i = target value for detector i (reference value in this case)

Results

A plot of the hourly measurement results obtained from the CRM is shown in Figure 1. The radon level in the bedroom showed considerable variation, especially during the last two days of the study which was probably related to increased ventilation rates in the basement. The occupant activity log notes that the children living in the home frequently opened the basement door leading to the outside during the evening of March 23 and the afternoon of March 24, 1989.

The measurement results for each type of detector are listed in Table 1. The measurements for the individual detectors from each company were reduced to the mean, standard deviation, coefficient of variation and MARE (Table 1) as a measure of the detectors' accuracy and precision.

The relatively small standard deviations within the three groups of electret ion chambers exposed for each time period indicate a fairly homogeneous radon concentration in the measurement area.

The MARE for the seven-day exposure detectors were all within the National RMP Programs requirement of ≤ 0.250.[4] The precision for the seven-day exposure duration detectors was below the 10 percent coefficient of variation as per the radon measurement protocol guidelines published by the EPA,[6] except for the radon detectors from the Radon Project which had a coefficient of variation of 25.9 percent.

Terradex's five-day exposure duration bare alpha track registration devices (BARDs) yielded a MARE in excess of the EPA's accuracy guidelines (≤ 0.250 required for accuracy) and a coefficient of variation in excess of the EPA's BARD radon measurement protocol precision guidelines[7] (≤ 20 percent required for precision).

The two-day exposure duration detectors from Ryan Nuclear Labs and Key Technology were both within the EPA guidelines for accuracy[4] and precision.[6]

Figure 2 shows the distribution of individual test results for each company and their relationship to the radon reference level.

The reported radon level for the blanks submitted to the companies were all < 0.5 pCi/L.

Discussion

The radon detectors that have already passed the National RMP Program displayed reasonable accuracy and precision when compared to the reference radon level generated by the continuous radon monitor. The diffusion barrier charcoal adsorption (DBCA) detector from American Radon Services and Air Check, Inc. appeared to be the best over-the-counter integrators of radon, yielding accurate results and acceptable precision for the week-long exposure period.

The accuracy of the charcoal adsorption (CA) detectors from Key Technology, Ryan Nuclear Labs, and the EPA was not as good as that attained for the DBCA detectors mentioned above, although the MARE from Ryan Nuclear Labs and Key Technology detectors was within 0.250. The EPA's CA detector accuracy was just above a MARE of 0.250 at 0.264. All three companies reported higher radon levels than the radon reference value for the 48-hour exposure period. The results from these detectors seem to be weighted by

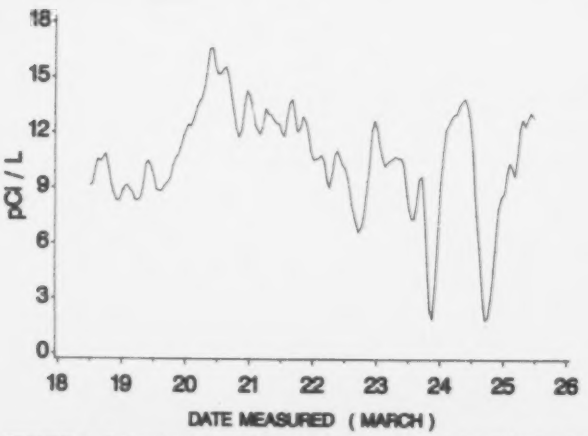

FIGURE 1—Interpolated Hourly Plot of the Radon Measurements Obtained from the Continuous Radon Monitor for the Period 1100 hrs March 18 to 1100 hrs March 25, 1989

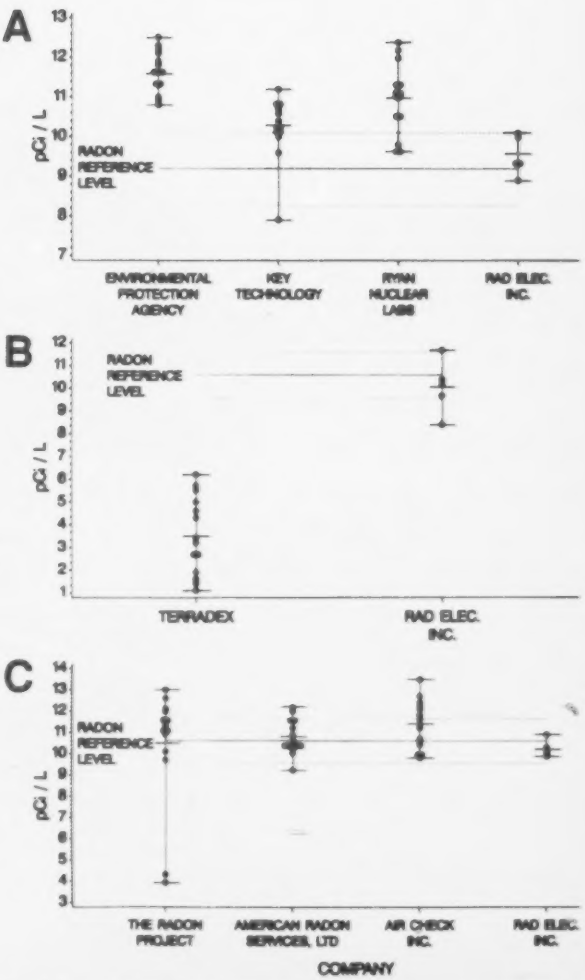

FIGURE 2—Comparison of the companies' individual detector results with the continuous radon monitor's reference value for the three exposure periods. Two (A), five (B), and seven (C) day exposure periods were initiated at 1100 hrs on March 18, March 20, and March 23, 1989, respectively. All three exposure periods ended on March 25, 1989 at 1100 hrs. The solid horizontal lines represent the radon reference values while the dotted horizontal lines indicate the confidence interval for the estimated accuracy of the continuous radon monitor. Each circle represents an individual radon detector measurement result. The means for the radon values reported by each company are indicated on the vertical range lines.

radon levels over the last 12 hours of the exposure period which averaged 11.9 pCi/L. These results are not unexpected, since the CA detectors do not integrate radon levels as well as the DBCA detectors. Despite the extreme fluctuations in radon levels, the two-day detectors performed reasonably well.

The detectors from the two companies awaiting proficiency testing did not perform well. Radon measurements using Terradex's BARD resulted in both poor accuracy and precision. Although the reference value was 10.6 pCi/L for the five-day exposure period, nine of the 15 detectors reported radon levels below 4 pCi/L (Figure 2B). The EPA recommends follow-up testing only if a screening radon test is above 4.0 pCi/L. If these detectors are representative of all Terradex's BARDs, then the majority of consumers with home radon levels and ambient conditions similar to the study site would theoretically receive radon measurement results below the EPA's action level of 4.0 pCi/L. This has significant public health implications, since these consumers would not perform follow-up testing or possible subsequent mitigation.

Several factors could influence BARD measurement variability, including ratio of radon daughters to radon in air, differences in the number of tracks used as background, locating objects less than 8 cm from the detector surface, variations in etching conditions and differences in readout.[7]

The results from the Radon Project's charcoal liquid scintillation (CLS) detectors underscore the importance of measuring precision. These detectors showed acceptable accuracy but poor precision (see Table 1). Since the National RMP Program has no requirements for precision the detectors would have passed proficiency, even though their coefficient of variation (25.9 percent) was over the EPA guidelines (≤10 percent) for precision. Thirteen detectors produced measured radon levels between 9.7 pCi/L and 13.0 pCi/L, but two detectors yielded results of 3.9 pCi/L and 4.3 pCi/L (see Figure 2C). The two lower results may be due to poor quality control of detector packaging. The two detectors that reported the highest (13.0 pCi/L) and the lowest radon level (3.9 pCi/L) both had loose vial caps upon initial examination. Loose caps compromise the detectors' seal, allowing both radon and water vapor to enter into the detector prior to actual deployment. Thus, the measured radon level could either be falsely elevated or lowered, depending on conditions that existed prior to detector use. For example, if the detectors with loose caps were stored in a high humidity environment, water molecules could reduce the number of radon adsorption sites on the charcoal, resulting in a lower measurement. On the other hand, if the detectors were stored in an area with high radon levels prior to deployment, a higher measurement could ensue.

In addition to the problems with the loose caps, six out of the 16 detectors purchased had diffusion barriers that had fallen out of the vial caps. The diffusion barrier is an integral part of this detector, allowing it to be marketed as a time-averaging device. It is unlikely that a consumer would search through the packaging material to replace a missing diffusion barrier. In this experiment all of the missing barriers were replaced into the vial caps. Securing the diffusion barrier to the cap would improve performance of this detector.

Finally, two of the 16 detector caps contained a second diffusion barrier which was not removed for our field experiment. These two detectors yielded results of 4.3 pCi/L and 11.4 pCi/L, compared to the reference CRM value of 10.6 pCi/L. Without further testing it is impossible to assess whether or not the extra vapor barrier affected the radon readings delivered by the detectors.

We offer the following recommendations to improve both the precision and accuracy of commercial radon detectors:

- States should require that companies pass proficiency testing by either the EPA or the state prior to selling their products. Interim or temporary certifications should not be granted while proficiency testing is being performed.
- The National RMP Program should require that detectors also pass a nominal precision criterion, as recommended in the Indoor Radon and Radon Decay Product Measurement Protocols[6,7] before passing proficiency testing.
- The National RMP Program should increase the rate at which it can test the detectors to accommodate the proliferating numbers of radon detecting companies.
- The National RMP Program should include occasional field testing of radon detectors.
- Radon chamber conditions should be diversified to better reflect the variety of actual home conditions.

ACKNOWLEDGMENTS

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APPENDIX A

Study Site in Southeastern Iowa

The bedroom has a poured concrete floor, and the south and east outside walls are constructed of concrete block. The east wall is about 1.5 m below grade while the south wall is even grade. The west and north walls are common to the rest of the basement. The floor covers an area of 14.4 m^2 and the ceiling height is 2.2 m, resulting in a total room volume of 31.7 m^3. There is a heating vent with an air flow rate of 0.12 m^3/min located 2 m above the floor on the northwest wall. Air flow rates in the study area averaged below 0.015 m/min. A well insulated window with blind is located in the south wall, and both windows and blind remained closed during the course of the study. However, the door to the room remained open so that the room could equilibrate with basement temperature, relative humidity, and radon concentrations.

APPENDIX B

Commercially Available Radon Monitors Used in Detector Intercomparison

Company, City	Measurement Method	Detector Type
Air Check, Inc., Arden, NC	Activated charcoal adsorption	Diffusion barrier charcoal loaded bag*
American Radon Services, Ltd., Ames, IA	Activated charcoal adsorption	Diffusion barrier charcoal adsorption canister*
Key Technology, Jonestown, PA	Activated charcoal adsorption	Charcoal adsorption canister*
The Radon Project, Pittsburgh, PA	Activated charcoal adsorption	Diffusion barrier charcoal adsorption liquid scintillation vial**
Ryan Nuclear Labs, W. Jefferson, OH	Activated charcoal adsorption	Charcoal loaded bag*
Terradex, Glenwood, IL	Alpha track detection	Bare alpha track registration device**

*Indicates company has passed Round 5 of the National Radon Measurement Proficiency Program for that particular detector.
**Indicates company did not participate in Round 5 of the National Radon Measurement Proficiency Program for that particular detector, but did participate in Round 6 of testing and is awaiting results.

Sites Listed for Nurse Scholars to Repay Educational Obligation

Recipients of scholarships for the undergraduate education of professional nurses are required upon graduation to serve at least two years in specified shortage sites. The Health Resources and Services Administration has announced a list of certain facilities where such recipients may repay their obligation. These include an Indian Health Service health center, a Native Hawaiian health center, a public hospital, a community or migrant health center, a nursing facility, a rural health clinic, or a health facility determined by DHHS to have a critical shortage of nurses. This announcement, which was published in the May 25 *Federal Register*, defines critical shortage facilities to include:

- All rural hospitals (classified by Medicare and Medicaid),
- All "disproportionate share" hospitals (those determined by Medicare and Medicaid as serving a disproportionate number of low income patients),
- Home health agencies approved for Medicare and Medicaid reimbursement,
- State and local health departments,
- Indian Health Service hospitals and other IHS facilities approved for Medicare and Medicaid reimbursement,
- Veterans hospitals, and
- Military health care facilities of the Department of Defense.

More information is available from the Division of Student Assistance, Bureau of Health Professions, 5600 Fishers Lane, Rm 8-48, Rockville, MD 20857. Tel: (301) 443-1173.

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HRSA also announced that it is accepting FY 1991 grant applications for advanced nurse education. Applications must be submitted by October 1 to the Grants Management Officer, Bureau of Health Professions, 5600 Fishers Lane, Rm 8C-26, Rockville, MD 20857. Tel: (301) 443-6960.

Elevated Blood Lead in California Adults, 1987: Results of a Statewide Surveillance Program Based on Laboratory Reports

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Abstract: California medical laboratories that test for blood lead are required to report results exceeding 1.21 μmol/L (25 μg/dl). Between April and December 1987, the California Department of Health Services received 3,077 blood lead reports from 34 laboratories for 1,293 civilian, non-institutionalized adults.

Approximately 1 percent of all reports exceeded 3.87 μmol/L (80 μg/dl), 7 percent exceeded 2.42 μmol/L (50 μg/dl), and 21 percent exceeded 1.93 μmol/L (40 μg/dl). Individuals tested were overwhelmingly male (94 percent), disproportionately Hispanic surnamed (44 percent), and most often residents of Los Angeles County (81 percent).

Workers in lead smelting, battery manufacturing, and brass foundries accounted for nearly 80 percent of reports. Construction, radiator repair, pottery and ceramics manufacturing, and gun firing ranges accounted for the remainder. All adults with reports of ≥2.90 μmol/L who were contacted reported an occupational exposure. Approximately half were not in routine medical monitoring programs. Despite OSHA standards, elevated blood lead with the potential for serious acute and chronic lead poisoning in California adults remains a significant public health and major occupational health concern. (*Am J Public Health* 1990; 80:931–934.)

Introduction

Under a state law in effect since January 1, 1987, California medical laboratories performing tests for blood lead have been required to report personal identifiers and blood lead levels of individuals with tests exceeding 1.21 μmol/L (25 μg/dl), irrespective of age. The California Department of Health Services (CDHS) was charged with notifying laboratories, establishing a proficiency program for laboratories, designing report forms, and tabulating the reports. Over 95 percent of blood lead reports received from this program in 1987 were for persons greater than 16 years of age, and are the basis of this report.

Workplace exposures to lead are regulated under a standard issued in 1978 by the federal Occupational Safety and Health Administration.[1] The standard established an exposure limit of 50 $\mu g/m^3$ for airborne lead exposures for an 8-hour work day, and required employers to provide training, respiratory protection, work clothes changes, shower facilities, and a medical surveillance program, including annual physical examinations and periodic blood lead tests to eligible employees. Workers in agriculture and construction are currently excluded. The standard requires that workers with blood lead levels exceeding 1.93 μmol/L be re-tested bimonthly and those exceeding 2.90 μg/dl (or average 2.41 μmol/L on three previous tests) be immediately transferred and placed in jobs with low or no exposure.

This article describes initial results from a pilot program for surveillance of blood lead reports from medical laboratories and from follow-up of adults with reported blood lead levels of 2.90 μmol/L.

Methods

Patient identifiers, demographics, blood lead level, and other relevant information were reported by laboratories on specially designed forms. If the gender field of the registrant was blank, one was designated on the basis of first name of the registrant. An indicator of Hispanic surname was also designated. Industry of employment was based on an assignment of the Standard Industrial Classification (SIC)[2] code for reports that included an employer identifier. Employer name and address on the report form were matched to listings in the 1987 California Manufacturers Directory.[3] The SIC code for the primary business activity was then assigned to the report. Due to a paucity of complete data, occupation was not coded.

Follow-up of Elevated Blood Lead Reports

A working draft protocol for follow-up of reports was implemented for cases exceeding 2.90 μmol/L. Individuals and their physicians and employers were traced, and structured telephone interviews were administered to determine: the level of direct medical supervision, if any; work history and source of exposure, occupational and/or non-occupational; number of household contacts less than 7 years of age; number of co-workers exposed to lead; use of personal protective equipment and hygienic practices at work; and application of medical removal requirements of the federal OSHA lead standard.

An information packet was sent to the worker, his/her physicians, and employer.[4–6] Subsequent blood lead reports were tracked until levels fell below 1.93 μmol/L; workers were re-contacted if their subsequent blood lead levels abruptly increased.

Employers were informed of free industrial hygiene services available from the state OSHA Consultation Services, or encouraged to seek private consultants for technical consultation. Apparent egregious violations of the OSHA lead standard were channeled to appropriate enforcement agencies for follow-up. A recommendation to screen children and/or household contacts for elevated blood lead was forwarded to the state and local health officials when household contacts included children at risk of parental take-home exposures or non-occupational activities, or when an adult's exposure was non-occupational.

Laboratory Certification

A proficiency program was established by CDHS, and, according to its protocol, laboratories are certified as proficient if lead concentrations are reported within 15 percent of

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target values for concentrations less than 1.93 μmol/L, or within .29 μmol/L of target values for concentrations greater than 1.93 μmol/L in 75 percent of at least nine reference samples.

Results

Blood Lead Distributions

Between April and December 1987, CDHS received 3,077 blood lead reports from 34 laboratories for 1,293 civilian, non-institutionalized adults, 16 years and older. Blood lead level was filled in on 3,024 report forms, and 2,643 reports were greater than 1.21 μmol/L (Table 1). Approximately 60 percent of individuals had only one test, 14 percent had two tests, 5 percent had three tests, and 22 percent had four or more tests in 1987.

Of reports greater than 1.21 μmol/L, approximately 1 percent (24) were equal or greater than 3.87 μmol/L, 7 percent (198) were equal or greater than 2.42 μmol/L, and 25 percent (649) were equal or greater than 1.93 μmol/L. Counting individuals once, but at their highest blood lead level in 1987, 355 individuals (31 percent) had a peak blood lead level of 1.93 μmol/L or greater; 124 (11 percent) peaked at 2.42 μmol/L or greater; 56 (5 percent) peaked at 2.90 μmol/L or greater; and 16 (1 percent) had peak levels at or above 3.87 μmol/L (Table 1).

Distribution by Age, Sex, Hispanic Surname, and Residence

The age distribution was that of a working population with 32 percent in their fourth decade (Table 2). Individuals were overwhelmingly male and disproportionately Hispanic surnamed. The blood lead distribution was similar in those with and without Hispanic surnames. Hispanics comprise approximately 24 percent of California's population. Of reports with known zip code, 81 percent were of residents of Los Angeles County (Table 2).

Distribution by Industry

Ninety-one employers were identifiable by name, and accounted for 2,521 (82 percent) reports. SIC codes were not obtainable for 1,109 (36 percent) of reports because the employer identifiers were blank or did not match entries in the California Manufacturer's Directory (Table 3). Based on the laboratory name (indicating that an employer-operated laboratory performed the blood lead analysis), three employers apparently accounted for 67 percent (2,070) of reports.

TABLE 1—Distribution of Blood Lead Reports at or Above 1.21 μmol/L, April–December 1987

Blood Lead Level	Total Reports[a]		Individuals at Peak Lead Level[b]	
μmol/L[c]	No.	%	No.	%
1.21–1.44	717	27	275	24
1.45–1.92	1277	48	508	45
1.93–2.41	451	17	231	20
2.42–2.89	98	4	68	6
2.90–3.37	52	2	26	2
3.38–3.86	24	.9	14	1
3.87–4.34	11	.4	5	.4
4.35–4.82	6	.2	6	.5
4.83–5.30	2	.1	2	.2
5.31+	5	.2	3	.3
Total	2643	100	1138	100

a) 382 reports less than 1.21 μmol/L (25 μg/dl) were received, but not included above.
b) Includes multiple reports on some individuals.
c) 1 μmol/L = 20.7 μg/dl; 10 μg/dl = .483 μmol/L.

TABLE 2—Demographic Characteristics of Individuals with a Blood Lead Report, April–December 1987

Item	No.	Percent
Age (years)[a]		
16–19	8	1
20–29	134	21
30–39	203	32
40–49	136	21
50–59	114	18
60+	45	7
Sex[b]		
Male	1059	94
Female	71	6
Surname[c]		
Non-Hispanic	687	56
Hispanic	547	44
County of Residence[d]		
Los Angeles	1092	81
Other	258	19

a) Excludes 653 reports not stating age.
b) Excludes 162 individuals for whom gender was not stated or determined.
c) Excludes 37 individuals for whom surname was not classified.
d) Excludes missing data (1457 reports) and 27 out-of-state reports.

Apparently, the reporting was done in the context of employer-based medical screening programs mandated by the OSHA lead standard. Three employers engaged in secondary lead smelting, 11 employers engaged in battery manufacture, and 12 brass, lead or copper foundries accounted for 1,656, or 55 percent of all reports. Construction, radiator repair, pottery and ceramics manufacturing, and gun firing ranges accounted for the remainder. Reports of construction workers in demolition and paint stripping represented 0.7 percent (23 of 3,077) of all reports, with 15 percent (15 of 100) of reports exceeding 2.90 μmol/L.

Follow-up of Persons with Elevated Blood Lead Levels

Of the 57 persons with blood lead levels exceeding 2.90 μmol/L, 33 (58 percent) were traced and completed telephone interviews; all persons reported occupational lead exposures. Sixteen reported that their blood lead test was part of an employer-sponsored medical monitoring program; 13 were symptomatic workers who sought medical care from a private physician. At least 10 persons had been hospitalized for treatment, including chelation. Many workers expressed deep reservations and fear of job discrimination when CDHS interviewers proposed pursuing follow-up activities with the employer (even without disclosing the name of the case).

Some individuals apparently covered by the OSHA lead standard had not been removed from their job, and some individuals who had been removed were working at other jobs within the same company at a lower pay rate in apparent violation of the OSHA lead standard. One worker alleged that employers colluded to prevent him from obtaining employment because of a history of elevated blood lead.

Because of worker concerns over potential employment discrimination, the protocol was modified and employer contact was generally limited to emergencies involving reports of blood lead exceeding 3.87 μmol/L. At levels between 2.90 and 3.87 μmol/L, follow-up with the employer was pursued if an employee did not express reservations about contact with the employer, or other workers at the worksite with elevated blood lead levels did not object to contact with the employer.

A number of physicians apparently charged with carrying out employer medical monitoring programs were unfa-

TABLE 3—Distribution of Blood Lead Reports by Industry, April–December 1987*

SIC Code	Industry	Reports**		Individuals		Facilities
		N	%	N	%	N
3341	Secondary lead smelting	1126	37	127	10	3
3691	Battery manufacturing	496	16	246	19	11
3362	Brass foundry	130	4	107	8	12
3721	Aircraft manufacturing	40	1	30	2	1
3269	Pottery products	38	1	20	2	2
9221	Firing range (police)	27	.9	13	1	6
1795	Construction, demolition	17	.6	5	.4	4
7539	Radiator repair	15	.5	8	.6	6
3674	Semi-conductors	9	.3	8	.6	1
7997	Firing ranges (sport)	8	.3	7	.5	4
2782	Printing	7	.2	5	.4	1
1721	Painting contractors	6	.2	3	.2	1
3079	Miscellaneous plastics	6	.2	5	.4	1
4811	Telephone communications	6	.2	4	.3	1
1000	Mining	5	.2	3	.2	2
3351	Copper manufacturing	5	.2	3	.2	1
3699	Electrical machinery	5	.2	5	.4	1
3714	Vehicle parts	5	.2	2	.2	2
——	Missing†	1109	36	677	52	

*Coding of the Standard Industrial Classification (SIC, 1972 Rev.) was based on matching the employer name on the lead report form to those listed in the 1987 California Manufacturer's Directory.

**Less than five reports were received in each of the SICs: inorganic pigment (2816), printing ink (2893), non-ferrous wire (3357), non-ferrous foundry (3360), fabricated metals (3499), special tool manufacturing (3544), local transportation (4111), ship manufacturing and repair (3731), and assay laboratories (7397). Table includes all reports received, including some reports below 1.21 μmol/L.

†Missing data: SIC could not be assigned either because employer identifiers were missing on report, or did not match to entries in the California Manufacturer's Directory (1987).

miliar with the OSHA lead standard. A few physicians were totally unfamiliar with normal limits of blood lead in adults.

Discussion

Before implications of these data can be discussed and conclusions drawn, limitations and potential biases of the data will be discussed. The most important potential bias is the undercounting of lead-exposed individuals with elevated blood lead levels who do not have their blood tested periodically for lead. This prevents the full estimation of the prevalence of elevated blood lead among California adults. Undercounting that differentially affects subgroups also can introduce distortions.

Certain geographic areas and certain industries were probably underrepresented. For example, data from the California Employment Development Department (CEDD) indicated that there were approximately 14,500 employees in 2,790 radiator repair establishments in 1985. However, only three employers, accounting for 15 reports were found in the lead registry in 1987. Similarly, in lead battery manufacturing, where there also is a high probability of direct lead exposure, 11 employers accounting for 496 reports were found in the lead registry, although CEDD data indicated that there were 33 employers with 2,500 workers in June 1985.

The lack of employer-based medical monitoring programs for lead-exposed workers appears to be a significant problem. A recent survey based on a probability sample of California employers indicated that only 2 percent of workers with direct exposures to lead were enrolled in an employer-sponsored, medical monitoring program.[7] Some industries, such as construction, are exempted from the lead standard and medical monitoring requirements.

Underreporting may also result when an out-of-state laboratory analyzed the blood lead of California workers, or an in-state laboratory failed to report. While some out-of-state laboratories did report on a voluntary basis, out-of-state laboratories appear to analyze 47 percent of blood tests of California employers.[7] Since virtually all California laboratories performing lead tests were contacted by CDHS, and failure to report can result in a fine, it is probable that few, if any, California laboratories did not report.

Lack of complete information on basic demographic data such as age, residence, and occupation occurred in a significant proportion of reports. This limits an accurate profile of subgroups where risk may be concentrated and surveillance needs to be expanded.

The timeliness of reporting is another concern for timely case follow-up, particularly for persons with highly elevated blood lead levels. On average, reports were received by CDHS 22 days after blood lead specimens were analyzed by the laboratory. Only 9 percent of report forms were received by CDHS within 72 hours of analysis as required by the reporting requirement. While emergency response was not initially anticipated, follow-up of highly elevated lead cases revealed situations in which imminent hazards had not been abated, and recommendations had or might have had a positive impact.

These data from California's newly implemented blood lead surveillance program indicate the need for intensified lead poisoning surveillance, education, and enforcement in California. Workers, employers, physicians and other health care personnel, laboratories, and government agencies would benefit from expanded training and education focused on lead-related health problems.

The wider implications of this finding is that without an immediate and concentrated effort to reduce occupational lead exposures, it is unlikely that the Public Health Service goal[8] of eliminating lead poisoning by 1990 will be met in California. These data also indicate that current regulations for construction workers exposed to lead do not prevent lead poisoning, and revisions need to be seriously considered. Further, these data indicate the need for occupational health and safety agencies to more vigorously and expeditiously enforce the lead standard.

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Johnson Foundation Announces $3 Million R&D Program to Improve Patient Care

An increasing amount of complex patient care in the United States is being delivered in nursing homes, in ambulatory care clinics, and at home. Yet, the tools and techniques needed to assure that patients receive high quality care in these settings have not yet been fully developed. In an effort to improve services for long-term and ambulatory care patients, the Robert Wood Johnson Foundation of Princeton, NJ, will make available up to $3 million to stimulate and support research and demonstration projects that will identify new and practical mechanisms to assure the delivery of high quality patient care.

The types of projects that will be considered for funding under the foundation's three-year initiative are: research and development of tools that assess the quality of patient care and identify patient care problems and exemplary practices: demonstrations of new approaches to improving quality of care; and evaluations of policies and of systems designed to improve quality of care.

Projects considered for funding may: develop new ways to identify problems in the quality of patient care in specific settings; test existing tools; design and demonstrate institution-wide systems to locate and solve patient care problems; or evaluate existing improvement systems or policies. Projects involving clinical interventions, treatment or drug therapies will *not* be considered under this initiative.

Institutions wishing to apply for funds are asked to submit five copies of a letter of intent of no more than four double-spaced pages, addressed to: Phyllis Kane, Program Assistant, Robert Wood Johnson Foundation, P.O. Box 2316, Route 1 and College Road East, Princeton, NJ, 08543-2316. Based on a review of the letter, the foundation may request a full proposal at a later date.

Technical assistance for the program will be provided by Jack D. McCue, MD, of Baystate Medical Center in Springfield, Mass., and Andrew Kramer, MD, of the University of Colorado Health Sciences Center in Denver.

Recent Time Trends in Uterine Cancer

VICTORIA PERSKY, MD, FAITH DAVIS, PHD, RICHARD BARRETT, PHD, ELIZABETH RUBY, MS, CAROLE SAILER, BA, AND PAUL LEVY, SCD

Abstract: Recent trends in corpus uterine cancer incidence rates were explored using 1979–86 data from the Surveillance and End Results Program (SEER); recent trends in hospitalizations for corpus uterine cancer were explored using 1979–86 data from National Hospital Discharge Surveys (NHDS); and recent trends in exogenous hormone use were delineated using data from the 1980, 1981, and 1985 National Ambulatory Medical Care Surveys (NAMCS). Uterine cancer incidence rates using SEER data have continued to decline since 1979. An acceleration in the decline since 1983–84 is suggested in all women and in women with intact uteri ages 45–64. Hospitalizations for uterine cancer have also declined since 1979, with a marked acceleration in the decline since 1983–84 for all women and for women ages 40–79 has increased 22 percent and use of unopposed exogenous estrogens in women of similar age has increased 7 percent, while use of exogenous progesterones have shown much more substantial increases of approximately 700 percent. Possible relationships between trends in exogenous hormone use and incidence rates of corpus uterine cancer are discussed. (*Am J Public Health* 1990;80:935-939.)

Introduction

Incidence rates of cancer of the corpus uterus showed striking increases in the early 1970s following increases in use of postmenopausal estrogens.[1-3] Subsequently, incidence rates of corpus uterine cancer declined in the late 1970s following declines in use of estrogens.[1] Since 1980, there has been an increase in use of progestins cycled with estrogens in an effort to combat the carcinogenic effect of unopposed estrogens while allowing for a protective effect of estrogens on osteoporosis.[4] The effect of exogenous progesterones on trends in uterine cancer, however, has not been documented. The purpose of the current paper is to delineate more recent time trends in corpus uterine cancer incidence and hospitalization rates in the 1980s in relation to the increasing use of exogenous progestins.

Methods

Age-specific and age-adjusted cancer incidence rates were obtained for the years 1979 through 1986 from the Surveillance Epidemiology and End Results (SEER) Program of the National Cancer Institute. The SEER Program identifies newly diagnosed cancer cases at 11 locations, including nine in the mainland United States plus Hawaii and Puerto Rico. These data, collected since 1973–75, cover approximately 10 percent of the total population of the United States and are considered fairly representative of the US population with respect to age, although Blacks and rural populations are underrepresented. Details of the SEER data collection have been published elsewhere.[5] In the current report, corpus uterine cancer includes ICD-O topography codes 180.0-182.8.[6]

Corpus uterine cancer hospitalization rates between 1979 and 1986 were obtained from the National Hospital Discharge Surveys (NHDS) of the National Center for Health Statistics.[7] Although hospitalization rates are not an exact measure of incidence rates, analyses of trends in the NHDS are presented as a second source of data which, in part, reflect trends in cancer incidence. The NHDS encompasses a representative sample of patients discharged from short stay hospitals, exclusive of federal hospitals, in the 50 states and the District of Columbia. No distinction in NHDS is made between initial admissions and readmissions for corpus uterine cancer. Population estimates are determined by multiplying individual data by sampling weights. In the current report, corpus uterine cancer includes ICD-9 CM code 182.0–182.8.[8]

Hormone use was determined from the National Ambulatory Medical Care Survey (NAMCS) conducted in 1980, 1981, and 1985 with a multistage probability sample of patient visits to office-based physicians in the United States. Physicians were asked to record up to five drugs prescribed at the visit for a random sample of office visits occurring during a randomly selected week during the year.[9,10] Physicians were asked to include both prescription and non-prescription drugs. All medicines containing either estrogens or progesterones were enumerated by year. Birth control pills were not include[d] in these analyses. Medications containing both estrogen and testosterone, however, were included. In 1980, 1981 and 1985, they accounted for an estimated 3.2 percent, 1.0 percent, and 0 percent, respectively, of all estrogens prescribed by physicians for women ages 40–79 in those years. Population estimates were determined by multiplying individual data by sampling weights.

Information on age-specific populations at risk was abstracted from census data.[11] Cancer rates from SEER and NHDS were then calculated by dividing the age-specific estimated numbers by the age-specific populations. In addition, the populations at risk (i.e., women with intact uteri) were establishing by estimating the cumulative age-specific proportions of women who had hysterectomies from hospital discharge data and excluding them from the denominator.[12] Cohort interpolation techniques were used to estimate the proportion of women with intact uteri at single years of age between 1979 and 1986; these figures were then combined to produce five-year rates. Age-adjusted uterine cancer rates from SEER were calculated both by standardization to the 1970 population with uterine cancer not otherwise specified (NOS) included in the data for purposes of comparability to other SEER publications, and by direct standardization to the 1980 population ages 40–79 without NOS included for purposes of comparability with NHDS data. Age-adjusted hospital admissions for uterine cancer rates from NHDS were

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calculated for ages 40–79 by direct standardization to the 1980 population. Age-specific and age-standardized rates were averaged over two-year intervals, to increase the stability of the estimates.

For the NAMCS and NHDS data, standard errors of the age-specific rates were obtained by use of charts, tables, or formulas developed by the National Center for Health Statistics for these surveys. By appropriate algebraic manipulation of these age-specific standard errors, 95 percent confidence intervals were obtained for differences between successive two-year averages of the age-adjusted rates. For the SEER data, the variance among the eight age-adjusted single year rates was computed and this estimated variance was used to obtain 95 percent confidence intervals for differences between successive two-year averages of age-adjusted rates. Also, for the SEER data, linear regression methods were used to estimate the magnitude and statistical significance of the linear component of the time trend in the age-adjusted incidence rates.

Results

For the SEER data, there was a consistent decline each year in the age-adjusted incidence rate from 1979 through 1986 ($p \leq 0.0001$). The average yearly decline in these rates from 1983 to 1986 ($0.77/10^5$) was higher than the decline from 1979 to 1982 ($0.43/10^5$). However, the differences between the two average declines was not statistically significant. As seen in Table 1, age-adjusted uterine cancer incidence rates in SEER declined approximately 1 percent per year between 1979–80 and 1983–84 and 2–3 percent per year between 1983–84 and 1985–86 for all women and for women with intact uteri. Confidence intervals for the declines in age-adjusted rates, however, for each time period included zero. Most of the decrease in uterine cancer rates occurred in the age groups 45–64, with 14–27 percent declines at those ages and with no particular trends apparent in the older and younger age groups (Figure 1).

As seen in Table 2, admissions for uterine cancer in the NHDS declined approximately 1–2 percent per year between the years 1979–80 and 1983–84 and 6 percent per year between 1983–84 and 1985–86. Confidence intervals for the declines in rates between 1983–84 and 1985–86, but not from 1979–80 to 1983–84, excluded zero. Overall trends were similar among different age groups (Figure 2).

As shown in Table 3, between 1980 and 1985 the total number of prescriptions for estrogens increased 22 percent from 11.3 to 13.8 per 100 persons ages 40–79. When women receiving both estrogen and progesterone were subtracted from the estimate, the prescriptions for unopposed estrogen use increased only 7 percent. Confidence intervals for both these increases included zero. During the same time period, however, total progesterone prescriptions increased from 0.6 to 4.9 per 100 persons (694%) and prescriptions for both estrogens and progesterones increased from 0.2 to 1.9 per 100 persons age 40–79 (946%). Confidence intervals for the increases in progesterone use excluded zero.

Discussion

The overall decline in uterine cancer rates since the mid-1970s[1–3] has been attributed primarily to a decrease in the use of unopposed estrogens. This paper documents the continuation of this trend from two different data sources and also suggests that since 1983–84 there may have been an acceleration in this decline in uterine cancer concomitant with little change, or even a slight increase, in unopposed estrogen use. Data from SEER, but not NHDS, suggest that the declines in uterine cancer rates may be localized to age groups between 45–64 years. Exogenous hormones are

TABLE 1—Age-Specific and Age-Adjusted Corpus Uterine Cancer Incidence Rates[a] per 100,000 US Women, 1979–86, for All Women and for Women with Intact Uteri, Surveillance Epidemiology and End Results (SEER)

	All Women				Women with Intact Uteri			
Age (years)	1979–80	1981–82	1983–84	1985–86	1979–80	1981–82	1983–84	1985–86
25–29	0.7	0.9	0.7	0.8	0.7	1.0	0.7	0.8
30–34	2.3	2.8	2.6	2.1	2.5	3.0	2.8	2.3
35–39	5.5	6.7	4.8	6.0	6.4	7.8	5.6	7.0
40–44	12.2	12.1	11.4	13.1	15.6	15.6	14.6	16.9
45–49	26.6	25.8	24.3	21.9	36.7	35.8	34.5	31.6
50–54	55.1	46.1	41.0	41.6	82.7	69.2	61.3	63.1
55–59	79.4	71.4	65.2	60.0	119.2	109.8	102.1	95.2
60–64	118.3	110.3	104.6	85.9	173.3	164.1	160.5	135.5
65–69	110.1	111.9	117.5	110.7	168.0	169.8	177.5	168.7
70–74	100.9	108.8	112.4	111.5	160.0	171.9	177.0	175.1
75–79	86.8	94.9	97.4	92.6	143.9	156.0	159.3	151.0
Age-Adj[b]	70.2	68.0	66.5	62.0	102.6	100.0	98.5	92.7
Decline[c]	−2.2	−1.6	−4.5			−2.6	−1.5	−5.8
(95% CI)[d]	(−8.7,4.5)	(−8.2,5.0)	(−11.1,2.1)			(−10.7,5.5)	(−9.6,6.5)	(−13.9,2.3)
% Decline[e]	−3.1	−2.2	−6.8			−2.5	−1.5	−5.9
Age-Adj[f]	24.5	23.8	23.1	21.6				
Decline[c]	−0.7	−0.7	−1.5					
(95% CI)[d]	(−3.1,1.7)	(−3.2,1.7)	(−3.9,0.9)					
% Decline[e]	−2.9	−2.9	−6.5					

a) Rates were averaged for the years 1979–80, 1981–82, 1983–84, and 1985–86.
b) Age-adjusted for the ages 40–79 by the direct method to the 1980 population of women ages 40–79.
c) Absolute decline in age-adjusted rates.
d) 95% confidence limits for the decline in age-adjusted rates.
e) Percentage decline in age-adjusted rates.
f) Age-adjusted for all ages by the direct method to the 1970 population.

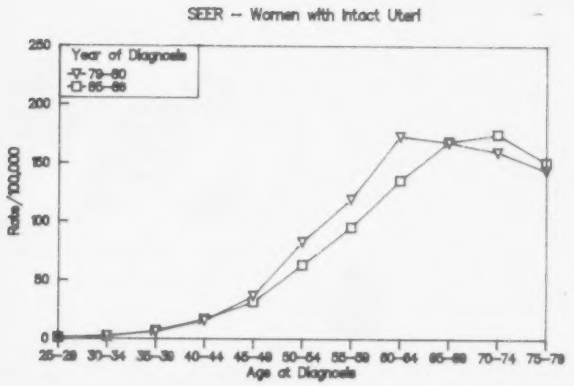

FIGURE 1—Age-Specific Uterine Cancer Incidence Rates by Year of Diagnosis: SEER—Women with Intact Uteri

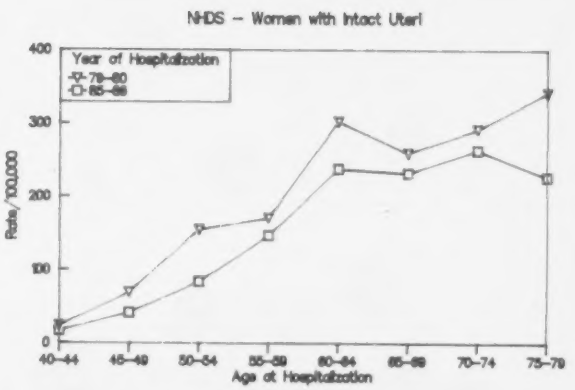

FIGURE 2—Age-Specific Uterine Cancer Hospitalization Rates by Year of Hospitalization: NHDS—Women with Intact Uteri

thought to act at the promotional stage of uterine corpus carcinogenesis and to act within a short period of time: in the 1970s, trends in cancer rates followed trends in hormone use by only a few years[1] and case-control studies[13] estimate latent periods of only three to six years. Thus it is possible that the decline since 1979 partly reflects a very recent increase in use of postmenopausal progestins. A protective effect of progestins is consistent with lower risks of uterine cancer observed in women on birth control pills containing both estrogens and progesterones,[14–16] and with two recent studies in which post-menopausal women using estrogen-progesterone combinations were at somewhat lower risk of uterine cancer (OR = 0.6, CI = 0.2–1.4) and (OR = 0.9, CI = 0.4–2.0) than women not using hormones.[17–18] Previous studies have also noted that adenomatous hyperplasia, thought to be a precursor to endometrial cancer, is reversed by the addition of progesterone.[19] Similarly, estrogen receptor activity is increased by estrogens and decreased by progesterones, lending biologic plausibility to a protective action of progesterone on estrogen-induced cancer.[20]

Another possible explanation for the declines in uterine cancer rates since 1979 is a protective effect of birth control pills on cancer development, manifesting itself 10–20 years after exposure to contraceptives. Women currently ages 45–64 were ages 20–40 when oral contraceptives were first introduced. Older women were never exposed to oral contraceptives and were unlikely to have been exposed to postmenopausal progestins, which have been used only since 1970 and are used infrequently for women over age 65 years. In the current study, only 3–5 percent of progestins being prescribed for women in the years 1980, 1981, and 1985 were prescribed for women over 65 years of age.

Hospital discharge data cannot accurately measure uterine cancer incidence rates because hospitalizations, rather than people, are enumerated, and the same person hospitalized repeatedly for the same illness may be counted more than once. Rates of hospitalization for cancer in NHDS are higher than cancer incidence rates in SEER.[21] In the current study we found that hospitalization rates for uterine cancer based on NHDS were approximately twice the incidence rates for uterine cancer in SEER. Although much of the acceleration in the declines in hospitalizations seen since 1983–84 might be attributed to overall changes in hospitalizations secondary to the introduction of DRGs (diagnosis-

TABLE 2—Age-Specific and Age-Adjusted Hospital Admission Rates[a] For Uterine Cancer (per 100,000) US Women, 1979–86, for all Women and for Women with Intact Uteri, National Hospital Discharge Survey (NHDS)

	All Women				Women with Intact Uteri			
Age (years)	1979–80	1981–82	1983–84	1985–86	1979–80	1981–82	1983–84	1985–86
40–44	18.5	24.7	13.2	12.8	23.6	31.8	16.8	16.5
45–49	50.0	28.7	37.5	28.8	69.0	39.9	53.1	41.5
50–54	102.9	67.1	61.8	55.1	154.6	100.6	92.4	83.8
55–59	113.3	117.5	112.4	92.5	170.3	180.6	176.1	146.8
60–64	206.7	209.9	183.9	151.1	302.8	320.1	282.0	237.9
65–69	170.3	229.4	215.7	152.2	259.7	347.9	325.6	232.0
70–74	184.6	216.0	184.8	168.0	292.0	340.9	290.8	263.6
75–79	206.5	156.0	171.7	139.4	342.2	256.3	280.3	226.9
Age-adj[b]	122.0	122.5	113.1	92.1	178.4	179.9	167.5	138.0
Decline[c]	−8.8		−21.0		−10.8		−29.6	
(95% CI)[d]	(−22.6,5.0)		(−30.4,−1.2)		(−31.9,10.3)		(−44.1,−1.5)	
% Decline[e]	−7.3		−18.6		−6.1		−17.6	

a) Rates were averaged for the years 1979–80, 1981–82, 1983–84, and 1985–86.
b) Age-adjusted by the direct method to the 1980 population of US women ages 40–79
c) The decline in averaged age-adjusted rates were calculated for the time periods 1979–80 to 1983–84 and 1983–84 to 1985–86.
d) 95% confidence intervals for the decline in averaged age-adjusted rates.
e) The percentage decline in averaged age-adjusted rates were calculated for the time periods 1979–80 to 1983–84 and 1983–84 to 1985–86.

TABLE 3—Age-Adjusted Exogenous Hormones Use per 100 Persons by Year for Ages 40–79, Adjusted to 1980 Age Distribution, National Ambulatory Medical Care Survey (NAMCS)

Hormone	Years			Change 1980–85 (95% CI)*	Percent Change 1980–85
	1980	1981	1985		
All Estrogen	11.29	11.23	13.81	2.52 (−1.48,6.51)	+22%
Unopposed Estrogens	11.11	10.91	11.88	0.78 (−3.06,4.61)	+7%
Estrogens and Progesterones	0.18	0.32	1.92	1.74 (0.78,2.70)	+946%
All Progesterones	0.61	1.25	4.87	4.26 (2.96,5.55)	+694%

*95% confidence intervals for change in rate 1980–85.

related groups) preferentially affecting the elderly, the accelerations in the overall declines in incidence rates for corpus uterine cancer in SEER suggest that there may have been alterations in trends in incidence rates as well.

The use of physician office visit prescriptions to estimate hormone use is subject to several potential errors. Although total use of progesterone can be approximated, use of progesterone simultaneously with estrogen cannot be precisely measured, since the NAMCS measures prescriptions at an individual visit, rather than total prescriptions. In addition, although every effort was made to eliminate birth control pills from the analysis, it is possible that some of the younger women may have been using birth control pills, rather than estrogen and progesterone, as noted. The restriction of the analyses to women ages 40–79 should minimize this error.

The number of drug prescriptions may well be underestimated, since physicians were limited to a total of five drugs prescribed at each visit. A recent study using data from the National Disease and Therapeutic Index (NDTI) reported between 15 and 43 total oral estrogen mentions per 100 women ages 45–64 in 1985.[4] They also noted a substantial increase in the percent of women prescribed both estrogens and progesterone. Use of both hormones account for 4 percent of all estrogen mentions in 1979 as compared to 28 percent in 1986. Unlike the NAMCS in the current study, the NDTI includes outpatient clinics, prescriptions written in hospitals, and telephone prescriptions. Since ascertainment of drug use in the NHDS, however, was similar in each year of the study, systematic underestimates of exogenous hormone use should not affect time trends.

A detailed breakdown of specific estrogens used also revealed that a small percentage of women in 1980 and 1981 ingested hormones containing both estrogens and testosterone. A total of 3.2 percent of women using estrogens in 1980, 1.0 percent of women using estrogens in 1981, and 0 percent using estrogens in 1985 used medication also containing testosterone. These results are consistent with those of Hemminki, *et al*,[4] who also noted that injected androgen/estrogen preparations decreased from 7 percent in 1974 to 3 percent in 1986. We were not able to find any previous human studies which examined the effect of exogenous testosterone on endometrial hyperplasia. Studies in rabbits[22] and in endometrial adenocarcinoma in vitro cell lines,[23] however, indicate that testosterone has no effect on the development of hyperplasia or on tumor cell growth. Since only a small number of women in 1980 and 1981 were using testosterone, they were retained in the analysis. Finally, the numbers of women using progesterone in specific age strata were small, precluding reports of age-specific rates. Despite the problems in estimating estrogen and progesterone use, consistency with other data and the magnitude of the increase between 1980 and 1985 support the possibility that the increase in use of progestins may be contributing to an acceleration in the decline in uterine cancer since 1984.

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Cancer Mortality in Native Americans in North Carolina

RONNIE D. HORNER, PHD

Abstract: This paper describes age-adjusted mortality from malignant neoplasms for Native Americans in North Carolina for 1968–72 and 1978–82. Sex-specific standardized mortality ratios were calculated from death certificate data, using the cancer mortality experience of White North Carolinians to obtain the number of expected deaths. For most categories and specific sites of cancer, mortality was at or below the expected level, but higher than expected mortality was found for genitourinary cancers in males (SMR = 1.62, 95% CI = 1.15, 2.21) for the 1978–82 period; within this category, there was a higher than expected level of mortality from prostate cancer (SMR = 2.00; 95% CI = 1.36, 2.83) and cancer of the penis and other male genital organs (SMR = 9.09; 95% CI = 1.10, 32.84). Female Native Americans had an elevated mortality from cervical cancer (SMR = 2.27, 95% CI = 1.09, 4.17) for the 1968–72 period only. (*Am J Public Health* 1990; 80:940–944.)

Introduction

The epidemiology of cancer in Native American tribes in the eastern United States is not well described. Studies of the Seneca Nation in New York State found lower than expected mortality from cancers of all sites for males and females of this tribe.[1,2] In comparison to White residents of New York State (exclusive of New York City), Seneca women had lower mortality from cancers of the pancreas and breast, but higher mortality from cervical cancer; Seneca men had site-specific cancer mortality at or below the expected level.[1]

Native Americans nationally and in the Southwest show a lower than expected overall cancer mortality and incidence.[3,4] Alaskan Native Americans experience a level of mortality and incidence from all cancers which is similar to White Americans.[5–9] For specific sites, elevated mortality (and incidence) is found for cancer of the gallbladder (particularly for females),[3,7,10–13] esophagus and nasopharynx (for Alaskan Native Americans),[5,6,8,14,15] stomach,[16,17] cervix,[3,5,8,18,19] and multiple myeloma (in males).[20] Significantly lower mortality (and incidence) is found for lung cancer,[3,5,7,16,21] cancer of the female breast,[3,6,7,8,16] melanomas,[3,6] lymphomas and leukemias,[20] pancreatic cancer,[3,16] colon cancer,[3] and cancer of the prostate gland (particularly for Eskimos and Aleuts).[3,5–8] Alaskan Native Americans have an increasing incidence of lung cancer for both sexes; for females, esophageal and cervical cancers are decreasing.[6,7,9]

This report describes cancer mortality in Native Americans in North Carolina for the 1968–72 and 1978–82 periods, to gain insights into possible changes over time.

Methods

Native Americans in North Carolina are from several tribes, including the Cherokee, Coharie, Eno-Occan-Eechee, Haliwa-Saponi, Lumbee, Meherrin, Tuscardra, and Waccamaw-Sioun. The Cherokee in western North Carolina and the Lumbee in the eastern region of the State are the largest tribes. There has been considerable interbreeding between Native Americans, Whites and, to a lesser extent, Blacks in the region.[22–25] As a consequence, there are few "full-blooded" Native Americans in any of the tribes in the State. Moreover, cancer mortality data by tribe are not available.

Population data for this study came from the 1970 and 1980 US census reports for North Carolina.[26,27] Sixteen age categories were used: <5 years old, 5–9, 10–14, . . ., 70–74, and 75+ years of age. The distribution of Native Americans by sex but not age was available for 1970, so the sex-specific age distributions of Native Americans for 1980 were used to estimate the 1970 age distributions.

Cancer mortality data were obtained from the detailed mortality data tapes of the North Carolina State Center for Health Statistics using the underlying cause of death for North Carolina residents. Probably some Native Americans were classified as White. (The magnitude of the racial misclassification bias is currently under investigation: Personal communication from Dr. Tim E. Aldrich, director of the Central Cancer Registry of North Carolina.)

The mortality data were classified according to the broad categories of malignant neoplasms found in the International Classification of Diseases (ICD) and by specific sites within these categories.

The use of broad categories was necessary because the data for specific cancer sites for Native Americans in North Carolina were sparse. The use of broad categories also reduced the effect of changes in the classification of cancers; ICD-8 and ICD-9, though, are essentially identical for malignant neoplasms at the three-digit code level.

Specific sites identified by previous studies as being above or below the expected level were also given attention.

The standardized mortality ratio (SMR) was calculated using the 1980 age- and sex-specific cancer mortality rates of White residents to obtain the expected number of deaths. Although SMRs between different areas (times) usually should not be compared because of different standards, the identical age distribution in the Native American population in 1970 and 1980 (for the reason described) and the use of the same set of rates permit comparisons between time periods.[28] In essence, the only difference between the 1970 and 1980 SMRs are the cancer mortality rates for Native Americans which are reflected in the observed number of deaths. Cancer deaths were assumed to follow a Poisson distribution. The 95% confidence intervals for the sex-specific SMRs were calculated according to the approach described by Kahn and Sempos.[29]

Results

The proportion of observed cancer deaths by category among Native American and White residents of North Carolina in 1968–72 and 1978–82 is shown in Table 1. For

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TABLE 1—Percentage Distribution of Deaths from Malignant Neoplasms, Native Americans and White Residents of North Carolina, 1968–72 and 1978–82, by Sex and Category*

Category of Malignancy (ICD Code)	Native Americans		Whites	
	1968–72	1978–82	1968–72	1978–82
Males	(n = 76)	(n = 150)	(n = 13,747)	(n = 20,317)
Lip, Oral Cavity, and Pharynx (140–149)	1.3	2.7	3.3	2.3
Digestive Organs and Peritoneum (150–159)	27.6	19.3	22.5	21.5
Respiratory and Intrathoracic Organs (160–165)	19.7	30.0	32.8	38.2
Bone, Connective Tissue, Skin, and Breast (170–175)	1.3	0.7	3.7	3.2
Genitourinary Organs (179–189)	23.7	26.0	14.8	14.4
Lymphatic and Hematopoietic Tissue (200–208)	14.5	11.3	11.1	9.2
Other and Unspecified Sites (190–199)	11.8	10.0	11.8	11.3
Females	(n = 44)	(n = 89)	(n = 11,268)	(n = 16,285)
Lip, Oral Cavity, and Pharynx (140–149)	2.3	3.4	2.0	1.6
Digestive Organs and Peritoneum (150–159)	18.2	27.0	25.5	25.2
Respiratory and Intrathoracic Organs (160–165)	2.3	3.4	7.3	13.8
Bone, Connective Tissue, Skin, and Breast (170–175)	18.2	19.1	22.7	22.1
Genitourinary Organs (179–189)	36.4	19.1	18.4	14.7
Lymphatic and Hematopoietic Tissue (200–208)	9.1	13.5	10.8	10.2
Other and Unspecified Sites (190–199)	13.6	14.6	13.2	12.4

*Percentages may not sum to 100% due to rounding.

White residents of both sexes, the proportion of cancer deaths within each category is fairly stable between the two periods. Among male Native Americans, deaths from cancer of the digestive organs and peritoneum decreased, but deaths from cancers of the respiratory and intrathoracic organs increased. For female Native Americans, deaths from cancers of the digestive organs and the peritoneum increased, but genitourinary cancer deaths decreased. None of the differences between time periods were statistically significant.

As Table 2 shows, Native Americans experienced a generally lower or equal level of mortality from the various cancers to that expected based on the experience of White North Carolinians. The only clearly elevated SMR is for genitourinary neoplasms in male Native Americans for the 1978–82 period (SMR = 1.62, 95% CI = 1.15, 2.21); the SMR for this category is also elevated for 1968–72.

The SMRs in Table 2 suggest that changes in cancer mortality for Native Americans may have occurred between 1968–72 and 1978–82. There is an increase in the number of cancer deaths for Native American females from significantly below to near the expected levels for digestive organ and peritoneum cancers. A similar change occurs for respiratory and intrathoracic cancer mortality for male Native Americans. Also as noted previously, deaths from genitourinary cancers increased from a level similar to the expected number to a higher number for male Native Americans. Both sexes may also be experiencing an increase in cancers of the lip, oral cavity, and pharynx.

Table 3 shows the SMRs associated with specific cancer sites which have been identified as having significantly above or below expected mortality for Native Americans. The SMRs for each three-digit site are available on request to the author. Most deaths from cancer at the various anatomic sites are at levels similar to that found among White residents of North Carolina. Deaths from cancer of the breast and colon are lower than expected in both time periods for females. Mortality from cancer of the cervix uteri was more than twice that of Whites in 1968–72 and in 1978–82. A slight increase in mortality from prostatic cancer is indicated; and rates are elevated in both periods. Mortality from cancer of the penis and other male genital organs (ICD 187) is also significantly elevated in both periods.

Discussion

The Native Americans in this report represent several tribes which have bred extensively with other racial groups, and yet their cancer mortality experience generally follows that of Native Americans elsewhere in the nation, with lower than expected mortality from cancers of all sites, respiratory and intrathoracic cancers, and cancers of the female breast, but a higher than expected mortality for cancer of the cervix uteri. The higher mortality from prostate cancer seen in this study was also found for Alaskan Indians.[3,5,6,8] A unique finding of this study is the elevated mortality from cancer of the penis and other male genital organs (ICD 187). Unlike Alaskan Native Americans, no nasopharynx cancers were reported in either study period. Nor was there a higher than expected mortality from cancers of the gallbladder and stomach which are common in tribes of the Southwest.

Clearly elevated mortality in North Carolinian Native Americans was found only for cancers of the prostate, male

TABLE 2—Standardized Mortality Ratios (95% CI) for Malignant Neoplasms, Native American Residents of North Carolina, 1968–72 and 1978–82, by Sex and Category

Category of Malignancy (ICD Code)	Males		Females	
	1968–72 (95% CI)	1978–82 (95% CI)	1968–72 (95% CI)	1978–82 (95% CI)
All Sites (140–208)	0.79 (0.62, 0.99)	0.87 (0.73, 1.02)	0.54 (0.39, 0.73)	0.71 (0.57, 0.87)
Lip, Oral Cavity and Pharynx (140–149)	0.32 (0.01, 1.80)	1.02 (0.28, 2.61)	0.64 (0.02, 3.57)	1.61 (0.33, 4.71)
Digestive Organs and Peritoneum (150–159)	1.02 (0.63, 1.57)	0.80 (0.53, 1.14)	0.41 (0.18, 0.82)	0.85 (0.54, 0.98)
Respiratory and Intrathoracic Organs (160–165)	0.48 (0.27, 0.79)	0.70 (0.51, 0.94)	0.17 (0.004, 0.94)	0.17 (0.03, 0.49)
Bone, Connective Tissue, Skin, and Breast (170–175)	0.25 (0.01, 1.38)	0.16 (0.004, 0.90)	0.42 (0.18, 0.82)	0.57 (0.33, 0.91)
Genitourinary Organs (179–189)	1.39 (0.82, 2.20)	1.62 (1.15, 2.21)	1.06 (0.61, 1.72)	0.93 (0.54, 1.48)
Lymphatic and Hematopoietic Tissue (200–208)	0.92 (0.46, 1.64)	0.85 (0.47, 1.40)	0.42 (0.12, 1.09)	0.91 (0.47, 1.59)
Other and Unspecified Sites (190–199)	0.74 (0.34, 1.41)	0.83 (0.48, 1.33)	0.55 (0.20, 1.20)	0.84 (0.44, 1.43)

TABLE 3—Standardized Mortality Ratios (95% CI) for Selected Malignant Neoplasms, Native American Residents of North Carolina, 1968–72 and 1978–82, by Sex

Site of Malignancy (ICD Code)	Males		Females	
	1968–72 (95% CI)	1978–82 (95% CI)	1968–72 (95% CI)	1978–82 (95% CI)
Nasopharynx (147)	0.00 (0.00, 12.30)	0.00 (0.00, 11.53)	0.00 (0.00, 26.34)	0.00 (0.00, 23.06)
Esophagus (150)	0.00 (0.00, 2.20)	0.97 (0.20, 2.83)	1.16 (0.03, 6.48)	3.57 (0.93, 9.14)
Stomach (151)	1.42 (0.46, 3.31)	1.60 (0.64, 3.29)	0.82 (0.10, 2.96)	1.47 (0.40, 3.77)
Colon (153)	0.64 (0.17, 1.63)	0.38 (0.12, 0.88)	0.24 (0.03, 0.87)	0.31 (0.08, 0.79)
Gallbladder (156)	0.00 (0.00, 5.76)	0.00 (0.00, 3.80)	0.84 (0.02, 6.07)	2.42 (0.50, 7.07)
Pancreas (157)	1.93 (0.93, 3.55)	1.00 (0.46, 1.90)	0.52 (0.06, 1.88)	0.95 (0.35, 2.08)
Trachea, Bronchus or Lung (162)	0.44 (0.23, 0.75)	0.71 (0.52, 0.96)	0.18 (0.005, 1.01)	0.17 (0.04, 0.51)
Breast, Female (174)	—	—	0.37 (0.13, 0.77)	0.59 (0.34, 0.99)
Cervix Uteri (180)	—	—	2.27 (1.09, 4.17)	2.30 (0.99, 4.53)
Prostate (185)	1.70 (0.91, 2.91)	2.00 (1.36, 2.83)	—	—
Penis and other male genital organs (187)	22.22 (2.69, 80.27)	9.09 (1.10, 32.84)	—	—
Lymphomas (200–202)	0.46 (0.06, 1.65)	0.30 (0.04, 1.10)	0.28 (0.01, 1.56)	0.84 (0.23, 2.16)
Multiple Myeloma (203)	0.72 (0.02, 4.04)	1.48 (0.40, 3.24)	0.00 (0.00, 3.32)	1.98 (0.54, 5.07)
Leukemias (204–208)	1.28 (0.55, 2.53)	1.07 (0.49, 2.03)	0.56 (0.12, 1.64)	0.63 (0.17, 1.60)

genitalia, and the cervix. Prostatic and penile cancers are more prevalent in Blacks than either Whites or Native Americans generally.[30,31] However, the racial mixing in Native Americans of North Carolina is unlikely to account for their unexpectedly higher mortality from these cancers because the Black contribution to the genetic pool of most tribes of the Carolinas is indicated to be minimal.[22–25] Diet may be involved in cancer of the prostate. Although controversial, there is epidemiologic evidence of a positive association between prostatic cancer and a diet high in fats and vitamin A, particularly in older males.[32–35] However, an inverse association between the level of consumption of green and yellow vegetables (and hence vitamin A) and prostatic cancer is found for Japanese males.[36] Dietary information on North Carolinian Native Americans is not available. The elevated mortality from cervical cancer may indicate a low level of cervical cancer screening among Native American females in North Carolina. An inverse association between extent of cervical cancer screening in populations and mortality from cancer of the cervix is reported for Native Americans elsewhere.[18,19]

There is indication that the cancer mortality experience of North Carolinian Native Americans may be worsening, which would suggest that the lower than expected cancer mortality for Native Americans is more strongly related to environmental factors than to genetics. Although the number of cases is too few for definitive conclusions, an increase in mortality may be occurring from: cancer of the lip, oral cavity, and pharynx for both sexes; cancers of the respiratory and intrathoracic organs, and genitourinary organs for males; and cancers of the digestive organs and peritoneum for females. For specific sites, an increase in mortality may be occurring for cancer of the prostate and the trachea, bronchus, and lung for males. It has been suggested that lung cancer mortality is increasing in Alaskan Native Americans as well.[6,7] Information on the use of tobacco products such as snuff and chewing tobacco by Native Americans in North Carolina is not available, although several reports indicate high prevalence of tobacco use among younger Native Americans elsewhere.[37–40]

It is recognized that the patterns reported in this study may reflect several biases. There is the possibility of racial misclassification bias in the mortality statistics, although its presence and magnitude have yet to be determined. Moreover, the age-specific population data for Native Americans in 1970 are only estimates. Diagnostic bias may also be present; however, the proportion of "other and unspecified" cancers has remained stable over time. The data also reflect coding under the eighth and ninth ICD; however, the two revisions are essentially identical for neoplasia. Perhaps of more concern, the data were aggregated across tribes which could obscure patterns of cancer mortality within tribes.

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HUD Issues Guidelines on Lead-Based Paint Abatement

The US Department of Housing and Urban Development (HUD) has issued the first federal guidelines for the identification and abatement of lead-based paint in all public and Indian housing developments. The interim guidelines, signed by HUD Secretary Jack Kemp and published in the Federal Register, became effective April 1 and apply to all rehabilitation programs in public housing from that date forward. While the guidelines are recommendations and not mandatory, they represent the first national technical rules and procedures for testing, abatement worker protection, clean-up and disposal of lead-based paint in residential structures. The standards outlined in the 160-page document include several procedures for lead paint testing and removal that have proven effective in cities where lead abatement programs are mandated, such as Baltimore and Boston. Lead-based paint is believed to exist in about 40 million homes, or 40 percent of the country's housing stock where lead paint was used prior to 1970.

The federal guidelines recommend three accepted abatement strategies: complete replacement of all lead-painted surfaces, encapsulation or sealing of lead-painted areas, or paint removal. The guidelines outline the various methods of paint stripping, sanding and chemical processing, and the appropriate precautions to be taken with each, including daily and final clean-up. Proper clean-up procedures involve a thorough vacuuming, using a High Efficiency Particulate Air (HEPA)-filtered vacuum, followed by wet wiping with a high-phosphate solution, followed by a second HEPA vacuuming. The guidelines also recommend that sanding flat surfaces should be done with a sander attached to a HEPA vacuum that can collect the dust particles as they are released. Nilfisk of America, Inc., of Malvern, PA, one of the nation's leading manufactures of HEPA-filtered vacuums used in removal of lead, asbestos, silica, fiberglass and other harmful dusts, was asked by the National Institute of Building Sciences to participate in drafting the rules and recommendations that formed the basis for HUD's interim guidelines. For further information, contact HUD Headquarters, 451 7th Street, SW, Washington, DC 20410. Tel: (202) 755-6422.

Defining Arthritis and Measuring Functional Status in Elders: Methodological Issues in the Study of Disease and Physical Disability

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Abstract: This study explores the degree to which the association of knee osteoarthritis with physical disability changes with different definitions of osteoarthritis in 1,416 Framingham Study elders. When all categories of radiographic osteoarthritis were aggregated into a single "arthritis" variable and dependence upon human assistance in one or more functional activities combined into a single variable, elders with osteoarthritis had only moderately increased odds for dependence after controlling for age or sex (OR = 1.25). A definition of osteoarthritis based only upon symptoms produced larger odds for dependency (OR = 1.85). Elders with mild osteoarthritis and infrequent knee pain had no significantly elevated risk for dependence in any of the seven functional activities. Elders with infrequently symptomatic, moderate to marked radiographic osteoarthritis were at increased risk for dependence in stair climbing, walking a mile, housekeeping, and carrying bundles. Elders with radiographic osteoarthritis accompanied by frequent pain had an increased odds of dependence in stair climbing, walking a mile, and housekeeping. Using a generic definition of "arthritis" and aggregating functional activities may underestimate the impact of osteoarthritis on physical disability in the elderly and obscure the task-specific nature of that relationship. (*Am J Public Health* 1990; 80:945–949.)

Introduction

Diseases are often defined broadly in epidemiologic studies of the elderly and ascertained by self-report and clinical proxy measures such as symptoms. Using "arthritis" as a generic term, previous studies have provided broad insights into the relation between "arthritis" and disability.[1–4] These results do not necessarily provide us with information that captures the relation of a particular type and severity of arthritis or arthritic symptoms to functional limitations. Osteoarthritis refers to a specific pathological process within joints which is well-captured by radiographs that can be used to grade disease severity. The presence or absence of joint symptoms creates another dimension of disease definition.

A second issue in the study of disease and disability that requires examination is specification of the functional outcome. Combining several kinds of functional items into a single index for an overall score to estimate prevalence of disability[5–9] may be justified when the disease affects functional activities globally (e.g. the effects of stroke on activities of daily living) or when the summary score aggregates functional tasks of the same biomechanical or sensorimotor type (e.g. hand dexterity). However, a summary score may obscure the impacts of a disease on specific functional activities, particularly if many of the other activities included in the index are relatively unaffected by that disease.

Osteoarthritis is the most common form of arthritis in the elderly, and osteoarthritis of the knee is commonly associated with clinical symptoms and disability.[10] The first purpose of this study is to identify how the association of knee osteoarthritis with physical disability among elders changes using specific radiographic grades of osteoarthritis and the presence of symptoms to define the disease. The second purpose is to use these definitions to explore the association of knee osteoarthritis with discrete limitations in particular functional activities.

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Methods

Study Population and Sample

The study population was the Framingham Heart Study cohort, a population-based cohort of the adult residents of Framingham, Massachusetts, designed to investigate factors associated with cardiovascular disease, and examined biennially since 1948. The prevalence of knee osteoarthritis during the 18th biennial examination (1983–85) has been reported elsewhere.[11] There were 1,826 participants in examination 18, of whom 1424 received knee radiographs. Eight persons were excluded from the present study; four with bilateral total knee replacements, another four with rheumatoid arthritis, leaving 1,416 cohort members.

Definitions of OA and Pain

Osteoarthritis (OA) was graded by an academically based bone and joint radiologist using the criteria of Kellgren and Lawrence:[12] grade 0 = normal radiograph; grade 1 = possible osteophytes; grade 2 = definite osteophytes; grade 3 = osteophytes and joint space narrowing; and grade 4 = large osteophytes, marked narrowing of joint space and definite deformity. Most studies have used grade 2 (the presence of definite osteophytes) as the criterion for defining disease, while a few others have required evidence of joint space narrowing (grade 3), corresponding to clinical definitions of disease, to designate osteoarthritis.[10] The radiographic grade for each subject in this study was assigned according to the most severely affected knee. Using the approach of the first National Health and Nutrition Examination Survey,[13] symptoms were defined as pain in or around the knee lasting at least a month within the previous year. Respondents who had either no symptoms or who may have had only infrequent pain were designated as asymptomatic.

We created three different definitions of radiographic osteoarthritis: asymptomatic grade 2 osteoarthritis, asymptomatic ≥ grade 3 osteoarthritis, and symptomatic ≥ grade 2 osteoarthritis. Subjects with symptoms were classified as those without radiographic evidence of disease and those with ≥ grade 2 osteoarthritis.

Measures of Physical Disability

Data were collected on 16 measures of physical disability at examination 18. Six items measuring the most basic activities of daily living were rejected for inclusion as we found little disability on these items, typical in a sample of

elders living independently in the community. Another three items measuring more complex activities were rejected for similar reasons. Of the seven remaining functional measures, two items covered activities explicitly involving the lower extremities: walking up and down a flight of stairs, and walking a mile; each of these items was adapted from the Rosow-Breslau Functional Health Index,[14] and had been used previously at Framingham. Five measures covered functional tasks requiring both upper and lower extremity use: housekeeping, heavy home chores including shoveling snow and washing windows, cooking, grocery shopping and carrying bundles weighing 10 pounds. The item on heavy home chores came from the Rosow-Breslau Functional Health Index; the last item was taken from an instrument developed by Nagi[15]; the items on housekeeping, cooking, and grocery shopping had been previously used at Framingham.[16]

Items from the Rosow-Breslau were recorded as able or unable to do without help. The items on housekeeping, cooking, and grocery shopping asked whether the respondent could perform that task entirely without help from another person. The item on carrying bundles was part of a physical performance battery administered by a trained nurse. It was scored as independent, dependent upon an assistive device, dependent upon human assistance, or unable to do. In order to make the definition of the dependent variable comparable across items, the need for human assistance was chosen as the criterion of disability on all seven dependent variables.

Data Analysis

We calculated two sets of prevalence odds ratios for dependence in performing each of the seven functional activities using logistic regression analyses, controlling for age as a continuous variable and sex. An odds ratio was defined as the relative odds of functional dependence among subjects with a specific definition of knee osteoarthritis divided by the relative odds of functional dependence among subjects without knee pain or osteoarthritis. The first logistic regression model explored odds for those elders with radiographic osteoarthritis. The second model explored odds for those elders with symptoms or symptoms plus radiographic disease.

Results

The age and sex of the subjects in our sample are described in Table 1. There were 589 men and 827 women in our sample (mean age = 72.7 years). There were 214 subjects with asymptomatic grade 2 osteoarthritis, 151 with asymptomatic osteoarthritis ≥ grade 3, 103 with knee pain and at least grade 2 osteoarthritis, and 57 with knee symptoms but no radiographic evidence of disease; 891 elders without knee pain or radiographic osteoarthritis served as controls. Due to missing data on functional measures, the number of elders in each of these groups is slightly lower in the results reported below.

The results of aggregating all radiographic osteoarthritic categories or using symptom reports as exposure variables while combining dependence in one or more functional activities into a single dependent variable are displayed in Table 2. Based upon these data, 37.5 percent of the subjects with osteoarthritis were dependent in one or more ADL compared to 29.8 percent of their peers without osteoarthritis. Furthermore, elders with osteoarthritis had only modestly increased odds for dependence in one or more functional activities after controlling for age and sex. Using a symptom-based definition of osteoarthritis, without respect to radiographic disease, showed an even larger proportion of disabled elders (46.2 percent). These elders had significantly increased odds for dependency in one or more functional activities. Using specific definitions of osteoarthritis and disaggregating functional measures, however, demonstrated quite different results.

The odds ratios for dependence upon human assistance to perform the two lower extremity activities are shown in Table 3. Overall, the elders in our sample exhibited little dependence in either activity. There were, however, substantially more elders in both the osteoarthritis and nonosteoarthritis groups dependent in walking a mile (14.2 percent) than dependent in stair climbing (3.2 percent). Combining all specifications of osteoarthritis into a single category reveals an increased odds ratio of dependence in each of these activities. Analysis by radiographic grade, however, demonstrates substantially lower and odds ratios for those elders with asymptomatic grade 2 osteoarthritis. Subjects with symptomatic osteoarthritis or asymptomatic osteoarthritis ≥ grade 3 had substantially increased odds for dependence in both lower extremity ADL. Elders who were classified as "arthritic" using symptom-based definitions had moderately increased odds for dependence in both lower extremity tasks.

Table 4 displays the odds ratios for dependence in each of the five complex ADL that combine both upper and lower extremity function. The largest proportion (27 percent) of disabled elders was found among those who relied upon human assistance for heavy home chores; there was little functional dependence among our subjects in the other four activities.

Considering all elders with any radiographic osteoarthritis as a single group showed very modest increased odds for dependence in any complex ADL. Subjects with asymptomatic grade 2 osteoarthritis showed no increased odds for dependence in these five activities. Elders with asymptomatic OA ≥ grade 3 had increased risks for dependence in housekeeping and carrying bundles. The odds for dependence among elders with symptomatic radiographic disease

TABLE 1—Sex and Age of Subjects (N = 1416)

Subjects	Male N	Male %	Female N	Female %
Sex	589		827	
Age (years)				
≤69	227	38.5	300	36.3
70–79	270	45.8	413	49.9
≥80	92	15.2	114	13.8
mean ± SD	72.6 ± 6.11		72.7 ± 5.98	
range	64–92		63–93	

TABLE 2—Odds Ratios for Dependence upon Human Assistance in One or More Functional Activities Aggregating All Grades of OA and All Reports of Knee Symptoms Adjusted for Age and Sex

	Proportion Disabled	(%)	Adjusted OR	(95% CI)
No OA, no knee symptoms	261/875	(29.8)	1.0	
All Radiographic Grades ≥ 2	172/459	(37.5)	1.25	(0.97, 1.60)
All Knee Symptoms	73/158	(46.2)	1.85	(1.30, 2.65)

TABLE 3—Odds Ratios for Dependence upon Human Assistance to Perform Lower Extremity Activity by Grade of Osteoarthritis and Presence of Knee Pain Adjusted for Age and Sex

Activity	Proportion Disabled	(%)	Adjusted OR	(95% CI)
Stairclimbing				
All subjects	42/1321	(3.2)		
No OA, no knee symptoms	17/869	(2.0)	1.0	
All Radiographic Grades ≥ 2	25/452	(5.5)	2.67	(1.42, 5.04)
Asymptomatic grade 2 OA	8/204	(3.9)	1.89	(0.80, 4.47)
Asymptomatic ≥ grade 3 OA	9/148	(6.1)	2.97	(1.28, 6.87)
Symptomatic ≥ grade 2 OA	8/100	(8.0)	3.84	(1.60, 9.22)
All Knee Symptoms	13/157	(8.3)	3.72	(1.75, 7.93)
No radiographic OA	5/57	(8.8)	4.41	(1.54, 12.64)
≥grade 2 OA	8/100	(8.0)	3.38	(1.39, 8.21)
Walking A Mile				
All subjects	184/1293	(14.2)		
No OA, no pain	96/852	(11.3)	1.0	
All Radiographic Grades ≥ 2	88/441	(20.0)	1.71	(1.23, 2.37)
Asymptomatic grade 2 OA	26/201	(12.9)	1.02	(0.63, 1.65)
Asymptomatic ≥ grade 3 OA	32/139	(23.0)	2.03	(1.27, 3.23)
Symptomatic ≥ grade 2 OA	30/101	(29.7)	2.90	(1.77, 4.75)
All Knee Symptoms	42/156	(26.9)	2.62	(1.71, 4.01)
No radiographic OA	12/55	(21.8)	2.16	(1.08, 4.31)
≥ grade 2 OA	30/101	(29.7)	2.87	(1.75, 4.70)

were slightly elevated in four of these tasks. The largest increased odds for dependence in complex ADL except cooking were found for those elders with knee pain but without radiographic disease.

Discussion

These results indicate the need for a specific definition of disease in the study of arthritis and disability in the elderly. Studying generic "arthritis" may underestimate the impact of osteoarthritis on physical disability in the elderly and obscure the task-specificity of that relationship. Our data demonstrate that dependence associated with knee osteoarthritis varies according to the criteria used to define osteoarthritis and show that knee osteoarthritis does not affect all types of functional activities equally. The magnitude of risk is clearly diminished when all degrees of osteoarthritis are combined, or minor radiographic changes are counted as osteoarthritis. Conversely, the odds are higher when definitions based upon symptom reporting are used. Our findings may help to explain the results of other studies. For example, a previous study of hand function and disease severity by Baron and colleagues[7] found little relationship between osteoarthritis severity measured radiographically and hand function. These investigators included all subjects with radiographic changes ≥ grade 2 in a single "osteoarthritis" category, which may explain the lack of association.

Summated indices of function that include many items unaffected by osteoarthritis may obscure the highly task-specific nature of these limitations, which, in the case of knee osteoarthritis, appear confined primarily to the lower extremity. The task specificity of osteoarthritis-related disability may explain the findings of Summers and coworkers[17] in their study of hip or knee osteoarthritis and functional disability in the elderly. Using 11 subscales from the Sickness Impact Profile (SIP), these investigators noted that only the correlation between disease severity and the SIP ambulation subscale was significant. This finding corroborates our own regarding the task-specific impacts of knee osteoarthritis on elderly function.

TABLE 4—Odds Ratios for Dependence upon Human Assistance to Perform Complex Activities of Daily Living by Grade of Osteoarthritis and Presence of Knee Pain Adjusted for Age and Sex

Activity	Proportion Disabled	(%)	Adjusted OR	(95% CI)
Housekeeping				
All subjects	71/1218	(5.8)		
No OA, no knee symptoms	42/799	(5.3)	1.0	
All Radiographic Grades ≥ 2	29/419	(6.9)	1.12	(0.68, 1.85)
Asymptomatic grade 2 OA	5/196	(2.6)	0.39	(0.15, 1.01)
Asymptomatic ≥ grade 3 OA	14/132	(10.6)	1.80	(1.93, 3.48)
Symptomatic ≥ grade 2 OA	10/91	(11.0)	1.86	(0.88, 3.92)
All Knee Symptoms	15/143	(10.5)	1.83	(0.96, 3.48)
No radiographic OA	5/52	(9.6)	1.90	(0.70, 5.16)
≥ grade 2 OA	10/91	(11.0)	1.80	(0.84, 3.83)
Heavy Home Chores				
All subjects	348/1310	(26.6)		
No OA, no knee symptoms	215/860	(25.0)	1.0	
All Radiographic Grades ≥ 2	133/450	(29.6)	1.10	(0.84, 1.43)
Asymptomatic grade 2 OA	52/205	(25.9)	0.89	(0.62, 1.29)
Asymptomatic ≥ grade 3 OA	46/144	(31.9)	1.24	(0.83, 1.84)
Symptomatic ≥ grade 2 OA	35/101	(34.7)	1.37	(0.87, 2.15)
All Knee Symptoms	51/158	(36.1)	1.51	(1.04, 2.20)
No radiographic OA	22/57	(38.6)	1.85	(1.04, 3.27)
≥ grade 2 OA	35/101	(34.7)	1.35	(0.85, 2.12)
Cooking				
All subjects	34/1211	(2.8)		
No OA, no knee symptoms	23/791	(2.9)	1.0	
All Radiographic Grades ≥ 2	11/420	(2.6)	0.93	(0.44, 1.95)
Asymptomatic grade 2 OA	4/196	(2.0)	0.74	(0.25, 2.18)
Asymptomatic ≥ grade 3 OA	5/131	(3.8)	1.21	(0.45, 3.27)
Symptomatic ≥ grade 2 OA	2/93	(2.2)	0.88	(0.20, 3.86)
All Knee Symptoms	2/142	(1.4)	0.58	(0.13, 2.50)
No radiographic OA	0/49	(0.0)		
≥ grade 2 OA	2/93	(2.2)	0.88	(0.20, 3.84)
Grocery Shopping				
All subjects	54/1209	(4.5)		
No OA, no knee symptoms	34/797	(4.3)	1.0	
All Radiographic Grades ≥ 2	20/412	(4.9)	1.94	(0.53, 1.69)
Asymptomatic grade 2 OA	8/196	(4.1)	0.79	(0.36, 1.77)
Asymptomatic ≥ grade 3 OA	7/126	(5.6)	1.06	(0.45, 2.51)
Symptomatic ≥ grade 2 OA	5/90	(5.6)	1.10	(0.41, 2.93)
All Knee Symptoms	8/139	(5.8)	1.18	(0.52, 2.67)
No radiographic OA	3/49	(6.1)	1.47	(0.43, 5.09)
≥ grade 2 OA	5/90	(5.6)	1.05	(0.39, 2.85)
Carrying Bundles				
All subjects	46/1280	(3.6)		
No OA, no knee symptoms	24/838	(2.9)	1.0	
All Radiographic Grades ≥ 2	22/442	(5.0)	1.31	(0.71, 2.42)
Asymptomatic grade 2 OA	5/205	(2.4)	0.63	(0.23, 1.71)
Asymptomatic ≥ grade 3 OA	12/139	(8.6)	2.47	(1.15, 5.31)
Symptomatic ≥ grade 2 OA	5/98	(5.1)	1.26	(0.46, 3.48)
All Knee Symptoms	9/150	(6.0)	1.62	(0.72, 3.68)
No radiographic OA	4/52	(7.7)	2.88	(0.92, 9.07)
≥ grade 2 OA	5/98	(5.1)	1.18	(0.42, 3.29)

Our previous study of the prevalence of osteoarthritis indicated that the proportions of elders with symptomatic knee osteoarthritis and those with ≥ grade 3 changes increase with age.[10] The present study demonstrates that the dependence in functional activities associated with these particular specifications of knee osteoarthritis varies according to the severity of osteoarthritis and the presence of symptoms. Thus the age composition of the sample is a crucial component of the study of osteoarthritis and disability. Studies of predominately younger elders may not have a sufficient number of cases severe enough to discern the impact of knee osteoarthritis on function.

Aggregated indices of function may be too imprecise with respect to knee osteoarthritis to identify the particular

nature of the functional deficits even when the sample has a sufficient number of severe cases. This study underscores the critical importance of choosing functional measures that can identify disability and also be meaningful in terms of the range of tasks and activities typically performed by elders. Using items that measure complex ADL only, e.g. cooking, we would have been unable to capture much of the relationship of knee osteoarthritis to physical disability. The difficulty of choosing discriminating functional measures is further complicated when the research interest focuses on physical function across diseases. "Walking a mile" serves as a case in point. The results of this study indicate that this task successfully identifies osteoarthritis-related disability, at least among the relatively more severe cases. It is also intuitively applicable to other important diseases of the elderly, e.g. cardiovascular diseases, but one must question how meaningful as an essential basic task walking a mile is within the context of an elder's daily activity.

The association of knee osteoarthritis and knee pain with lower extremity disability is not surprising. It is not clear, however, why severe knee osteoarthritis (≥ grade 3) should also be associated with limitations in housekeeping and carrying bundles, and not with other complex ADL. Biomechanical analyses of joint function and studies of muscle performance during these tasks may uncover why this particular degree of joint changes affects these ADL, but not others. Possibly these tasks also reflect choices made by elders. Functional activities are also sociological phenomena, not merely indicators of physical capacities for activity or disease outcomes. It is usually not a cultural expectation or a personal necessity to do heavy chores such as shoveling snow. On the other hand, elders may have little choice with regards to preparing their own meals. Light housekeeping and carrying bundles may be two tasks which elders can elect to perform, and therefore are sensitive to mild disability in elderly populations.

Our study indicated that a substantial number of elders without frequent symptoms had increased odds of disability. This association may be explained, in part, by the wording of our symptom question which required pain to persist for most days of a month. Pain in osteoarthritis is typically elicited by movement of the joint. Therefore, those elders with asymptomatic osteoarthritis ≥ grade 3 may be asymptomatic precisely because they avoid the tasks that would exacerbate the symptoms. If this is the case, then the impact of symptomatic radiographic disease on complex ADL may easily be underestimated given the transient activity-induced nature of pain in osteoarthritis. Questioning subjects on the presence of pain when performing each task might be more productive in demonstrating a relationship among osteoarthritis, pain, and functional disability.

Our data indicate that using knee pain as a proxy measure of arthritis, as might be done in studies using self-reported arthritis, has strengths and weaknesses. Defining osteoarthritis on the basis of symptoms alone did manage to capture many individuals with radiographic evidence of disease. The 57 elders reporting knee pain without radiographic disease may have had other related knee problems (e.g. anserine bursitis, patelofemoral disease, or referred pain from the hip), which are relevant to the study of musculoskeletal impairment and physical disability. Constructing the definition of arthritis solely on the grounds of knee pain, however, excludes a substantial number of subjects with radiographic disease but infrequent symptoms who also had physical disability. Investigators who do not use radiographs to corroborate self-reported arthritis should evaluate whether this degree of imprecision in classifying patients is consistent with their research goal.

In any case, the presence of symptoms should not be considered a necessary condition for exploring arthritis-related disability, a comment previously made with respect to the format of the National Health and Nutrition Examination Survey I.[18] Similarly, pain and functional limitations also should be explored separately in a clinical encounter.

Comorbidities, which are potential confounders of the association of knee osteoarthritis and physical disability, were not included in the model used to estimate odds ratios in this study. Our previous work has indicated a strong association between marked obesity and knee osteoarthritis.[19] The association of knee osteoarthritis with other conditions such as angina and diabetes, which are themselves associated with physical disability, is unclear and we are currently investigating this issue. Although the magnitude of the association between knee OA and physical disability may change in a more complete model, we do not anticipate that the distinctions found when using different definitions of osteoarthritis will be eradicated.

The disability prevalence estimates of our study are limited to those Framingham elders who were able to appear at the study site for a knee radiograph. Therefore, these estimates do not reflect the disability of elders who were home-bound or institutionalized and more likely to be functionally limited.

Although the present study confined itself to knee osteoarthritis, the methodology of this study is applicable to the study of other diseases. Defining diseases broadly, e.g. "stroke," "pulmonary disorders," or "cancer," allows only for a crude approximation of the relationship between disease and disability in the elderly. Symptomatology in other important medical conditions is also activity-induced. Elders with angina, emphysema or intermittent claudication may have learned over time which activities exacerbate their symptoms. These individuals may readily report that they have not recently experienced symptoms, yet have severely curtailed their level of functional activities. Analyses that contribute to our understanding of physical disability in elders by differentiating subgroups of disease and identifying their associations with particular functional limitations are an essential step in designing effective clinical interventions to remediate these deficits.

In summary, combining all severities of osteoarthritis into "arthritis," on using symptom-based definitions of disease alone, and aggregating all functional activities into a single measure of function obscures two facts: 1) that the degree of association between osteoarthritis and physical disability and the elderly depends on the definition of osteoarthritis; and 2) that the impact of any definition of osteoarthritis on functional status is highly task-specific. Future studies of the relationship of arthritis and physical disability in elders should use specific definitions of osteoarthritis and report the impact of specific severity levels of disease on particular functional activities. Such precision should also benefit our understanding of the relationship of other diseases and physical disability as well.

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Two New Publications Available from MCH Clearinghouse

Two new publications which address the health of mothers and babies have been announced by the Health Resources and Services Administration of the federal government.

- *Nutritional Management of the Pregnant Adolescent: A Practical Reference Guide* is a 182-page manual on how to improve the nutrition of prospective young mothers and thus promote the birth of healthy babies. The manual was published through the joint efforts of HRSA's Maternal and Child Health Bureau, the US Department of Agriculture, and the March of Dimes Birth Defects Foundation.
- *Recommendations from the National Symposium on Genetic Services for Underserved Populations* is a new 41-page booklet based on the recommendations drafted at a symposium in May 1989, urging improved genetic services for the underserved. The symposium was sponsored by the Mid-Atlantic Regional Human Genetics Network and the March of Dimes Birth Defects Foundation; publication of the document was supported by the Health Resources and Services Administration.

Single copies of these publications are available from the National Maternal and Child Health Clearinghouse, 38th and R Streets, NW, Washington, DC 20057. Tel: (202) 625-8410.

The Present and the Future of AIDS and Tuberculosis in Illinois

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Abstract: The relation between the acquired immune deficiency syndrome (AIDS) and tuberculosis (TB) was examined by matching the Illinois AIDS and TB registries. The match group was examined and compared with patients with only one disease by race, method of human immunodeficiency virus (HIV) transmission, site of tuberculous disease, radiographic findings, and results of Mantoux tests. The time of TB diagnosis was centrally distributed around the time of AIDS diagnosis; from this, it was determined that 4.1 percent of AIDS patients develop active TB. Projections for future AIDS cases were made by fitting a polynomial model to historical data. These projections were then used to predict the future impact of AIDS-related TB upon state TB rates. The rise in TB rates call for special efforts to minimize this impact. (*Am J Public Health* 1990; 80:950–953.)

". . . many of those who had been long gradually declining took to bed with symptoms of phthisis; in many cases of a formerly doubtful character the disease then became confirmed; in these the constitution inclined to the phthisical. Many, and in fact the most of them, died. . ."

—Hippocrates[1]

Introduction

Since the time of Hippocrates, a special susceptibility to clinical tuberculosis (TB) by those already infirm has been noted again and again. Patients with pre-existing malignancy, silicosis, or renal failure, and those undergoing corticosteroid therapy are all at increased risk for developing TB.[2] Two millennia of clinical observation and recent advances in cellular immunobiology[3] support the same generalized conclusion: insult to the salubrity of the human organism predisposes to TB. Reports that patients with the acquired immune deficiency syndrome (AIDS) are at increased risk for TB are consistent with this principle.

Since 1983, data regarding the AIDS/TB connection have come from hospital patient lists or population-based disease registries. In several studies (see Appendix) it has been possible to determine the proportion of persons with AIDS who develop TB[4–12]; the variation in this figure may reflect variation in the prevalence of tuberculosis infection among human immunodeficiency virus (HIV) transmission categories. In the populations studied to date, AIDS patients who are intravenous drug users or Haitian appear to be at higher risk for TB than other AIDS patients. The experience of central regions of the United States is important to an evaluation of the likelihood of a national resurgence of TB.

This paper reports the co-occurrence of AIDS and TB in Illinois, projections of future AIDS and AIDS/TB cases, and a strategy for control of AIDS-related tuberculosis.

Methods

Using a hybrid method of computerized and manual review, in September 1988 the electronic TB Registry for the State of Illinois was matched to the Illinois AIDS Registry. All TB and AIDS cases met the definitions established by the Centers for Disease Control and in effect at the time the diagnosis was made.[13,14] The electronic TB Registry contained 3,033 cases of TB diagnosed in Illinois from January 1985 to July 1988. By September of 1988, the records of all persons reported to the State in the calendar years 1986 and 1987 and some persons reported in calendar years 1985 and 1988 had been entered into the TB Registry and the AIDS Registry contained all 1,992 AIDS patients reported from 1980 through August 1988. An independent evaluation[15] of the Chicago portion of the AIDS Registry indicated that completeness of reporting was 95 percent.

Three fields were used to match TB Registry cases with AIDS Registry cases: last name, first name, and date of birth. Last names and dates of birth were considered critical fields. To be considered a match, one pair of critical fields had to match exactly and the other pair could vary by no more than a single character or a transposition. Additionally, during manual review first names had to be meaningful equivalents. Exact matches on dates of birth and last name were identified using a personal computer; satisfaction of the match definition was assured by manual review of the computer output.

The projected number of Illinois AIDS cases for 1988 through 1993 was obtained by fitting a third order polynomial ($y = \beta_0 + \beta_1 \times + \beta_2 \times^2 + \beta_3 \times^3$) to the case count from 1981 through 1987 and determining the 95 percent confidence intervals around the model.[17,18] "Y" represents the number of cases, "X" the year of projection, and each "β" is a constant coefficient. Goodness-of-fit was measured by the R squared statistic.

Comparisons of patient characteristics predisposing to AIDS were done using Yates-corrected chi-square analysis.

Results

A comparison of all AIDS and TB cases yielded 46 persons with both diseases. A profile of the characteristics of AIDS patients with and without TB is displayed in Table 1. Mantoux test results were available for 20 of these 46 patients; 13 (65 percent) were positive. AIDS patients who were Black were at increased risk of developing tuberculosis (RR = 2.65, 95% CI = 1.50, 4.69) as were persons who had intravenous drug abuse as a risk factor (RR = 1.72, 95% CI = 1.06, 3.49). After stratification, the effects of Black race and HIV were independently significant. Residence and sex were not significantly associated with the development of TB in AIDS patients.

Thirty-nine of the 46 persons (85 percent) with both diseases had pulmonary TB. Of these 39, chest x-ray results were available for 26 patients (66 percent); 18 (69 percent) of

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TABLE 1—Comparison of AIDS Patients with and without Tuberculosis

Variables	% Tuberculosis Yes N = 46	No N = 1,946	Relative Risk	95% CI
Risk Factors				
Homosexual/bisexual male	59	75	.49	.28, .88
Gay and IV drug use	9	5	1.80	.66, 4.93
IV drug use only	17	9	2.02	.96, 4.27
All IV drug users	26	14	1.72	1.06, 3.49
Other	15	11	1.40	.63, 3.10
Ethnic Background				
White	33	61	.35	.19, .65
Black	52	29	2.65	1.50, 4.69
Other	15	10	1.24	.53, 2.89
Sex				
Male	96	95	1.08	.27, 4.38
Female	4	5	.93	.23, 3.77
Residence				
Chicago	65	74	.66	.36, 1.20
Cook Co. excluding Chicago	15	9	1.69	.77, 3.73
Other Illinois	20	16	1.18	.58, 2.41

these showed non-cavitary lesions, seven (27 percent) showed cavitary lesions, and one was within normal limits in a patient with positive cultures for *M. tuberculosis*. Seven persons (15 percent) with both diseases had only extra-pulmonary tuberculous disease, and 21 (46 percent) had only pulmonary tuberculosis. Thus 25 persons or 54 percent of cases with both TB and AIDS had extra-pulmonary involvement compared to 16 percent in TB patients nationwide.[16] Disseminated disease was miliary in six persons, involved cervical lymph nodes in four; involvement of gastrointestinal, genitourinary, other lymph nodes, and hematogenous sites occurred in two persons each, and tuberculous involvement of liver, brain, meninges and bone marrow each occurred in one case.

The distribution of intervals between time of TB diagnosis and time of AIDS diagnosis is symmetrical around the time of AIDS diagnosis. This distribution has a mean of .87 months and a standard deviation of 7.27 months. TB was diagnosed within six months of AIDS diagnosis in approximately 70 percent of persons with both diseases. This distribution is consistent with the findings of others[5,7] and forms the basis for the analysis below.

Because the Illinois TB Registry is complete for two consecutive years only, an analysis of limited time periods was performed to determine the fraction of TB patients who also contract AIDS and the fraction of AIDS patients who develop TB. TB Registry cases with TB diagnoses in calendar years 1986 or 1987 were compared to AIDS Registry cases with AIDS diagnoses from July 1, 1986 to June 30, 1987. After manual review of the 2,235 TB patients reported in 1986 or 1987 (years for which the electronic TB Registry held all TB reports), 104 incomplete or duplicate records were eliminated and 2,131 records remained. Of these 2,131 TB patients, 35 (1.6 percent) were also reported as having AIDS at some time from 1980 to 1987.

Persons diagnosed with AIDS from July 1, 1986 to June 30, 1987 (n = 603) could develop TB before or after calendar years 1986 and 1987. Therefore, the fraction of AIDS patients who contracted TB could not be calculated without including some adjustment for such TB cases. Comparison of the AIDS and TB intervals yielded 21 matches. Assuming that the distribution of onset intervals were normal with a mean and standard deviation of the observed distribution, the number of AIDS patients expected to develop TB before or after the two-year interval was calculated, yielding an estimate of four additional TB cases. Thus we estimate that 25 TB/AIDS cases occurred, of which four or 16 percent were undetected and that 4.1 percent of the 603 AIDS patients developed TB (95% CI = 0.0276, 0.0614).[17]

Projections of AIDS and TB/AIDS

Illinois AIDS case data were fitted to the above polynomial with the following coefficients: $\beta_0 = -41.2857$, $\beta_1 = 61.66666$, $\beta_2 = -23.25$, $\beta_3 = 4.083333$. The R squared statistic was .999305. The goodness-of-fit, projected AIDS cases and the 95% CI of the projections[18] are shown graphically in Figure 1.

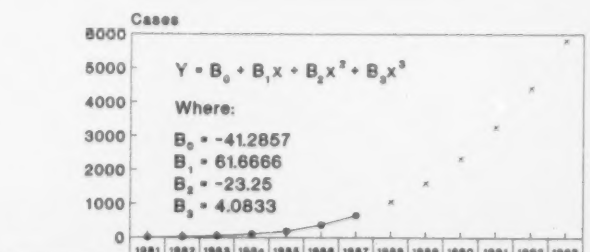
Based on these projections, the centrally distributed AIDS/TB interval, and the proportion of AIDS cases who develop TB (4.1 percent), the number of AIDS patients with TB for each year from 1985 to 1993 was calculated (Table 2, column 4). Subtracting these cases from all TB cases reported and dividing by population figures yielded rates of non-AIDS associated TB (Table 2, column 5). Assuming an unchanged intensity in tuberculosis control measures and a constant 1988 level of non-AIDS associated TB, estimates of future rates for tuberculosis in Illinois can be projected (Figure 2).

Discussion

The reported diagnoses of tuberculosis in AIDS cases characteristically occur near the time of AIDS diagnoses, although this may reflect more careful examination rather than the co-occurrence of disease. The pattern of tuberculous organ involvement is bizarre in AIDS/TB, and radiologic findings are frequently atypical. While the Mantoux test result is often a false negative in AIDS/TB patients, the test remains a useful diagnostic tool.

The recent resurgence of TB in New York[7] and Florida[19] and the sudden arrest of the downward trend in US TB rates[20] have been clearly attributed to TB among AIDS cases. Without intensification of TB control measures, a similar rise in TB incidence can be expected in America's heartland.

The computed projections of TB in Illinois have several limiting deficiencies which need to be addressed. Determination of the proportion of AIDS patients who develop TB was based on a match of AIDS Registry cases who met the early definition of AIDS; the proportion of AIDS patients



* Source: Kmenta, Elements of Econometrics, MacMillan Publishing Co., Inc. New York. 1971. pp. 227-229

FIGURE 1—Comparison of AIDS Cases Reported and Predicted by the Third Order Polynomial

TABLE 2—Historical and Projected Rates of Tuberculosis in Illinois: Unassociated with AIDS and Total Rates

Year	Il. Popul × 1,000	TB Cases Reported	TB/AIDS Cases Computed	TB-only Rate	TB only +TB/AIDS Rate
1982	11,481	1653		14.4	14.4
1983	11,494	1380		12.0	12.0
1984	11,526	1207		10.5	10.5
1985	11,540	1193	8	10.3	10.3
1986	11,555	1144	16	9.8	9.9
1987	11,586	1091	26	9.2	9.4
1988	11,595	1078	44	8.9	9.3
1989	11,603*		67*	8.9*	9.5*
1990	11,612*		97*	8.9*	9.7*
1991	11,633*		135*	8.9*	10.1*
1992	11,654*		182*	8.9*	10.5*
1993	11,675*		239*	8.9*	10.9*

*Projected figures

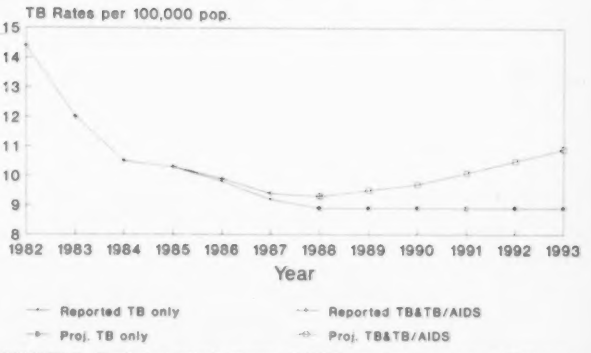


FIGURE 2—Projected Illinois Rates of AIDS-associated and non-AIDS-associated TB

who meet the broader 1987 definition may be different. The projection of future AIDS cases was based on empiric data and may be inaccurate. The assumption of an unchanging rate of tuberculosis unassociated with AIDS assumes no change in the characteristics of the population. The projections do not consider the effect of secondary transmission to uninfected individuals with or without AIDS by AIDS cases with TB. Finally, the projections only extend five years but the most serious public health impact of AIDS-associated TB may occur over the course of decades.

The Reports of the Advisory Committee for Elimination of Tuberculosis[21,22] provides an excellent blueprint for a strategy to minimize the impact of AIDS on future rates of TB. While of undocumented efficacy, early identification and prophylactic treatment of HIV-infected persons who are also infected with *M. tuberculosis* is a reasonable component of such a plan. Specifically, all intravenous drug users should be screened for both HIV and tuberculous infection. Regardless of age, isoniazid preventive therapy should be offered to all TB-infected persons who are intravenous drug users or HIV-infected. Greater utilization of the time-tested public health measures of contact tracing and targeted case finding is indicated at this critical juncture as the epidemiology of TB changes. Prompt recognition of this new group of infectious individuals will only be achieved by a heightened awareness by clinicians of the variegated presentations of TB in HIV-infected persons. Undoubtedly, these measures require a political conviction to confront the resurgence of a nearly vanquished disease and an allocation of resources commensurate with such conviction.

APPENDIX
Literature Review: AIDS and Tuberculosis

Author	Location	AIDS Population N =	R/H* Study	% IVDU§	% Haitian	%TB in AIDS Population
Louie[4]	New York City	280	H	32		8.6
Hadler[5]	Connecticut	299	R			6.0
Handwerger[6]	New York City	386	H	33		7.8
Stoneburner[7]	New York City	4,892	R	29	3	5.3
Sunderam[8]	New Jersey	136	H	75	6	21.3
Murray[9]	Multi-regional	1,067	H	70		1.4
Maayan[10]	Westchester County, NY	40	H	70	2	10.0
Pitchenik[11]	Florida	82	R		54	34.1
CDC[12]	24 States & 4 Loc.	15,181	R			4.2

*Registry or Hospital-based study
§Intravenous Drug Users

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Agreements Signed to Establish European Center on Environment/Health

The WHO Regional Director for Europe, Dr. J. E. Asvall, signed agreements in mid-June with officials in Italy and the Netherlands to establish a European Center on Environment and Health, with units in Rome, Italy, and Bilthoven, Netherlands. Together the two units will form a single WHO Center which will become an integral part of the WHO Regional Office for Europe. By early 1991, each unit will have interdisciplinary teams of scientists and support staff and will be fully equipped to greatly expand the existing European program of WHO in the field of environment and health.

The proposal to establish a Center was made last year by the Frankfurt Conference when Ministers from 29 countries approved a European Charter on Environment and Health. One of the key recommendations was to carry out a feasibility study for such a Center and this was accomplished early in 1990. An agreement was reached to establish a single Center with units adjacent to the Institute for Public Health and Environmental Protection (RIVM) in Bilthoven, Netherlands and adjacent to the Institute of Public Health (ISS) in Rome, Italy. Both these institutions have an international reputation in the field of enviromental health and both will provide strong support to the Center.

A high priority for the Center will be to study the interrelationship between environmental effects and human health. The scientific work of the Center will cover a wide range of activities including health aspects of air and water pollution control, chemical and food safety, radiation protection and occupational health.

The Center will have an initial operating budget of about US $3 million per year and other European countries will be encouraged to actively participate in its work. Facilities are being provided both in Bilthoven and Rome for development of both long-term and short-term staff and for the holding of seminars and other meetings. Equipment will include advanced data processing facilities and there will be complete operational linkages with the WHO Regional Office in Copenhagen and with a wide range of scientific institutions and governmental centers.

The Relationship of Smoking Cessation to Coronary Heart Disease and Lung Cancer in the Multiple Risk Factor Intervention Trial (MRFIT)

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Abstract: The impact of smoking cessation on coronary heart disease (CHD) and lung cancer was assessed after 10.5 years of follow-up in the 12,866 men in the Multiple Risk Factor Intervention Trial (MRFIT). Those men who died of lung cancer (n = 119) were either cigarette smokers at entry or ex-smokers; no lung cancer deaths occurred among the 1,859 men who reported never smoking cigarettes. The risk of lung cancer for smokers, adjusted for selected baseline variables using a Cox proportional hazards model, increased as the number of cigarettes smoked increased (B = 0.0203, SE = 0.0076). There was not the same graded response for CHD among smokers at entry. The risk of CHD death was greater among smokers than nonsmokers (RR = 1.57) (B = −0.0034, S.E. = 0.0048). After one year of cessation, the relative risk of dying of CHD for the quitters as compared to non-quitters (RR = 0.63) was significantly lower even after adjusting for baseline differences and changes in other risk factors. The relative risk for smokers who quit for at least the first three years of the trial was even lower compared to non-quitters (RR = 0.38). However, the relative risk for lung cancer for quitters versus non-quitters was close to 1 both for quitters at 12 months and at three years. These data support the benefits of cessation in relation to CHD and are consistent with other epidemiologic studies which suggest that the lag time for a beneficial effect of smoking cessation on lung cancer may be as long as 20 years. (*Am J Public Health* 1990; 80:954–958.)

Introduction

In the last two decades there have been continuing decreases in coronary heart disease (CHD) mortality rates in all United States populations[1–3] while lung cancer mortality rates continue to increase in certain populations.[4,5] Epidemiologic studies have consistently demonstrated a significantly higher relative risk for smokers compared to former smokers for each of these diseases.[1,2,6–8]

The beneficial effect of smoking cessation on CHD is rapid; as the period of cessation increases, the ex-smoker's relative risk approaches that of the never smoker.[3] However, in one study this return occurred gradually over a 20-year period.[8] The relative risk of developing lung cancer has been demonstrated to decrease as the period of cessation increases for smokers who have stopped smoking,[6–8] but their relative risk is still significantly elevated after 10 to 20 years of cessation compared to lifetime never smokers and is related to both the duration of smoking and length of cessation.[6–8]

In the available studies, the effect of smoking cessation on the risk of developing CHD and lung cancer was estimated either using data gathered retrospectively[6–8] or by considering that smoking habits were unchanged over the period of follow-up.[6,7] It is likely that during the course of these studies some ex-smokers became current smokers and vice versa. In addition, use of retrospective data entails problems of recall, and reports of cessation during the course of follow-up of these studies were not validated with biochemical measures. As a result, the mortality ratios reported for ex-smokers may be too high, and the benefits for cessation may actually be greater than those previously demonstrated for lung cancer and for CHD.

Using the detailed smoking behavior data gathered prospectively in the Multiple Risk Factor Intervention Trial (MRFIT), biochemically validated with serum thiocyanate measurements, we evaluated the relationship of smoking cessation to the development of CHD and lung cancer among middle-aged men, after 10.5 years of follow-up.

Methods

The MRFIT[9–21] was sponsored by the National Heart, Lung and Blood Institute (NHLBI) as a clinical trial, to test the effect of a multifactor intervention program on mortality from CHD. Other objectives included examination of decreases in other mortality and morbidity end points including lung cancer. In summary, the participants were 12,866 men ages 35–57 years at increased risk for CHD and free of overt CHD on entry. Men were designated at increased risk if their levels of the three major CHD risk factors—serum cholesterol, cigarette smoking, and blood pressure—were high enough at first screening to place them in the top 15 percent (later changed to 10 percent for 72 percent of the men screened) of a risk score distribution based on data from the Framingham Heart Study. Men with diastolic blood pressure of 115 mmHg or higher and those with total cholesterol of 350 mg/dl or higher were excluded.

Of the 12,866 men randomized to the special intervention (SI) and usual care (UC) groups, 64 percent (n = 8194) reported smoking cigarettes at the initial screening examinations, an average of 33.9 cigarettes/day. Randomization occurred from December 1973 to February 28, 1976. Thus, on February 28, 1982, when active intervention was terminated, each surviving man had been in the trial for at least six years, with a mean duration of 6.9 years of follow-up from randomization.

All SI men were encouraged and helped to make dietary changes directed at lowering their blood cholesterol levels. The smoking intervention program used an initial intensive group approach as well as individual counseling sessions when indicated. Both approaches included the use of behavioral and cognitive interventions. SI men who were hypertensive (mean diastolic blood pressure greater than or equal to 90 mm Hg on two consecutive visits, or on antihypertensive medication from their personal physicians) were treated with a stepped care approach.[13,18] UC men were not offered

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intervention at the clinical centers and were referred back to their usual source of medical care. They were asked to return to the MRFIT clinical center once a year for a comprehensive evaluation, including assessment of risk factors and morbidity status.

Baseline and annual data collection for both groups during the trial included a medical history and examination, information on past smoking patterns, and reported present cigarettes per day. Serum thiocyanate (Scn) was also measured at baseline and annually to validate reported smoking status. In addition to cigarette smoking, Scn may be elevated by the use of pipes, cigars and cigarillos, certain foods and diuretics.[22] Subjects with an Scn level of more than 100 μ mols/L were assessed to be smoking.

Mortality Ascertainment

During the trial and at termination of active intervention on February 28, 1982, deaths were ascertained by clinic staff through contact with family or friends of the deceased, routine follow-up of missed clinic visits, response to postcards requesting change-of-address information sent twice yearly to UC participants, and searches of publicly accessible files of deceased persons. The main source of vital status information after completion of participation in February 1982 has been the Social Security Administration and the National Death Index. Mortality ascertainment through December 31, 1985 was estimated to be approximately 100 percent complete using these data sources.[18]

The cause of death used in this report is based on death certificate reports. Cause-specific death rates are based on coding of death certificates by trained nosologists, according to the Ninth Revision of the International Classification of Diseases (ICD-9).[23] Each death certificate was independently coded by two nosologists and disagreements were adjudicated by a third nosologist. ICD-9 codes 410 to 414 and 429.2 were used to identify CHD deaths; ICD-9 code 162 was used to identify lung cancer deaths.

Statistical Methods

The SI-UC differences for the end points CHD and lung cancer mortality were tested for statistical significance using the log-rank test.[24] As in other MRFIT publications, 90 percent confidence intervals for the percentage difference in death rates are presented. Mortality rates are presented as number of cases per 1,000 person-years.

The relationship between smoking-related variables and end points was assessed for the combined MRFIT SI and UC smokers at entry into the study using the cox proportional hazards model.[25,26] In one analysis, smokers at entry who attended the first annual examination were classified as *quitters* if they reported not smoking at the first annual examination and had serum Scn levels that were less than 100 μ mols/L, or as *smokers*. In other analyses, smokers at entry who attended the first three annual examinations were classified as persistent quitters, if at each of the three annual examinations they reported not smoking and had serum Scn levels which were less than 100 μ mols/L at each visit, or as *other smokers/quitters*.

Relative risk (RR) estimates and 95 percent confidence intervals for quitters compared to non-quitters were calculated using the proportional hazards regression model. RRs are presented both unadjusted and adjusted for age, baseline blood pressure, cholesterol, number of cigarettes smoked per day, and years of education (as an index of socioeconomic status) added to the model as continuous variables. Differences in baseline characteristics and changes in risk factor levels from baseline to the third annual examination for persistent quitters compared to other smokers/quitters were tested for statistical significance using Student's t test.

Results

As of December 31, 1985, after an average period of follow-up of 10.5 years, there were 1,033 deaths in the MRFIT. There were 146 CHD deaths (3.42/1,000 person-years) among the SI smokers at entry and 165 (3.89/1,000 person-years) among the UC smokers at entry, a relative difference of −12.1 percent (90% CI = −27.1, 5.9). The CHD mortality rates were 2.26/1,000 person-years and 2.44/1,000 person-years for SI and UC non-smokers at entry, respectively. There were 56 lung cancer deaths (1.31/1,000 person-years) among the SI smokers at entry and 50 (1.18/1,000 person-years) among the UC smokers at entry, a relative difference of +11.3 percent (90% CI = −19.2, 53.3). The nine SI and four UC non-smokers at entry who died of lung cancer had been smokers at some point prior to entry into the study.

Mortality in Subgroups Defined after Randomization

Although smoking intervention in the MRFIT resulted in a significantly higher cessation rate for SI compared to UC men ($p < .001$), the lung cancer and CHD mortality rates were not significantly different for these two groups. (Smoking cessation results in the MRFIT will be presented in detail in a subsequent section.) The within group analyses presented in Tables 1–5 are observational, combining data for SI and UC participants.

No lung cancer deaths occurred among the 1,859 MRFIT men who had never smoked cigarettes. Among cigarette smokers there appeared to be a graded relationship between the number of cigarettes smoked per day at baseline, and the lung cancer mortality rate. However, number of cigarettes smoked per day at baseline and risk of CHD mortality did not appear to have as strong a relationship as for lung cancer. Ex-smokers appeared to have a risk of dying from CHD that approached that of never smokers (Table 1).

In the MRFIT study group, never smokers had higher cholesterol and/or blood pressure levels than smokers at baseline in order to be in the top 10 percent of CHD risk. Regression analyses to account for confounders shown in Tables 2 and 3 indicate that only age, the screen 1 cigarettes/day, and baseline Scn are of importance for lung cancer; for CHD mortality age, baseline Scn, DBP, and cholesterol were all significant risk factors ($p < 0.01$). Removing Scn from the regression analysis did not result in a significant relationship between number of cigarettes smoked and CHD risk among smokers.

TABLE 1—CHD and Lung Cancer Deaths and Mortality Rates per 1,000 Person-Years by Number of Cigarettes Smoked per Day at Baseline for MRFIT SI and UC Men Combined

		CHD Deaths		Lung Cancer Deaths	
	Total	N	Rate/1000 Person-Years	N	Rate/1000 Person-Years
Never-smokers	1859	44	2.22	0	0
Ex-smokers	2813	73	2.44	13	0.43
Smokers*					
1–19	856	23	2.56	2	0.22
20–39	3747	173	4.45	50	1.29
≥40	3591	115	3.08	54	1.45

*Number of cigarettes/day smoked

TABLE 2—Regression Coefficients from Cox Proportional Hazards Model for the End Point Lung Cancer Death for MRFIT SI and UC Men Who Reported Smoking Cigarettes at Entry

Variables	Coefficient	SE
Age (years)	0.1239	0.0198
Screen 1 cigarettes/day	0.0203	0.0076
Serum Thiocyanate (μmoles/l)	0.0041	0.0019
Age began smoking (years)	−0.0147	0.0270
Filter/non-filter (1 = filter, 0 = non-filter)	−0.6440	0.4076
Tar	−0.1334	0.0952
Nicotine	1.9110	1.3438
Alcoholic drinks/week	−0.0120	0.0090
Screen 1 DBP (mmHg)	0.0153	0.0155
Screen 1 cholesterol (mg/dl)	−0.0025	0.0030

TABLE 3—Regression Coefficients from the Cox Proportional Hazards Model for the End Point CHD Death for MRFIT SI and UC Men who Smoked Cigarettes at Entry

Variables	Coefficient	SE
Age (years)	0.0626	0.0104
Screen 1 cigarettes/day	−0.0034	0.0048
Serum Thiocyanate (μmoles/l)	0.0029	0.0011
Age began smoking	−0.0127	0.0154
Filter/non-filter (1 = filter, 0 = non-filter)	−0.3337	0.2447
Tar	0.0489	0.0567
Nicotine	−0.9942	0.7927
Alcoholic drinks/week	−0.0150	0.0055
Screen 1 DBP (mmHg)	0.0317	0.0091
Screen 1 cholesterol (mg/dl)	0.0047	0.0017

CHD and lung cancer mortality rates for all men who quit smoking during the first year were compared with the rates of those who continued to smoke (Table 4). The total group of smokers who reported quitting in year one and had Scn < 100 μ mols/L had a relative risk of dying from CHD .63 that of non-quitters. The relative risk estimate adjusted for entry risk variables were similar to unadjusted estimates (Table 4). The quitters had the same lung cancer mortality rate from year one to the end of the trial as those who were non-quitters at year one (Table 4). (This was true regardless of baseline levels of smoking or whether they were in the UC or SI group.)

TABLE 4—Number of CHD and Lung Cancer Deaths and Mortality Rate (per 1,000 person-years) from the 12-month visit through 12/31/85[a] for Baseline Smokers by Smoking Status at 12 months, and Relative Risk Estimates (Quitters/Not Quitters) for MRFIT SI and UC Men

Smoking Status	# of Participants SI + UC	Deaths SI + UC	Relative Risk Quit/Not Quit (95% CI)	Adjusted Relative Risk[d] (95% CI)
		Coronary Heart Disease		
Quit[b]	1365	33 (2.54)[c]	.63 (.44,.90)	.58 (.40,.84)
Not Quit	6298	239 (4.04)		
		Lung Cancer		
Quit[b]	1365	17 (1.31)[c]	0.99 (.59,1.67)	1.05 (.62,1.79)
Not Quit	6298	78 (1.32)		

[a] This represents an average of 9.5 years of followup
[b] Quitters are S_1 smokers who reported no smoking at year 1 with SCN < 100 μ moles/L.
[c] Rates per 1000 person-years.
[d] Adjusted for age, baseline cigarettes/day, cholesterol, blood pressure, education

We also looked at persons who had quit smoking for at least the first three years of the trial (persistent quitters). Compared to the continuing smokers, the persistent quitters experienced a reduced risk of CHD death (RR = .38) but no change in risk of dying of lung cancer (Table 5). These comparisons are limited by the small number of quitters who persisted for three years which was less than the number who persisted for one year. (Participants who died in the first three years of the trial are also not included.) Persistent quitters were also lighter smokers than those who did not persist (average 30.1 vs 34.2 cigarettes per day).

Persistent quitters and continuing smokers had differences in baseline risk factors and in changes in other risk factors through the three years the two groups were compared. On average, persistent quitters smoked four fewer cigarettes per day and drank 2.2 fewer alcoholic drinks per week at baseline than did other smokers or short-term quitters. Persistent quitters experienced significantly greater weight gain by three years than other smokers or quitters. Adjusting for the differences in risk factors at baseline did not change the relative risk estimates (0.38 versus 0.35), nor did adjusting for the differences in risk factor changes (RR = 0.36, 95% CI = 0.20, 0.65). Likewise, none of these models produced changes in relative risk estimates for lung cancer.

The distribution of lung cancer deaths throughout the span of the trial does not indicate any difference in early versus late mortality by randomization status. Therefore, there is no evidence to date for a trend toward fewer SI as compared to UC lung cancer deaths as time from entry becomes longer, or closer to the 10.5 year maximum follow-up. Because of the relatively small number of participants who were long-term quitters of three or more years and died of lung cancer, it is not possible to evaluate whether the lung cancer death rates might be declining in the latter years of the trial among persistent quitters.

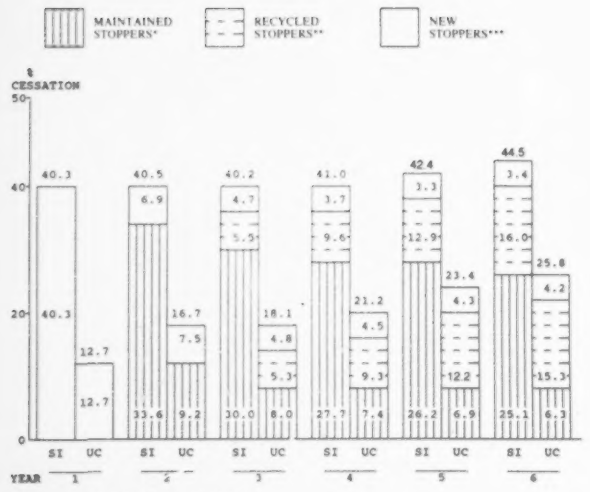
Cessation and Relapse Patterns

Although CHD and lung cancer mortality rates were not significantly different between the randomized groups, smoking cessation rates were significantly different for SI compared to UC men (Figure 1). The cross-sectional data indicate that, throughout the six years of follow-up in the trial, cessation rates for SI smokers were approximately 17 percentage points greater than they were for UC smokers.

TABLE 5—Number of CHD and Lung Cancer Deaths and Mortality Rate (per 1,000 person-years) from the 36-month visit through 12/31/85[a] for Baseline Smokers by Smoking Status at 36 months, and Relative Risk Estimates (Quitters/Not Quitters) for MRFIT SI and UC Men

Smoking Status	# of Participants SI + UC	Deaths SI + UC	Relative Risk Quit/Not Quit (95% CI)	Adjusted Relative Risk[d] (95% CI)
		Coronary Heart Disease		
Quit[b]	908	12 (1.72)[c]	0.38 (.21, .68)	0.35 (.20,.63)
Not Quit	6035	205 (4.52)		
		Lung Cancer		
Quit[b]	908	9 (1.29)[c]	.96 (.48,1.93)	1.02 (.50,2.05)
Not Quit	6035	61 (1.34)		

[a] This represents an average of 7.5 years of followup
[b] Quitters are S_1 smokers who reported no smoking at years 1, 2 and 3 with Scn < 100 μ moles/L.
[c] Rates per 1000 person-years
[d] Adjusted for age, baseline cigarettes/day, cholesterol, blood pressure, education



If missed visit then participant is assumed smoking.
Cessation rates are adjusted for death.
*Smokers who maintained cessation from year one to the noted year of follow-up.
**Smokers who stopped, relapsed and stopped again; or smokers who stopped after year one and remained off cigarettes.
***Smokers who had not previously reported cessation in the trial but stopped after noted annual examination.

FIGURE 1—Distribution of Smoking Cessation History for Each Annual Visit for Special Intervention(SI) and Usual Care(UC) Smokers.
***Smokers who maintained cessation from year one to the noted year of follow-up.**
****Smokers who stopped, relapsed and stopped again; or smokers who stopped after year one and remained off cigarettes.**
*****Smokers who had not previously reported cessation in the trial but stopped after noted annual examination.**

(Reported rates are used in Figure 1 because by year six there was very little discrepancy between the reported rates and the rates validated by serum Scn.) Cross sectional data do not indicate what proportion of smokers stopped and continued off for the duration of the trial. Cohort data (vertical striped bar) indicate that 25 percent of the SI smokers (60 percent of those who quit in year one) remained abstinent through year six of the trial. For the UC smokers, 6.9 percent (54 percent of those who quit in year one) remained abstinent through year six.

The three groups—maintained stoppers, recycled stoppers, and new stoppers—were calculated for light (<25 cigarettes/day) versus heavy (≥25 cigarettes/day) smokers for the SI and UC groups, respectively. There were lower cross-sectional cessation rates among heavy smokers compared to light smokers in both the SI and UC groups; an approximately two-fold difference in maintained cessation rates for light versus heavy smokers; and a four-fold difference for SI versus UC smokers. For example, only 19.4 percent of SI heavy smokers stayed off of cigarettes for the six years of the trial and only 5.2 percent of UC heavy smokers did so.

Discussion

Smoking cessation during the trial had a substantial and fairly rapid beneficial effect on CHD mortality. The lack of an observable beneficial effect of stopping smoking on lung cancer at the 10.5 year follow-up may in part reflect the long lag period associated with the development of lung cancer. The MRFIT data are consistent with observational studies of lung cancer and smoking cessation indicating that the effect of smoking cessation is related to length of cessation and that there is still a substantial increase in risk for lung cancer among former smokers even after relatively long periods of cessation when compared to never-smokers.[6–8] Given that the MRFIT smoking data were prospectively collected, biochemically validated, and substantially more precise than data from any previous epidemiologic study, they strengthen previous evidence reflecting the immediate beneficial effect of smoking cessation on CHD and the long lag period with respect to a potential beneficial effect on lung cancer.

The sample size estimate for the MRFIT was based on CHD end points and changes in multiple risk factors. Therefore, it was not designed to detect the specific effect of smoking cessation nor differences in lung cancer mortality, which has a lower incidence than CHD mortality. The 90 percent confidence interval bounds for the +11.3 percentage point difference in lung cancer mortality rates for SI compared to UC smokers ranged from −19.2 to +53.3 percent. It is therefore unlikely that a substantial effect (i.e., greater than 20–30 percent lower lung cancer mortality for SI smokers), was missed due to lack of power.

To our knowledge, the MRFIT is only the second intervention trial which has examined the relationship between lung cancer mortality and smoking in randomized groups. The first such trial was the Whitehall Civil Servants Smoking Intervention Trial carried out by Rose and his colleagues.[27] After 10 years of follow-up, there were 22 lung cancer deaths in the intervention group and 25 in the normal care group. Intervention subjects (N = 714) reported a significant net decrease of 7.6 cigarettes/day compared to the control subjects (N = 731) after nine years of follow-up. This trial did not have adequate power to detect a difference in lung cancer. In addition, no objective measures of smoking were obtained. There are no data available from this trial with regard to persistence of quitting or cessation and maintenance rates for heavy versus lighter smokers.

The data confirm results from studies indicating an overwhelming smoking effect on lung cancer risk, with a greater relative risk for heavier smokers as compared to lighter smokers.[1,2,4,6–8] We found no effect of tar or nicotine levels on lung cancer either by univariate or multivariate analysis, suggesting that smokers should not develop a false sense of security by switching to lower tar and nicotine cigarettes.

In previous analysis, we have shown that dose of smoking is an important CHD risk factor when MRFIT smokers and ex-smokers are combined.[13,18] However, the number of cigarettes smoked per day at entry had little effect on risk for CHD when the analysis was limited to smokers only. In part, this is due to the multifactorial etiology of CHD, and may also be related to the focus of MRFIT recruitment on high-risk men, yielding a skewed distribution with a preponderance of relatively heavy smokers. Using a multivariate Cox Proportional Hazard model, baseline Scn level, which is a better marker of acute dose of exposure to cigarettes than reported number of cigarettes smoked, had a significant effect on CHD risk among smokers.

Quitters tended to gain more weight than non-quitters. However, the differences were modest and did not affect the relative risk estimates for CHD of quitters compared to non-quitters when included in the model. CHD deaths did occur among never-smokers, also confirming the multifactorial etiology of CHD.[3,28–34] By the nature of the MRFIT inclusion criteria, those individuals who were non-smokers on entry into the trial had relatively higher levels of serum cholesterol and blood pressure.

In most studies, a large number of persistent quitters is not available to adequately test the relationship of smoking

cessation to reduction in death rates; and most persistent quitters are lower risk, lighter smokers. This lack of persistent quitters is likely to attenuate the relationship between cessation and risk. If many years of persistent quitting is required to produce a beneficial effect on lung cancer mortality rates, it will be difficult to carry out studies to demonstrate such an effect.

The disparity in the beneficial effects of smoking cessation observed in this study between lung cancer and CHD mortality may help to explain the disparity seen nationally between sharply decreasing CHD mortality rates and lung cancer mortality rates which show little change. The greater smoking prevalence in earlier birth cohorts also accounts in part for the persistent high lung cancer death rates in these older individuals. Furthermore, some older smokers may have avoided cardiovascular death by smoking cessation and may now be at high risk of lung cancer, further increasing the pool of lung cancer susceptibles.

Lung cancer continues to be a major public health problem. Prevention of smoking and cessation at the youngest possible age for those individuals already smoking are the most important determinants of the future trends of lung cancer mortality.

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Onset of Fertility-Related Events during Adolescence: A Prospective Comparison of Mexican American and Non-Hispanic White Females

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Abstract: Ethnic differences in the first occurrence of fertility-related events are assessed for non-Hispanic White and Mexican American female adolescents. A community-based sample of 1,023 females ages 13 to 19 years was interviewed in 1984–85; 874 (85.4 percent) were reinterviewed approximately two years later. Mexico-born Mexican Americans have the lowest rate of early sexual intercourse, but the highest rate of early births because they are most likely to become pregnant if sexually active, and most likely to have a birth if pregnant. Non-Hispanic Whites have the highest rate of early sexual intercourse, but the lowest rate of early births because pregnant non-Hispanic Whites terminate pregnancies most often. US-born Mexican Americans are intermediate between the other two groups. Delays in the onset of sexual activity among Mexican Americans are not converted into corresponding delays in first pregnancies and births. Early marriage among Mexico-born Mexican Americans, however, accounts for much of the ethnic difference in early fertility. (*Am J Public Health* 1990; 80:959–963.)

Introduction

The well-known health, social, and economic costs associated with teenage fertility have made the prevention of teenage pregnancy a public health priority, particularly in the context of comparatively high US rates.[1] The early initiation of sexual intercourse lengthens the period of risk for teenage pregnancy; an additional consideration is the high rate of sexually transmitted disease among teens and the associated risk of HIV (human immunodeficiency virus) infection. Comparison of intergroup differences in teenage sexual initiation, contraception, pregnancy, abortion, and fertility have focused almost exclusively on the substantial differences existing between Black and White adolescents.[1–5] Comparatively little research has examined early fertility-related behavior among Hispanics in general or among discrete Hispanic subgroups. Yet recent research indicates that fertility among teenagers of Hispanic or Latino ethnic background is closer to the high rate of Black teens than to the lower rate of non-Hispanic White teens.[6–12]

Information about discrete Hispanic groups is limited because Hispanics often are analyzed as a single, undifferentiated social group. Mexican-origin women, however, have markedly higher fertility rates than Puerto Rican and Cuban women.[9] Similar intraethnic heterogeneity apparently exists among teens and young women.[7–10]

For Mexican American females, rates of early sexual intercourse, contraception, and abortion appear to be relatively low, in contrast to comparatively high rates of early fertility. A 1979 household survey of four border states revealed that Mexican American teenagers (15 to 19 years) were less likely than non-Hispanic Whites to be sexually active, and that sexually active Mexican Americans were less likely than non-Hispanic White teens to contracept.[14] We have shown that a high birth rate among Mexican American teens can be attributed to a small base of sexually active females who become pregnant and who elect live births (rather than abortions) at rates higher than the comparatively large base of sexually active non-Hispanic Whites.[12] High early fertility rates among Mexican American females, therefore, may coincide with relatively low rates of early sexual intercourse as a result of especially low rates of contraception among sexually active teens and/or low rates of abortion among pregnant teens.

This report extends previous research by examining multiple fertility-related events (early sexual intercourse, pregnancy, live births, and abortions) utilizing a prospective panel survey of a community-based sample. It also examines differences between Mexican American adolescents born in the United States and those born in Mexico.

Methods

Sample

The data for this study were derived from two sets of interviews conducted with an area probability sample of Los Angeles County Mexican-American and non-Hispanic White adolescent females. Time 1 interviews were conducted between January 1984 and May 1985 and time 2 interviews between January 1986 and May 1987. The average interval between completed interviews was 23 months.

A three-stage cluster sampling frame was used to select respondents: census tracts, blocks, and household addresses. Mexican American adolescents were oversampled by sampling two strata of census tracts: those with a high density (17.8 percent or more) of Hispanics and those with a plurality of non-Hispanic Whites. Sampled household addresses were screened to determine eligibility, defined as being a female resident of the household between the ages of 13 and 19 years who self-identified as being either Mexican American, Mexican or Chicano, or as being White and not of the above or other Latin American background.

One eligible respondent per household was selected using a modified version of the Kish selection method.[15] Mexican American households were more likely than non-Hispanic White households to contain more than one eligible adolescent (29.9 percent versus 19.2 percent). The data were weighted to adjust for this selection probability, but unweighted data are reported, following the suggestion of Kish, because weighted and unweighted analyses are virtually identical.[15]

Of 19,258 field-listed addresses, 861 were unoccupied dwelling units, 721 were not successfully screened, and

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16,552 screened households did not contain an eligible adolescent. Of the 1,124 known eligible households, 1,023 (91.0 percent) participated in the survey, 67 (6.0 percent) refused, and the remainder did not participate for other reasons. Of those interviewed, 874 (85.4 percent) were reinterviewed at time 2. Sample sizes for time 1 and time 2, respectively, are: 317 and 284 (90.0 percent) for non-Hispanic Whites; 398 and 358 (90.0 percent) for US-born Mexican Americans; and 308 and 232 (75.3 percent) for Mexico-born Mexican Americans. The cumulative response rate across time 1 and time 2 was 74.7 percent assuming proportional eligibility among unscreened households. The major source of attrition was an inability to locate participants who had moved, which accounted for 85.2 percent of the 149 nonrespondents. In addition, 10.7 percent were lost because of refusals, 1.3 percent were deceased, and 2.7 percent were lost for other reasons. Loss to follow-up was disproportionately high among Mexico-born Mexican Americans and among the sexually experienced, particularly those who had ever been pregnant.[16]

Detailed descriptions of the sampling procedures and characteristics of the sample are provided elsewhere.[12,16] The average socioeconomic status of the adolescent's family is lower for Mexican Americans than for non-Hispanic Whites, which conforms to census data for Los Angeles County.[17] In addition, the sample school dropout pattern conforms to ethnic patterns in the County.[18] In general, the characteristics of the sample are similar to those of the population from which it was selected.

Procedures

Interviews were conducted by professional female staff from the UCLA Institute for Social Science Research, Survey Research Center. Interviews lasted approximately one hour and were conducted in either English or Spanish. All of the time 1 interviews were conducted at the respondent's home, but 13.5 percent of the reinterviews were conducted by telephone, usually because the respondent had moved.

The interviews were similar at the two times and asked about a wide range of fertility-related attitudes and behavior; only a portion of the information obtained is reported here. The respondent's sexual status was ascertained by asking: "Sometimes people refer to sexual intercourse as 'doing sex,' 'having sex,' 'making love,' or 'going all the way.' Have you ever had sexual intercourse?" Respondents who answered in the affirmative were asked a series of questions concerning their sexual experiences including age of first intercourse and number of partners. Those who had ever been pregnant were asked whether each pregnancy mentioned resulted in a live birth, a stillbirth, a miscarriage, or an abortion.

Analysis

Lifetime prevalence estimates are based on the total sample at time 1, and on the reinterview sample at time 2. The combined age distribution for Hispanics and non-Hispanic Whites from the 1980 Census is used as the standard population to calculate sample weights for time 1 and time 2 that are equivalent to the direct standardization method of age-adjustment. Also, for prevalence estimates, the panel data are weighted to adjust for attrition utilizing a predicted probability of being reinterviewed.[19] Due to the complex sample, confidence intervals are probably slightly underestimated.

Survival analysis is employed for age of first intercourse, pregnancy, and birth because those negative on these events had not completed the period of risk. Those negative on events at reinterview are censored at their time 2 age, while those negative at time 1 and lost to follow-up are censored at their time 1 age. For nonmarital events, the married are censored at their age of marriage. Survival functions are evaluated through age 20 years. There are too few abortions across the age and ethnic groups to conduct meaningful analysis. Survival analyses were conducted with the BMDP Statistical Software program P1L.[19] The Savage-Mantel-Cox statistic is used to assess intergroup differences in the survival functions.[20]

Results

As shown in Table 1, the lifetime prevalence of sexual intercourse is substantially higher at follow-up than at base-

TABLE 1—Age-Adjusted Percent Sexual Intercourse, Pregnancy, Live Birth, and Abortion at Baseline and Follow-up by Ethnic Group

Fertility-Related Variable	Total Sample		Sexually Experienced		Ever Pregnant	
	Time 1	Time 2	Time 1	Time 2	Time 1	Time 2
Sexual Intercourse						
NHW	34.3 (29.1, 39.5)	61.8 (56.0, 67.6)	—	—	—	—
US-MA	27.8 (23.4, 32.2)	56.5 (51.2, 61.8)	—	—	—	—
M-MA	16.4 (12.2, 20.6)	39.2 (33.2, 45.2)	—	—	—	—
Pregnancy						
NHW	8.6 (5.7, 11.9)	20.4 (15.6, 25.2)	25.7 (17.5, 33.9)	33.0 (25.8, 40.2)	—	—
US-MA	12.1 (8.9, 15.3)	26.0 (21.3, 30.7)	43.6 (34.3, 52.9)	46.1 (39.0, 53.2)	—	—
M-MA	10.6 (7.1, 14.1)	29.1 (23.5, 34.7)	64.7 (51.5, 77.9)	74.4 (65.8, 83.0)	—	—
Live Birth						
NHW	3.3 (1.3, 5.3)	9.0 (5.6, 12.4)	9.7 (4.1, 15.3)	14.6 (9.2, 20.0)	37.9 (19.9, 55.9)	44.4 (31.1, 57.7)
US-MA	7.0 (4.5, 9.5)	18.5 (14.4, 22.6)	25.4 (17.2, 33.6)	32.9 (26.2, 39.6)	58.7 (44.6, 72.8)	72.0 (62.6, 81.4)
M-MA	8.2 (5.1, 11.3)	23.8 (18.6, 29.0)	49.7 (35.8, 63.6)	61.1 (51.5, 70.7)	76.8 (62.2, 91.4)	82.1 (73.4, 90.8)
Abortion (induced)						
NHW	4.8 (2.4, 7.2)	12.5 (8.5, 16.5)	13.9 (7.4, 20.4)	20.3 (14.1, 26.5)	54.1 (35.6, 72.6)	61.4 (48.4, 74.4)
US-MA	2.5 (1.0, 4.0)	3.1 (1.3, 1.9)	9.1 (3.7, 14.5)	5.5 (2.3, 8.7)	21.1 (9.4, 32.8)	11.9 (5.2, 18.6)
M-MA	0.5 (0.0, 1.3)	1.5 (0.0, 3.0)	3.2 (0.0, 8.1)	4.0 (0.2, 7.8)	5.0 (0.0, 12.6)	5.3 (0.2, 10.4)

NOTE: NHW = Non-Hispanic White; US-MA = US born Mexican American; M-MA = Mexico-born Mexican American. Time 2 percents are weighted to adjust for sample attrition. Live birth and abortion percents among the ever-pregnant do not sum to 100 percent because of repeated pregnancies, small numbers of spontaneous abortions, and current first pregnancies. Time 1, Unweighted N, Total Sample: NHW = 315; US-MA = 396; M-MA = 305; Sexually Experienced: NHW = 135; US-MA = 86; M-MA = 41; Ever Pregnant: NHW = 26; US-MA = 33; M-MA = 25. Time 2, Unweighted N, Total Sample: NHW = 281; US-MA = 358; M-MA = 227; Sexually Experienced: NHW = 157; US-MA = 176; M-MA = 74; Ever Pregnant: NHW = 44; MA-US = 72; M-MA = 52. 95% confidence interval in parentheses.

line for all three ethnic groups, as would be expected given that the sample has aged, on average, two years. The prevalence of sexual intercourse is substantially lower among Mexico-born Mexican Americans than among the other two groups, with non-Hispanic Whites having the highest lifetime prevalence of sexual intercourse at baseline and at follow-up.

The percent ever pregnant more than doubles across time for all three groups. Non-Hispanic Whites have the lowest rate of ever having been pregnant at both times, with US-born Mexican Americans and Mexico-born Mexican Americans having the highest rates at time 1 and time 2, respectively. At both times, however, the confidence intervals overlap for all three groups. Among only those who have ever had sexual intercourse, the percent ever-pregnant is substantially higher among Mexico-born Mexican Americans than among non-Hispanic Whites at both baseline and follow-up: US-born Mexican Americans are intermediate between these other groups. The increase across time is modest, indicating a similar risk of pregnancy.

For the percent ever having had a live birth, similar ethnic differences are observed for the total sample, those who have ever had sexual intercourse, and those who have ever been pregnant. This percentage is highest for Mexico-born Mexican Americans and lowest for non-Hispanic Whites. These ethnic differences are more pronounced for the sexually experienced and the ever-pregnant than for the total sample and tend to be somewhat more pronounced at follow-up than at baseline.

Ethnic group differences in abortion mirror the live birth pattern. Non-Hispanic Whites have the highest percent having had an abortion at both times and the greatest increase across time. Among the sexually experienced and the ever-pregnant, the percent who have had an abortion decreases over time among US-born Mexican Americans.

Non-Hispanic White female adolescents are substantially more likely than Mexican-American adolescents to have had multiple sexual partners, although the mode is one partner for all three groups. For non-Hispanic Whites, 29.7 percent have had only one partner, with 12.1 percent having more than 10 partners. For Mexican Americans, 48.9 percent of the US-born and 79.6 percent of the Mexico-born have had only one partner, with 10 being the maximum number of partners in both groups. This difference persists when the number of years of sexual activity is controlled statistically.

The survival functions for age of initiating sexual intercourse differ among the three ethnic groups (Savage-Mantel-Cox statistic = 47.25, d.f. = 2, $p < .001$). As shown in Table 2, the cumulative proportion surviving without initiating intercourse is substantially higher among Mexico-born Mexican Americans than among the other groups from the age of 15 years onwards. The median survival age is substantially older among Mexico-born Mexican Americans (19.7 years) than US-born Mexican Americans (17.8 years), or non-Hispanic Whites (17.4 years).

The hazard rates, the probability of first intercourse at a specific age given no intercourse before that age, also differ by ethnic group, as shown in Table 2. For non-Hispanic Whites and US-born Mexican Americans, marked increases occur between the ages of 15 and 17 years. For the Mexico-born, the hazard rate increases substantially between 17 and 19 years. The peak hazard rate for all three groups is between 18 and 19 years.

Contrastingly, the survival functions for age of first pregnancy are similar across these three groups (Savage-Mantel-Cox = 3.37, d.f. = 2, $p > .10$). The decline in the proportion surviving without a pregnancy is somewhat more marked among the two Mexican American groups than among non-Hispanic Whites, as shown in Table 3. For both Mexican American groups, hazard rates are relatively low until a sharp increase occurs between 17 and 18 years. For non-Hispanic Whites, the hazard rate increases approximately a year later.

Despite similar patterns for pregnancy, there are substantial ethnic differences in the survival functions for first birth (Savage-Mantel-Cox statistic = 13.30, d.f. = 2, $p < .005$). As shown in Table 4, the cumulative proportion surviving without a live birth is similarly high across ethnic groups until age 17 years, and thereafter declines more rapidly among the two Mexican American groups than among the non-Hispanic White group. The pattern for the two Mexican American groups is similar until age 18; thereafter the proportion surviving declines more among the Mexico-born than among the US-born. For non-Hispanic Whites, the hazard rate is highest between the ages of 17 and 18 years. At this age, the Mexican-American groups have a similar hazard rate, but this rate increases further over time.

Ethnic differences in early marriage may contribute to these patterns. The percent ever-married is substantially higher among Mexico-born Mexican Americans (14.3 percent) than among US-born Mexican Americans (6.5 percent) or non-Hispanic Whites (4.7 percent). Moreover, among the ever-married, the Mexico-born are most likely to have married prior to first sexual intercourse, pregnancy, and live birth. For US-born Mexican Americans and non-Hispanic Whites, prevalence rates for nonmarital fertility events are quite similar to those for all events. For Mexico-born Mexican Americans, however, the prevalence rates for nonmarital events are substantially lower than all events. The

TABLE 2—Survival Analysis of Age of First Sexual Intercourse by Ethnic Group

Age (Years)	Cumulative Proportion Surviving			Hazard Rate		
	NHW	US-MA	M-MA	NHW	US-MA	M-MA
12	.981 (.008)	.995 (.004)	.984 (.007)	.019 (.008)	.005 (.004)	.017 (.007)
13	.942 (.013)	.962 (.010)	.970 (.010)	.040 (.012)	.034 (.009)	.014 (.007)
14	.874 (.019)	.928 (.013)	.945 (.013)	.079 (.017)	.033 (.010)	.026 (.010)
15	.767 (.025)	.802 (.021)	.894 (.019)	.130 (.024)	.146 (.022)	.056 (.016)
16	.559 (.031)	.631 (.027)	.831 (.024)	.314 (.044)	.241 (.034)	.072 (.020)
17	.406 (.033)	.475 (.031)	.704 (.033)	.317 (.058)	.282 (.048)	.166 (.036)
18	.273 (.034)	.334 (.033)	.567 (.040)	.391 (.090)	.349 (.073)	.215 (.053)
19	.224 (.036)	.249 (.036)	.465 (.047)	.195 (.097)	.291 (.102)	.197 (.069)
20	.168 (.044)	.187 (.047)	.403 (.057)	—	—	—

NOTE: Standard errors in parentheses. NHW = Non-Hispanic White, N = 314; US-MA = US-born Mexican American, N = 397; M-MA = Mexico-born Mexican American, N = 303.

TABLE 3—Survival Analysis of Age of First Pregnancy by Ethnic Group

Age (Years)	Cumulative Proportion Surviving			Hazard Rate		
	NHW	US-MA	M-MA	NHW	US-MA	M-MA
12	1.000 (.000)	1.000 (.000)	.997 (.003)	.000 (.000)	.000 (.000)	.003 (.003)
13	.990 (.006)	1.000 (.000)	.997 (.003)	.010 (.006)	.000 (.000)	.000 (.000)
14	.984 (.007)	.990 (.005)	.975 (.009)	.007 (.005)	.010 (.005)	.022 (.009)
15	.952 (.013)	.937 (.013)	.943 (.014)	.033 (.011)	.054 (.013)	.033 (.012)
16	.909 (.018)	.898 (.017)	.914 (.018)	.046 (.015)	.043 (.013)	.032 (.013)
17	.876 (.022)	.806 (.025)	.826 (.028)	.037 (.015)	.107 (.024)	.101 (.027)
18	.794 (.031)	.734 (.031)	.755 (.035)	.098 (.030)	.094 (.028)	.090 (.032)
19	.725 (.039)	.636 (.039)	.599 (.049)	.090 (.037)	.144 (.045)	.231 (.066)
20	.636 (.059)	.558 (.054)	.507 (.064)	—	—	—

NOTE: Standard errors in parentheses. NHW = Non-Hispanic White, N = 314; US-MA = US-born Mexican American, N = 396; M-MA = Mexico-born Mexican American, N = 302.

TABLE 4—Survival Analysis of Age of First Live Birth by Ethnic Group

Age (Years)	Cumulative Proportion Surviving			Hazard Rate		
	NHW	US-MA	M-MA	NHW	US-MA	M-MA
12	1.000 (.000)	1.000 (.000)	.997 (.003)	.000 (.000)	.000 (.000)	.003 (.003)
13	1.000 (.000)	1.000 (.000)	.997 (.003)	.000 (.000)	.000 (.000)	.000 (.000)
14	.997 (.003)	.995 (.004)	.993 (.005)	.003 (.003)	.005 (.004)	.004 (.004)
15	.993 (.005)	.974 (.008)	.977 (.009)	.004 (.004)	.021 (.008)	.016 (.008)
16	.980 (.009)	.945 (.013)	.947 (.015)	.013 (.008)	.030 (.011)	.031 (.013)
17	.941 (.017)	.897 (.019)	.909 (.021)	.041 (.015)	.052 (.017)	.041 (.017)
18	.918 (.021)	.799 (.029)	.803 (.034)	.025 (.014)	.116 (.030)	.124 (.036)
19	.895 (.026)	.750 (.035)	.709 (.045)	.026 (.018)	.063 (.028)	.124 (.047)
20	.832 (.049)	.674 (.052)	.643 (.060)	—	—	—

NOTE: Standard errors in parentheses. NHW = Non-Hispanic White, N = 313; US-MA = US-born Mexican American, N = 395; M-MA = Mexico-born Mexican American, N = 299.

cumulative proportion surviving to age 20 years without nonmarital intercourse is .183 (SE = .046) for non-Hispanic Whites, .201 (SE = .050) for US-born Mexican Americans, and .637 (SE = .046) for Mexico-born Mexican Americans (Savage-Mantel-Cox Statistic = 72.25, d.f. = 2, $p < .001$; see Table 2). For nonmarital pregnancy, the three ethnic groups remain similar (Savage-Mantel-Cox Statistic = 3.73, d.f. = 2, $p > .10$), with the proportion surviving to age 20 years being .673 (SE = .060) for non-Hispanic Whites and .616 (SE = .057) for Mexico-born and .695 (SE = .070) for US-born Mexican Americans (see Table 3). In contrast, the survival functions differ by ethnicity for nonmarital births (Savage-Mantel-Cox Statistic = 9.49, d.f. = 2, $p < .01$), with the cumulative proportion surviving to age 20 years being .869 (SE = .048) for non-Hispanic Whites, .772 (SE = .043) for US-born Mexican Americans, and .876 (SE = .052) for Mexico-born Mexican Americans (see Table 4).

Discussion

In sum, non-Hispanic Whites have the lowest level of early fertility despite having the highest level of sexual activity because sexually experienced non-Hispanic Whites are the least likely to become pregnant and because pregnant non-Hispanic Whites are most likely to elect abortion. In sharp contrast, Mexico-born Mexican Americans have the lowest level of early sexual intercourse, but are most likely to become pregnant if they have sexual intercourse, and most likely to have a birth if they become pregnant. Consequently, the Mexico-born have the highest fertility rate. US-born Mexican Americans tend to be intermediate between the other two groups across the various events studied. Age of first pregnancy is similar across the three ethnic groups, but Mexico-born Mexican Americans initiate sexual intercourse at later ages than the other two groups, and both Mexican American groups have first births at earlier ages than non-Hispanic Whites.

The role of early marriage in fertility-related events is most pronounced among Mexico-born Mexican Americans. Only a small proportion of young women in any of these groups marry early, but the somewhat greater propensity of Mexico-born Mexican Americans to do so accounts for most of the difference in early fertility in comparison to non-Hispanic Whites. Early marriage, however, does not account for differences in early fertility between US-born Mexican Americans and non-Hispanic Whites. Thus, while Mexico-born Mexican Americans are most likely to have early births overall, US-born Mexican Americans are most likely to have early nonmarital births.

The estimates of early fertility obtained in this investigation for Los Angeles County females are somewhat below the national estimates provided by Michael and Tuma.[6] These differences may be due to regional effects, the later study date of the present investigation, differences in study procedures, or variation within the Hispanic population. In general, the age-specific rate of first births (see Table 4) closely approximates the 1980 age-specific fertility rate for Los Angeles County for Hispanics and for non-Hispanics Whites.[11] These rates are not directly comparable for a variety of reasons. Nonetheless, this similarity suggests that underreporting of live births by study participants is not a serious source of misestimation.

These data reveal substantial potential risk for adolescent females for transmission of HIV infection or other sexually transmitted disease through multiple sexual partners. All else being equal, non-Hispanic White females would

be at highest risk because they have the highest rate of early sexual intercourse and are most likely to have multiple sexual partners. While the relative risk of AIDS is higher among Hispanic than non-Hispanic White men, this higher risk appears limited to Puerto Ricans rather than Mexican Americans.[21] Assuming within-group selection of sexual partners, young Mexico-born Mexican American females, therefore, would appear to be at low risk, due to their relatively late age of initiating sex and generally low number of partners.

Public policy debate concerning teenage pregnancy centers upon the alternatives of primary prevention (reducing risk exposure by delaying the onset of sexual activity) versus secondary prevention (reducing the impact of exposure by increasing effective contraception among the sexually active). The gains that can be achieved through primary prevention are greatest for groups with high exposure, i.e., those who initiate sexual intercourse early—non-Hispanic Whites and US-born Mexican Americans. Relative to these groups, Mexico-born Mexican Americans delay the onset of sexual activity considerably, but nonetheless experience high fertility. Conversely, the gains that can be achieved through secondary prevention are greatest for groups with high rates of pregnancy among the sexually active. Although such pregnancy rates are high for all three ethnic groups, the most vulnerable are the two Mexican American groups—the Mexico-born for all pregnancies and the US-born for nonmarital pregnancies. Effective primary or secondary interventions would serve to diminish early fertility across ethnic groups. Our results suggest, however, that the potential reduction in early births that can be realized at various junctures in the sequence of fertility-related events may differ by ethnicity.

The timing of such interventions is critical. Common wisdom asserts that prevention should commence early, a recommendation supported by the early onset of sexual activity documented here. The high hazard rates during the middle to late teen years, however, indicate that prevention activities should continue throughout the adolescent years. Moreover, given the accelerating pattern of sexual activity with increasing age, the focus of prevention activities should shift over time toward a greater concentration on secondary prevention. Also, such prevention activities need to incorporate consideration of marital as well as nonmarital contraception. Programs that focus solely on preventing nonmarital sexual relations leave young women unprepared for reproductive decisions within marriage.

Finally, public policy and funding decisions pertaining to abortion are likely to impact most upon non-Hispanic White adolescents. While young women in all three ethnic groups terminate pregnancies, non-Hispanic Whites are most likely to resolve pregnancies with abortions. Reduction in the availability of abortion, particularly to juveniles, is therefore likely to increase births sharply among young non-Hispanic Whites, but to have comparatively little impact among young Mexican Americans.

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Prior Condom Use and the Risk of Tubal Pregnancy

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Abstract: The relationship between prior condom use and tubal pregnancy was assessed in a population-based case-control study at Group Health Cooperative of Puget Sound during 1981–86. We interviewed 227 women with a tubal pregnancy who had no clinical indication of infertility and no history of sterilization and 674 similarly defined controls who were matched to the cases on age and county of residence. A history of condom use for more than one year was associated with a decreased risk of subsequent tubal pregnancy (RR = 0.74, 95% CI = 0.44, 1.26) adjusted for the effects of age, current use of contraceptive methods, educational level, and age at first intercourse. The effect was more pronounced when condoms had been used during five-year periods with more than one partner (RR = 0.38, 95% CI = 0.15, 1.0) than during five-year periods with one partner (RR = 0.89, 95% CI = 0.45, 1.76). Condom use for less than one year was unrelated to risk of ectopic pregnancy. Since the use of condoms offers protection against sexually transmitted diseases, one or more of which are likely to be causally related to tubal pregnancy, the observed negative association plausibly represents a protective influence of long-term condom use on the occurrence of tubal pregnancy. (*Am J Public Health* 1990; 80:964-966.)

Introduction

Some sexually transmitted diseases (STDs) are now considered to be major risk factors for tubal pregnancy,[1] and condom use is believed to be protective against sexually transmitted diseases,[2] so condom use should protect women against tubal pregnancy. Nevertheless, direct evidence to support this inference is lacking. We therefore analyzed data gathered in a population-based case-control study to assess whether condom use is associated with a reduced risk of subsequent tubal pregnancy.

Methods

Details of the study methods can be found elsewhere.[3] Briefly, all subjects in this study were members of the Group Health Cooperative (GHC), a health maintenance organization in the Puget Sound area of Washington State. We identified 396 newly diagnosed cases of ectopic pregnancy through a computerized hospital discharge system between October 1981 and September 1986. After 11 (2.8 percent) refusals for participation from patients' physicians, 39 (9.8 percent) refusals from patients themselves, and nine (2.3 percent) lost to follow-up, 337 (85.1 percent) cases participated in the study.

Controls were randomly selected from the GHC membership file from the calendar quarter of the diagnosis of the tubal pregnancy, and were matched to cases on age and county of residence. In order to be eligible for participation in the study, controls had to be at risk of conceiving and being diagnosed with a tubal pregnancy, i.e., sexually active within 12 months prior to the time of being selected; without a male sexual partner(s) who was known to be sterile; and not pregnant, or less than four months pregnant, at the time selected. Of 1,139 controls initially chosen, 32 (2.8 percent) were not available for the study due to their physicians' refusal, 168 (14.7 percent) refused to participate, 46 (4.0 percent) were lost to follow-up. A total of 893 (78.4 percent) control subjects agreed to participate in the study.

A structured personal interview was conducted with each subject to obtain information on demographic characteristics, reproductive history, contraceptive experience, and medical history. Women were asked not only the total number of sexual partner(s) and the age of first intercourse, but also the number of sexual partners during specific intervals: less than 16 years of age, ages 16–20, 21–25, 26–30, 31–35, and 36–40. Details of life-time contraceptive practices included the types of contraceptive methods used, brand (if applicable), starting and ending dates, and the reasons for stopping contraception.

In order to make case and control groups comparable with respect to their opportunity for exposure (i.e., duration of condom use), women with characteristics likely to be related independently to the occurrences of tubal pregnancy—a history of previous sterilization or previous infertility tests—were excluded from the analysis. Furthermore, those with a prior ectopic pregnancy and those whose ectopic pregnancy was not in the fallopian tube were also excluded, leaving 227 cases and 674 controls available for analysis. A "reference date" was established for each subject—the estimated date of conception for cases and the mid-point of that same quarter for controls—and only exposures at and prior to that time were ascertained.

To determine if the association of condom use and tubal pregnancy varied with subject's sexual behavior, the relation was evaluated separately for those five-year periods during which there had or had not been more than one sexual partner.

The odds ratio, accompanied by its 95% confidence interval, was used as an estimate of relative risk. Logistic regression analysis was employed to adjust for potential confounders.[4]

Results

Table 1 presents some characteristics of the study subjects. Age remained comparable between case and control groups after the exclusions made in this study. Compared to controls, cases were more likely to have been married (or living as married) at the date of diagnosis of the tubal pregnancy, to be Black, and to have had less education.

Cases were more likely than controls to have had a greater total number of sexual partners, to have become

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TABLE 1—Characteristics of Cases and Controls

Characteristics	Percentage	
	Cases (N = 227)	Controls (N = 674)
Age (years)		
<20	4.0	2.4
20–24	18.5	18.5
25–29	29.1	30.9
30–34	35.7	34.9
≥35	12.8	13.4
Marital status		
Stable[a]	92.1	83.9
Separated or divorced	4.8	6.4
Never married	3.1	9.8
Race		
White	81.1	87.1
Black	13.7	5.8
Other	5.3	7.1
Education		
>high school	64.3	72.0
Age at first intercourse (years)		
≤16	35.7	23.0
17–19	45.4	43.8
≥20	19.0	33.3
Number of sexual partners, life-time		
One	13.3	27.5
2–4	35.8	31.1
5–14	34.5	28.3
≥15	16.4	13.1
History of gonorrhea		
Yes	12.9	6.9
Current contraceptive use		
No	73.2	19.1
The Pill	5.7	29.1
IUC	7.9	8.8
Other[b]	13.2	42.3

[a]Including married, living as married and regular sexual partnership.
[b]Including barrier methods and withdrawal.

sexually active at an earlier age, and to have had a history of infection with gonorrhea (Table 1). Cases were far less likely than controls to have been using any kind of contraceptive method at the reference date, although use of the intrauterine device (IUD) at that time was comparable between the two groups (Table 1).

The relation between duration of condom use and tubal pregnancy is shown in Table 2. Condom use was associated with a decreased risk of a subsequent occurrence of tubal pregnancy only when it had been used for a relatively long time (i.e., more than one year). After taking into account her age, current contraceptive use, educational level, and age at first intercourse, a woman whose partners used condoms for more than one year during any five-year period in which she had more than one sexual partner had only 38 percent of the risk for subsequent occurrence of tubal pregnancy compared to never-users (95% CI = 0.15, 1.0). Condom use restricted to periods in which the woman had only one sexual partner was not associated with a decreased risk of tubal pregnancy, nor was condom use for less than one year, irrespective of the number of partners. Further adjustment for the following variables did not influence the results: number of life-time sexual partners, history of gonorrhea infection, oral contraceptive and IUD use, reproductive history including gravidity, parity, spontaneous and induced abortion, religious briefs, smoking habits, the year of diagnosis of ectopic pregnancy, family income, and race. The number of five-year intervals in which more than one partner had been present was similar between case and control women, and so this did not produce differences in the size of the association with condom use.

Discussion

Our finding of a negative association between prior condom use and the occurrence of tubal pregnancy might have been anticipated, given that condom use can reduce the risk of transmission of many forms of sexually transmitted diseases, some of which are important etiologic factors for tubal pregnancy. Nonetheless, it is necessary to keep in mind several limitations of our study when interpreting these results. First, our ability to assess exposure to sexually transmitted diseases themselves was extremely limited. We were able to measure only a distant correlate of such exposure—the presence of more than one sexual partner during various periods of a woman's reproductive life. Thus, our finding of a reduced risk of tubal pregnancy associated with condom use restricted to women whose partner's use occurred during periods of her life in which she had more than one partner could be a reflection of yet a far greater protective effect of condom use among women who truly had been exposed to one or more sexually transmitted diseases. On the other hand, we made numerous comparisons in this data set, and the restriction of a negative association to this subgroup of women could have been simply the result of chance.

Second, the control group of women chosen in this study—women capable of becoming pregnant—had certain disadvantages in examining the influence of prior condom use. The use of the particular control group was dictated by our interest in studying the influence of exposures at the time of conception (e.g., IUD use), plus our concern that the commonly used alternative group (women completing an intrauterine pregnancy) provided a substantial opportunity for bias,[5] since a sizable portion of women who conceive a pregnancy do not go on to deliver it. The selection of controls who were believed to be capable of becoming pregnant led to a large case-control difference in the frequency of contraception (of any type, see Table 1) at the reference date. As expected, far fewer cases than controls were attempting to contracept at that time. Nonetheless, since there were sizable numbers of contraceptors and noncontraceptors among both cases and controls, and since we were interested in the effect of condoms on tubal pregnancy above and beyond their ability to prevent pregnancy per se, we were able to adjust for the use of contraception at reference date and thus obtain an unconfounded (with respect to this factor) evaluation of the influence of past condom use.

In our analysis, we sought to address the possibility that the reduced risk among women whose partner(s) had used a condom resulted only from an increased risk associated with use of certain other forms of contraception. First, we restricted the analysis to women who had not been sterilized. Then, we assessed the influence of condom use adjusting for prior use of contraceptive methods that plausibly could affect the occurrence of pelvic infection (and thus tubal pregnancy), i.e., IUDs and oral contraceptives.[1, 6] A negative association between condom use and tubal pregnancy persisted despite such adjustments.

While several studies of tubal pregnancy have examined the influence of barrier methods of contraception in general,[1, 7–10] only our earlier[9] and current studies focused on prior condom use. The earlier study did find a 60 percent reduction in the incidence of tubal pregnancy among women whose partner(s) had used a condom, but neither duration of use nor the further influence of number of partners was analyzed. Although the use of barrier methods in general has been found

TABLE 2—Logistic Regression Results of the Relation between Condom Use and Tubal Pregnancy

	Cases (%)	Controls (%)	Crude RR (95% CI)	RRa[a] (95% CI)
Duration (years) of total condom use				
Never[c]	176 (77.5)	488 (72.4)		
≤1	24 (10.6)	52 (7.7)	1.28 (0.77,2.14)	1.47 (0.79,2.74)
>1	27 (11.9)	134 (19.9)	0.56 (0.36,0.87)	0.74 (0.44,1.26)
Duration (years) of condom use during 5-year time interval in which there was >1 partner				
Never[c]	156 (86.7)	366 (82.1)		
≤1	17 (9.4)	39 (8.7)	1.02 (0.56,1.86)	1.49 (0.72,3.09)
>1	7 (3.9)	41 (9.2)	0.40 (0.18,0.91)	0.38 (0.15,1.00)
Duration (years) of condom use during 5-year time interval in which there was only one partner[b]				
Never[c]	144 (84.2)	400 (78.3)		
≤1	12 (7.0)	39 (7.6)	0.85 (0.44,1.68)	0.94 (0.43,2.05)
>1	15 (8.8)	72 (14.1)	0.58 (0.32,1.04)	0.89 (0.45,1.76)

[c]Reference group.
[a]Risk relative to the women who never used condoms adjusted for age, current contraceptive use, education level and age at first intercourse that were categorized as shown in Table 1.
[b]Women whose partners used condoms during any five-year period in which there had been more than one sexual partner were excluded.

to be associated with a modest reduction in the risk of tubal pregnancy,[7,8,10] the possibly different impact on tubal pregnancy of individual barrier methods limits the interpretation of the results with respect to any one of the methods. In addition, by studying current users versus current non-users,[8,10] previous studies allowed for the possibility that the low risk was due simply to the ability of barrier methods to prevent conception.

In conclusion, after controlling the "need" for contraceptive use, age, current use of contraceptive methods, education level, and age at first intercourse, we found that use of a condom for more than one year reduced the risk of tubal pregnancy. The effect was more pronounced when condom use was during any five-year period in which there had been more than one sexual partner. This observed negative association may plausibly represent a protective effect of long term use of condom on the occurrence of tubal pregnancy through the prevention of one or more sexually transmitted diseases.

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Age and Human Immunodeficiency Virus Infection in Persons with Hemophilia in California

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Abstract: Thirteen hemophilia centers provide comprehensive care to approximately 90 percent of persons with hemophilia in California. For 1987, these centers reported patient human immunodeficiency virus (HIV) antibody status, age group, level of clotting factor deficiency, and hemophilia type on 1,438 persons with hemophilia A and B; HIV serologic status was known for 860 persons (59.8 percent) of whom 537 (62.4 percent) were HIV-antibody-positive. The HIV positivity rate increased with age after taking into account hemophilia type, clotting factor level and treatment center type. The three-year cumulative incidence of reported AIDS (acquired immunodeficiency syndrome) cases based on the number of HIV positive patients, was 11.6 percent. The cumulative incidence rate was 14.6 percent (54 of 370) for those patients over 20 years of age and 4.8 percent (8 of 167) for those under 21 years of age. Although a comparable distribution of the date of diagnoses of AIDS was seen by age group, there appeared to be a bimodal distribution in the rate of AIDS among the age groups, with the 6–12-year-olds and the 21 and older age groups showing higher incidence rates. (*Am J Public Health* 1990; 80:967–969.)

Introduction

Acquired immunodeficiency syndrome (AIDS) was first documented in persons with hemophilia early in the course of the AIDS epidemic in the United States.[1] Studies of stored serum specimens showed that a high percentage of hemophilic persons became infected with human immunodeficiency virus (HIV) over a relatively short time. Most patients became infected between 1981 and 1984; the peak incidence of infection occurred in 1982 and 1983.[2,3] The reasons for the quick spread of infection in this population were the widespread use of commercial clotting factor concentrates prepared from pools of sera collected from between 2,000 to 20,000 donors[4] and the widening prevalence of this infection in the United States, and therefore in the donor population, before HIV blood screening practices were in effect.

The Centers for Disease Control (CDC) collects data characterizing AIDS in the United States hemophilic population through state health departments.[5,6] This reporting mechanism has been exceptionally efficient, with at least 97 percent of the cases reported.[5]

The duration of HIV infection before the onset of AIDS varies from as short as a few months to an as yet indeterminate number of years, with a median of nearly 10 years.[7] However, the duration or latency period to AIDS in persons with hemophilia may differ from that of other AIDS risk groups.[8–10] The most important factor in disease progression is time from primary infection, but factors as diverse as geographic location[11] and HLA (human lymphocyte antigen) type[12] may play a role. In this study, the HIV seroprevalence and incidence of AIDS in hemophilic persons in California were estimated to assess associations with age, hemophilia type, and clotting factor level.

Methods

Five federally funded California hemophilia centers (FFC) provide comprehensive care to approximately 65 percent of the persons with hemophilia A and B in California[13] and report activities annually to the federally funded agency. An additional 25 percent of the patients receive care through eight other centers (SSC) and report voluntarily. It is estimated that the remaining 10 percent of the patients receive care through health maintenance programs, the military, or private practitioners. As part of their 1987 annual report, both groups of centers were asked to include additional information for all hemophilia patients on HIV antibody test result (positive, negative, or not tested); age group of patient (<5 years, 6 to 12 years, 13 to 16 years, 17 to 20 years, or above 20 years); level of clotting factor deficiency (<5 percent or 5 percent–40 percent); and type of hemophilia (Factor VIII or Factor IX deficiency). The HIV antibody testing of hemophilia persons is voluntary, with encouragement to all patients. Mild hemophilia persons were usually followed less actively at the centers, and centers were less aggressive about testing younger children since they were much less likely to be infected because viral-activated products had usually been used.

For this study, AIDS cases were those reported to CDC as of 9/2/88 by any center, physician, and local department of health within California, that were diagnosed between 1/1/85 and 12/31/87, and were verified by the reporting official to have received their care from one of the 13 reporting centers. The inclusive time period of 1985–87 was used because use of viral inactivated products was instituted in 1984, and seroconversion had virtually ceased by 1985.[14] Thus the number of seropositive persons was stable for this period and the cumulative incidence of AIDS is the number of reported AIDS cases divided by the number of known HIV seropositive patients during the inclusive three-year period of 1985–87. The age group of AIDS cases is based on age as of 1987, the year of the annual report. Similar analyses were repeated when California hemophilia-associated AIDS cases included those not reported by FFCs or SSCs.

Logistic regression analysis was used for both the univariate and multivariate analyses to calculate odds ratios (OR) and 95 percent confidence intervals (CI).[15,16] Univariate analyses were performed on each of the four categorical variables (age group, hemophilia type, clotting factor level, and treatment center type). Next, for HIV-tested patients, a multivariate analysis was used to analyze the four variables in a full model to adjust for the effects of potential confound-

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ing variables. Logistic regression analysis was also used to compare between the HIV-positive patients in relation to outcome of AIDS. For this analysis, the three categorical variables were age group (≤20 and ≥21 years of age), hemophilia type, and clotting factor level.

Results

Most patients (72.0 percent) were reported from the five major centers which also tested proportionally more patients (Table 1). Clotting factor levels <5 percent and age were also associated with testing, but factor deficiency was not associated.

Of the 860 tested patients, 537 (62.4 percent) were HIV-antibody-positive and 323 (37.6 percent) were antibody-negative. The odds ratio of HIV seropositivity increased with age group (Table 2). The odds of HIV seropositivity may have stabilized beginning with the 13–16-year age group: ORs of the 13–16 and 17–20 year-old age groups did not differ from those 21 years of age or older (OR = 1.3, CI = [0.7–2.4]; OR = 1.5, CI = [0.9–2.7], respectively). Factor VIII deficient patients were more likely to be HIV positive than factor IX deficient patients, and patients with clotting factor levels <5 percent were more likely to be HIV positive than patients with clotting factor levels ≥5 percent. Using a full logistic model, there were no differences in HIV positive rates in patients from the two groups of centers; the differences in HIV positive rates by age, hemophilia type, and clotting level remained.

TABLE 1—HIV Testing Status of Hemophilia Patients by Patient Characteristics

Characteristics	Number of Patients	Percent Tested	OR	(95% CI*)
Age Group (year)				
≤5	155	45.8	1.0**	
6–12	222	57.2	1.6	(1.0, 2.4)
13–16	98	57.1	1.6	(0.9, 2.6)
17–20	127	62.2	1.9	(1.2, 3.1)
≥21	836	63.0	2.0	(1.4, 2.9)
Factor Deficiency				
IX	306	55.2	1.0**	
VIII	1132	61.0	1.3	(1.0, 1.6)
Clotting Factor Level				
≥5%	404	44.8	1.0**	
<5%	1034	65.7	2.4	(1.9, 3.0)
Treatment Center Type				
SSC	402	49.8	1.0**	
FFC	1036	63.7	1.8	(1.4, 2.2)

*95% confidence interval.
**Reference group.

Sixty-two patients from the hemophilia reporting centers were reported with an AIDS diagnosis made in 1985–87 in California. This represented 11.6 percent of known seropositive patients in the centers. Age was the only factor associated with AIDS diagnosis (Table 3). Eight of 167 of those less than 21 years old developed AIDS, six persons 6–12 and 2 persons 17–20. Distribution of the date of diagnosis for the eight persons <21 years of age was similar to that of the older population. However, a difference in the rate of AIDS was seen between age groups. This distribution was bimodal with the 6–12-year-old group showing higher rate (10.0 percent) than those in the 13–16 and 17–20 age groups (0.0 percent and 3.2 percent, respectively), although not as high as the ≥21 age group. Results similar to those described above were also seen in the analysis of all 72 patients who were reported by the centers as developing AIDS through 1987.

Discussion

Most persons with hemophilia became infected with HIV in 1981 to 1984.[2,3] After 1984, viral-attenuated clotting factor concentrates became widely used resulting in only rare seroconversions in uninfected persons.[17] Therefore, a low seroprevalence in hemophilic children under 6 years of age and an increasingly higher prevalence in older persons with hemophilia was to be expected.[18] In addition, we found this association to remain when taking into account the hemophilia type, clotting factor level, and treatment center type. We also found that older persons with hemophilia were at a higher risk of AIDS, as was shown in a recent study.[19] Although patients between 6 and 12 years had a higher rate than those of other age groups <21 years, there was a small number of cases in each of those age groups.

Why patients followed by one set of centers were more likely to be tested than patients from the other set is not

TABLE 2—HIV Serologic Status of Patients by Patient Characteristics

Characteristics	No.	Percent HIV+	Univariate OR	Univariate (95% CI*)	Multivariate OR	Multivariate (95% CI*)
Age Group (year)						
≤ 5	71	4.2	1.0**		1.0**	
6–12	127	47.2	20.3	(8.1, 67.9)	22.2	(6.6, 75.0)
13–16	56	75.0	68.0	(18.4, 250.7)	91.2	(23.9, 347.6)
17–20	79	78.5	82.7	(23.1, 295.8)	152.0	(40.8, 566.2)
≥21	527	70.2	53.4	(16.6, 172.3)	103.9	(31.6, 340.9)
Factor Deficiency						
IX	169	52.1	1.0**		1.0**	
VIII	691	65.0	1.7	(1.2, 2.4)	2.0	(1.3, 3.0)
Clotting Factor Level						
≥5%	181	30.4	1.0**		1.0**	
<5%	679	71.0	5.6	(3.9, 8.0)	9.7	(6.5, 14.6)
Treatment Center Type						
FFC	660	60.3	1.0**		1.0**	
SSC	200	69.5	1.5	(1.1, 2.1)	1.1	(0.7, 1.7)

*95% confidence interval.
**Reference group.

TABLE 3—AIDS Status of Hemophilia Patients by Patient Characteristics

Characteristics	Number HIV +	Percent AIDS	OR	(95% CI*)
Age Group (year)				
≤20	167	4.8	1.0**	
≥21	370	14.6	3.4	(1.6, 7.3)
Factor Deficiency				
IX	88	9.1	1.0**	
VIII	449	12.0	0.7	(0.3, 1.6)
Clotting Factor Level				
≥5%	55	10.9	1.0**	
<5%	482	11.6	0.9	(0.4, 2.3)

*95% confidence interval.
**Reference group.

known. The eight hemophilia programs with a lower testing rate were usually staffed by health care workers providing care to patients with a variety of hematologic and oncologic diseases, whereas the five centers with a higher testing rate were more hemophilia focused.[13]

In this retrospective study, the denominator of HIV seropositive persons shifted over part of this period, with the prevalence of infection increasing during 1981 through 1984. Although this might affect our results, the substantial differences observed suggest that these estimates, which cannot be better specified, are not likely to account for the observed differences. Also, as expected, the tested group consisted of patients that were older and/or with severe hemophilia, both of these factors would tend to bias the associations with HIV seropositivity toward nonsignificance. That is, the patients not tested were younger and probably at a lower risk of infection; therefore, if tested, the seropositivity rate of younger patients would be lower and the observed difference detected in age may be even greater than noted by our estimates. The analysis of the rate of AIDS was performed using AIDS cases from the defined centers as the numerator and patients with a positive HIV antibody status determined from those tested as the denominator. Thus, the age bias in HIV testing would only tend to underestimate the denominator for younger persons. The observed age difference of the rate of AIDS may thus be more dramatic than estimated herein.

The reason for the discrepancy in AIDS prevalence between age groups may be that younger hemophilic patients received less clotting factor than older hemophilic patients, that is, they were exposed to the HIV virus later than their older counterparts and have an "earlier" phase of the infection. Although it cannot be proved or disproved by this study, because specific dates of seroconversion are not available on these patients, this hypothesis is unlikely. The critical variable in hemophilia treatment may be the frequency of infusions and extent of exposure to different clotting factor concentrate lots, not age. Persons bleeding more frequently will be exposed to more lots of commercially prepared clotting factor concentrates and therefore be more likely to receive concentrates from a contaminated lot. Young, active hemophilic boys ages 3 or 4 years through adolescence are the most physically active and consequently have frequent hemorrhages requiring infusion therapy.[20] However, the rate of AIDS would be expected to be high through adolescence, not just for the 6- to 12-year olds age group. While exceptions certainly occur, the size of the currently reported population would result in a relatively homogeneous group. Further, the epidemiologic curves of AIDS diagnosis over time for those <21 and ≥21 years are similar. Consequently, reasons for the differences in the biological response to HIV infection between the two broad age groups should be sought among physiologic mechanisms. In addition, if age differences were representative of the true differences in the population, and age is an important cofactor in the development of AIDS, the important scientific and public health implications merit further investigation.

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The Infrastructure of Religious Communities: A Neglected Resource for Care of People with AIDS

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Abstract: The AIDS Interfaith Council of Houston, sponsored by the Foundation for Interfaith Research and Ministry, recruits, trains, and coordinates volunteers for home-based care for adults and children with AIDS (acquired immunodeficiency syndrome). The program provides compassionate, non-judgmental care for the sick and respite support for the primary care provider. Care is provided by highly motivated, trained volunteers. The respite care team model also represents a support system to complement medical management and other health care services for people with AIDS. With the expanding number of cases and the growing preference for clients to remain at home, such a model will become increasingly important as people continue to live longer with AIDS. (*Am J Public Health* 1990; 80:970–972.)

Introduction

With the advent of acquired immunodeficiency syndrome (AIDS), biomedical scientists, physicians, providers of social services, and social communities face a complex and serious challenge. Progress has been made in understanding the etiologic virus—human immunodeficiency virus (HIV)—its destructive effect on a human host, and palliative therapies for HIV infection. Improved medical management of associated illnesses has been developed. Given the psychosocial, emotional, medical, and economic burdens related to HIV disease, the importance of supportive services is clear.[1,2] Traditionally, in times of illness, religious communities have been an effective support system for individuals, their families, and friends. In the AIDS epidemic, these communities are challenged to respond to the sick and suffering with compassionate, non-judgmental support.

The Houston (Texas) Standard Metropolitan Statistical Area presently ranks fourth among cities in the United States for cumulative reported cases of AIDS.[3] As of January 2, 1990, the cumulative case report for people diagnosed with AIDS was 3,558, of whom 1,177 are living.* It is projected that the cumulative number of reported AIDS patients in Houston alive at the end of 1992 will range from 4,573 to 5,740.**

Relatives and friends of people with AIDS face the pressures of caring for people who are seriously ill as well as dealing with the economic and psychosocial concerns that accompany AIDS. Outpatient and home-based care are important for both economic and emotional reasons.[4,5] Along with the deterioration of their health and the fear of abandonment, patients and caretakers must cope with loss of employment and insurance. Also, an increasing number of people with AIDS have no insurance at the beginning of their illness. If the rate of progression and the preferences of people with AIDS for outpatient and in-home terminal care remain constant, the need for volunteers to provide in-home respite and supportive care will grow. In this article we describe an innovative service project which addresses the needs of people living with AIDS. A detailed description of the program, including the content and process of the educational program for the volunteers, is available from the authors.

The AIDS Interfaith Council

The AIDS Interfaith Council of Houston is an association of clergy and laity which provides educational and service programs in response to the expanding AIDS epidemic. A program sponsored by the Foundation for Interfaith Research and Ministry, the Council was created in January 1986 to offer congregation-based supportive care to people with AIDS and their loved ones. Over 650 volunteers organized in congregation-based teams offer friendship and respite from some of the time consuming tasks involved in caring for a person who has become seriously ill. The paid staff of the Foundation, which coordinates the activities of the Council, presently consists of 10 people who are responsible for administrative, supervisory, educational, training, service, resource development, fund raising, and counseling duties.

The AIDS Interfaith Council appears to be the oldest, largest, and most active congregation-based supportive program in the United States. Surgeon General C. Everett Koop noted during a speech to the Council in May 1988, "I think that what you've done here is tremendous . . . you're way ahead of the rest of the country. If it would be possible to take what you've done to other parts of the country, we would see networks such as you have here spring up all over the land."[6]

Program Description

The respite care teams are the heart of the program. Teams of volunteers from individual congregations are recruited and trained to provide social support, physical care, emotional support, and spiritual care to dependent, home-based patients and their loved ones. At the end of 1989, 28 Houston area churches and synagogues each provide a team of volunteers sponsored, trained, and coordinated by the AIDS Interfaith Council. No fees are charged for any services of the Council or the care teams.

Volunteers are recruited during educational programs in congregations at which facts regarding AIDS and risk reduction are given. Recruiters remind people who attend that they have a religiously grounded moral obligation to care for sick and dependent people regardless of the affliction or the means by which the dependency developed.[7] Education becomes an essential prerequisite to the recruitment of volunteer teams, since it is during these sessions that AIDS-related myths and fears are addressed.

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*Houston Department of Health and Human Services, Bureau of HIV Prevention Update, January 2, 1990.

**Harris County (Texas) Department of Health: The AIDS/HIV Forecast for Houston: A Report to the Long Range Planning Committee of the Greater Houston AIDS Alliance, November 1989.

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Once recruited, teams of at least 14 people receive three types of training for their services to people living with AIDS. One component focuses on organizational and operational matters. A second component is on the development of listening and care skills, including how to provide emotional support to sick and dying people. A third component deals with basic nursing and hospice care of people with AIDS. The three components require seven hours of orientation and training. The team chooses a team leader who assumes overall administrative responsibilities for scheduling, communication, and record-keeping. The teams schedule monthly meetings for discussion of patient progress, continuing education, peer support, and social support.

Teams then are assigned patients who are either self-referred or have been referred to the Council by physicians, social workers, discharge planners, and other service agencies. Ordinarily, paired team members meet with each patient at a frequency per week agreed upon by the client and caretaker and consistent with the person's needs. Visits usually last between three and four hours. As a client or caretaker desires, e.g., during a crisis period, additional team members are assigned. By the time a client progresses to a terminal state, services typically are provided seven times a week. If a client enters hospital for acute or terminal care, the team maintains contact until discharge to home or death. Team members know, as they accept responsibility for the care of a new client, that they may expect to share in his or her death.

This commitment enables the person with AIDS to retain a degree of autonomy and independence that otherwise would be difficult to maintain. Depending upon the person's needs and desires, the teams provide social and emotional support, accompany the client on shopping activities, and provide housekeeping assistance, transportation, basic nursing care, and other appropriate "hands-on" care. Care team members encourage clients to set their own agendas, thereby reinforcing their autonomy. Their readiness to help a client sift through options and explore possible actions, to "listen" as the person works through his or her emotional concerns, together with offers of practical aid, affirm the client's dignity.[8] Pastoral care and sacramental ministries are available upon request by the client. No spiritual or moral agenda is forwarded by any team member.

The focus of care and support is not restricted to the person with AIDS. Other people in the home (roommate, lover, family member) often welcome the respite and emotional support provided by team members. Primary care providers are supplemented, not replaced, by these teams. Strong bonds tend to be established over time. Survivors of deceased loved ones often become involved in this program in gratitude for the care received and to continue their commitment to people touched by AIDS.

We have learned that people in the advanced stages of HIV infection have in-home needs that tend to be too great for one or two people to provide. The availability of 14–20 people to supplement or to provide primary supportive care tends not only to improve the quality of life for a dependent client, but also appears to be a means for caring for people at home rather than in a higher cost institution. The presence of volunteers also allows people who so desire to die at home. Without this sort of support, many indigent and non-indigent people would spend their last weeks in hospital or nursing home (if the nursing home would accept a person with AIDS) against their wishes and at a higher expense to them or the community.

The nature of the activities and context in which teams are involved creates stress, grief, and bereavement which must be addressed to maintain the volunteers' emotional well-being and the quality of services. Supervision of team activities and group therapy for teams, therefore, are crucial. Each team meets with supervisory/therapeutic personnel (MSW or doctoral level professionals) approximately every 4–8 weeks. At these meetings, individual and team concerns are addressed. Guidance and intervention by supervisory or therapeutic personnel occurs as indicated. The volunteer commitment and congregational endorsement of the teams, plus careful supervision, appear to minimize volunteer turnover.

TABLE 1—Trends in Volunteer Services by Client Breakdown, AIDS Interfaith Council of Houston

	1986	1987	1988	1989	Total
Number of Teams (cumulative per year)	6	11	21	28	28
Clients (per year)					
New	66	100	147	173	486
Clients receiving team service	60	75	119	172	426
(Percentage increase)	—	(25)	(58)	(44)	—
Clients receiving financial aid or pastoral care	6	25	28	1	60
Terminated (death, lost to system)	49	92	126	134	401
Year end census	17	25	46	85	—
Cumulative (year end census plus new)	66	117	172	219	—
Average hours per client/per year	*	*	168.6	200.9	
Houston-Harris County AIDS Cases (new per year)	596	756**	935**	1127**	
(Percentage increase)	—	(26)	(23)	(20)	

*No record of client contact hours was kept in 1986 or 1987.
**Estimated number of reported new cases per year, Harris County Health Department, December, 1988.

Activity Statistics

The program has served 16 percent of the cumulative AIDS cases in the Houston SMSA since the Council began in 1986 (486 of 3,005 cases diagnosed). There has been an increase in the number of teams in each year of the program. The number of people with AIDS who have made use of the care teams also has grown every year since 1986 (Table 1). The growth in the provision of volunteer services clearly outdistances the rate of growth of the epidemic in Houston. The rate of increase in clients doubles the rate of increase of new diagnoses in Houston in 1988 and 1989 (Table 1). Over 73,000 contact hours of service were given in 1988–89. At a value of $8/hour for comparable home attendant care,*** volunteers provided $584,000 worth of services during the two years.

Discussion

The value of the program is clear. The program seems to be a logistically efficient means of providing home-based care for people touched by AIDS. By recruiting volunteers from religious communities, the program draws upon a ready pool of motivated people already oriented to visiting the sick. The team model provides peer and supervisory personnel to resolve volunteer and client problems.

The adult care team model has the capacity for replication in any neighborhood and is being duplicated in other cities (e.g., New Orleans, Baton Rouge, San Antonio, Little Rock, Albu-

***According to the Visiting Nurses' Association, the value of home attendant care in the Houston area is $8/hour.

querque, and Dallas-Ft. Worth).† Finally, given the widespread misunderstanding of HIV/AIDS in the general public, the program encourages a greater degree of knowledge and compassion for affected people. As more congregations become involved, a discrediting of stereotypes about people with AIDS occurs. The work of the Care Teams becomes a form of community education both within the general community about HIV/AIDS and for at-risk people about the perceived indifference to their condition by the general population.

The care team model has encountered several problems. First, since the medical knowledge and treatment of AIDS changes rapidly, there is a need for continuing education of volunteers. Volunteers are open to continuing education only if the programs are conducted in the setting of the team's regular monthly meeting. Traveling to 28 locations for continuing education sessions makes for inefficient use of staff time, but elicits a higher response from volunteers. Second, nearly all clients are followed to death. As volunteers become increasingly involved in the lives of their clients, the issues of grief and bereavement are very real. Therefore, both individual and group supervision of the volunteers are important aspects of the program. Therapy is made available at no charge to the volunteers who request it.

We have found if difficult to develop a volunteer profile. It is our impression that the teams generally reflect the demographic profile of their congregations. However, our experience has been that volunteers would interpret questions regarding their personal lives as intrusive and irrelevant to their service. Their priority clearly is patient care, no participation in record-keeping.

The 1989 rate of increase in new Council clients moderates an upward trend but continues to outpace the increase in new diagnoses. The clients on average are receiving care for a longer period of time, thus reducing the number of new clients a team can serve during the year (Table 1). The number of referrals and requests for service increased in 1989. All requests could not be met, however, because clients resided in areas in which presently there is little or no team coverage.

Therefore, a fourth area of concern is the resistance of congregation leadership to an identification with AIDS and those people affected by it. Resistance to educational programs among some religious groups ranges from specific concerns about condom use, drug use, and sexuality in general. Some religious leaders seem to fear that discussion of HIV/AIDS is equivalent to promoting of high-risk behaviors. The Council maintains that objective information about HIV/AIDS is a primary means for containing the disease. Combined with a sensitive concern for the moral and theological positions of the various communities, knowledge about HIV disease lessens personal anxieties and fears and helps build community support.

Fifth, as in the rest of the country, there is an increasing number of IVDUs (intravenous drug users) with AIDS in Houston. Studies show that minority populations are disproportionately represented in the AIDS epidemic.[9,10] This development highlights the need of involving Black and Hispanic churches in the Council's work. There has been some recent success in recruiting teams from several Black churches. Outreach to Hispanic groups has become a priority. Nevertheless, given the large number of Black and Hispanic congregations in Houston, response has been disappointing. Cultural factors such as the stigma of homosexuality play a role, as does the reluctance to link AIDS closely to minority communities. Until religious leadership in all areas of Houston recognize the need to respond to the AIDS crisis, the availability of congregation-based care team services to minorities will be limited.

Finally, with the increasing rate of HIV infection among female IVDUs or the sexual partners of IVDUs, the number of pediatric AIDS cases is increasing in Houston. Presently, the care teams are expanding their services to include children and their families. The needs of families with HIV seropositive children are different than those of adults.[11,12] Special features associated with these needs include infection in one or both parents, perhaps a single parent family setting, as well as the possible presence of siblings. Building upon experience gained in the last four years, the Care Team model has been revised to provide services to these families. Beginning in July 1989, the Foundation committed its resources to recruiting, training, and supervising Pediatric Care Teams. A four-hour training program oriented toward the features of pediatric AIDS was added to the adult training component. Eight of the 28 care teams are working with children who have AIDS. Since July 1989, these volunteers have extended care to 15 children, of which two have died.

The problems and concerns of men, women, and children living with AIDS are many and severe. As the numbers of people with HIV disease increase, the demand for medical and support services will continue to grow. One resource that can be developed and utilized to ease the psychosocial burdens of people with AIDS is the congregation-based care team model.

†Based upon the work of the AIDS Interfaith Council's Adult AIDS Care Team model, the staff of the Foundation participated in writing the proposal for the creation of a regional interfaith project including the cities of New Orleans, Baton Rouge, San Antonio, Little Rock, and Albuquerque. This proposal was funded by the Robert Wood Johnson Foundation in 1988. Dallas-Ft. Worth has followed a similar course with funding from Robert Wood Johnson Foundation.

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Maintenance of Safer Sexual Behaviors and Predictors of Risky Sex: The San Francisco Men's Health Study

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Abstract: This paper describes the sexual behavior changes made by 686 gay and bisexual men in San Francisco between 1984 and 1988, focusing on the individual maintenance of this behavior change over time. There were drastic reductions in insertive and receptive unprotective anal intercourse over time and the vast majority of subjects were able to maintain these changes for at least 12 months prior to the last interview. A total of 12 percent of participants admitted to relapsing to unprotected receptive anal intercourse following initial behavior change; 10 percent reported engaging in unprotected receptive anal sex during every year of the study period. Men were more likely to practice unprotected anal intercourse in 1988 if at baseline they were younger, practiced unprotected anal intercourse, reported more sex partners, did not have a close friend or lover with AIDS, and engaged in fewer other health-related behaviors. (*Am J Public Health* 1990; 80:973–977.)

Introduction

This study seeks to document the stability of change and psychosocial predictors of unprotected anal intercourse in a probability sample of San Francisco gay and bisexual men. Gay men account for 95.7 percent of all AIDS (acquired immunodeficiency syndrome) cases in San Francisco to date[1] and 68 percent of the cases in the United States to date.[2] San Francisco gay and bisexual men have been particularly hard hit by this epidemic with infection rates estimated at 52 percent.[3]

Earlier studies [3–5, *, **] have documented drastic sexual behavior changes in volunteer samples of gay and bisexual men in San Francisco. Although these findings are encouraging, it is crucial that the changes are maintained over time. There have been few studies examining the long term stability of this behavior change. Joseph, Montgomery, Kessler, *et al*,*** reported that between 25 and 35 percent of their sample of Chicago gay men resumed high-risk behavior at least once following initial risk reduction. In Boston, 13 percent of the gay men who reported practicing safe sex in year one relapsed during the following year.† The present study examines both the prevalence and decline of high-risk behavior among all gay and bisexual subjects as well as the stability of this change and the relation between high-risk behavior and demographic and psychosocial variables, HIV (human immunodeficiency virus) antibody status, HIV-related symptoms, and other health-related behaviors.

Methods

Subjects

The San Francisco Men's Health Study is a prospective study of single, predominately White, well-educated males between the ages of 25 and 54 recruited by multistage probability sampling from the six kilometer square area in San Francisco with the highest cumulative incidence of AIDS in 1984. Sampling methods, as well as statistical, and data collection procedures have been described previously.[3,5,6]

The analyses in the present study include data on 686 gay and bisexual men who participated in the first year (July 1984–June 1985) of data collection, 633 who participated in the second year (July 1985–June 1986), 627 from year three (July 1986–June 1987), and 592 from year four (July 1987–June 1988). At least 57 subjects died between July 1984 and June 1988 and an additional 25 subjects had died from 1988 to 1989.

Demographic subject characteristics for this subsample, obtained in July 1984, show that this area is populated mostly by White, employed, and well-educated gay males.

Analysis of Attrition Bias

We compared those subjects who remained in the study to those who dropped out between 1984 and 1988. The scores of these two groups on all predictor and outcome variables in year one did not differ appreciably with regard to sexual behaviors (Table 1). Dropout subjects were more likely to be HIV positive in year one, and to report HIV-related symptoms at that time; they were more depressed, drank less alcohol, and thought of themselves as gay at an earlier age than subjects who remained in the study. These differences probably reflect the differing percentages of HIV positive subjects in the two groups.

Measures

Sexual Behavior—Self-report data on high-risk sexual behavior were obtained eight times from July 1984 to June 1988. Subjects were asked whether they had engaged in any sexual activity during the previous six months. Those who answered "yes" were then asked whether they had engaged in any of 22 specific behaviors. For each of these activities, the subjects were asked to report the number of partners with whom they had engaged in that behavior since the last clinic

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*Bye L, Capell F, Anderson R, *et al:* Prevalence of AIDS risk factors in the population of the State of California. Presented at the IV International Conference on AIDS, Stockholm, 1988.

**Doll L, Damon W, Jaffee H, *et al:* Self-reported changes in sexual behaviors in gay and bisexual men from the San Francisco City Cohort. Presented at the III International Conference on AIDS, Washington, DC, 1987.

***Joseph J, Montgomery S, Kessler R, *et al:* Determinants of high risk behavior and recidivism among gay men. Paper presented at the IV International Conference on AIDS, Stockholm, June 1988.

†Saltzman S, Stoddard A, McCusker J, Mayer K: Factors associated with recurrence of unsafe sex practices in a cohort of gay men previously engaging in 'safer sex'. Poster presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

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TABLE 1—Comparison of Panel Members and Dropout Subjects at Year One (1984–85)

Variables	Stayed (n = 592)	Dropped (n = 94)	(95% CI of differences)
		1. proportions	
Any unprotected anal intercourse	.78	.76	(−.10,.06)
Oral sex	.95	.94	(−.06,.04)
Oral-anal contact	.37	.46	(−.03,.19)
Anonymous partners	.50	.54	(−.07,.15)
Multiple partners	.82	.74	(−.16,.02)
Smoked regularly	.37	.47	(−.01,.21)
Antibody positive	.48	.72	(.14,.34)
HIV-related symptoms	.40	.58	(.07,.29)
Friend, lover with AIDS	.40	.50	(−.02,.20)
		2. means	
Age (years)	36.5	36.5	(−1.5,1.4)
Age (years) at which first thought of self as gay	18.9	17.4	(−3.0,−.1)
Age (years) at which first socialized as gay	21.4	20.6	(−1.7,.2)
Alcohol consumption (mean)	4.3	3.8	(−.8,−.1)
Loneliness (mean)	4.3	4.0	(−.1,.7)
Depression (mean)	8.7	11.1	(.5,4.3)
Amount of social support (mean)	81.2	89.9	(−1.0,.6)
Social support satisfaction (mean)	32.1	32.3	(−7.4,24.6)

visit. Although unprotected insertive and receptive anal intercourse and multiple sexual partners are the only firmly established major risk factors for seroconversion in male-to-male relationships,[7] we also examined the prevalence of anal-oral contact, genital-oral contact, fist-anal contact, and whether the respondent had engaged in sex with anonymous partners. To facilitate presentation, we combined these eight six-month segments into four one-year intervals. Since all of the sexual behaviors have been dichotomized, an affirmative response during either six-month segment in a given year resulted in an individual being classified as having engaged in the behavior during that year.

To examine correlates of risky sex, two multiple logistic analyses were conducted, predicting unprotected receptive and insertive anal intercourse in waves two (January to June 1985) and eight (January to June 1988). The predictor variables in these analyses (Tables 2 and 3) also included items developed for the psychosocial component of the project and were administered to the subjects in wave 2 (January to June 1985); they are defined in detail in the Appendix.

Procedure

We examined yearly prevalence rates of sexual behaviors for the entire gay and bisexual sample. Group changes in sexual behaviors from 1984 to 1988 were then analyzed using McNemar tests.[8]

We also examined fluctuations in individual behavior patterns. In order to do this, the study period was divided into four 12-month segments consisting of: 1) July 1984 to July 1985; 2) July 1985 to July 1986; 3) July 1986 to July 1987; and 4) July 1987 to July 1988. A subject was considered "high risk" for a given period if he reported engaging in any unprotected receptive or insertive anal intercourse with or without ejaculation during that time. This yielded 16 behavior patterns which were combined into four groups; stable low risk, stable high risk, change from high to low risk, and relapse.

The relation between the predictor variables and unprotected anal sex was examined using multiple logistic regression.[9] A dichotomous score was created classifying subjects as high risk if they reported having engaged in either insertive or receptive unprotected anal intercourse during January to June 1985 and 1988.

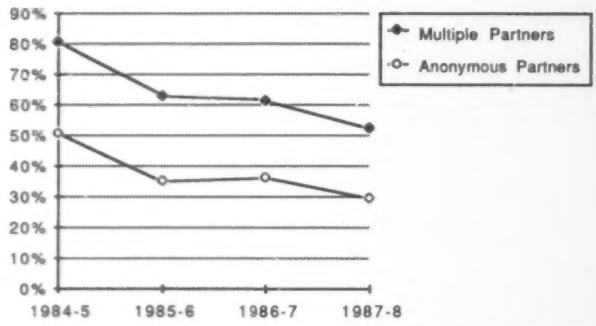

FIGURE 2—Percent Men Who Reported Having Sex with Multiple and Anonymous Partners Each Year

Results

Behavior Change

Figures 1, 2, and 3 present data showing the percent of men who reported engaging in each of the sexual behaviors in years one through four (i.e., 1984–5, 1985–6, 1986–7, and 1987–8). The most dramatic changes have occurred in unprotected insertive (37 percent to 2 percent) and receptive (34 percent to 4 percent) anal intercourse with ejaculation. Significant changes were also reported in insertive and

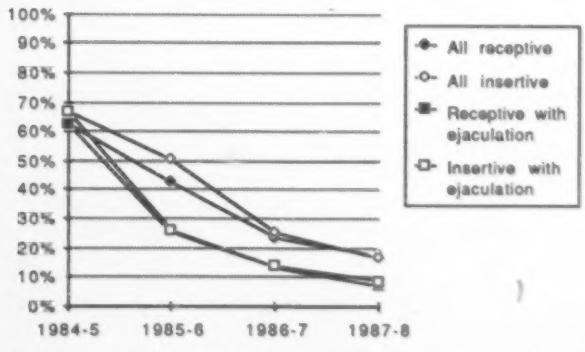

FIGURE 1—Percent Men Who Reported Engaging in Unprotected Anal Intercourse Each Year

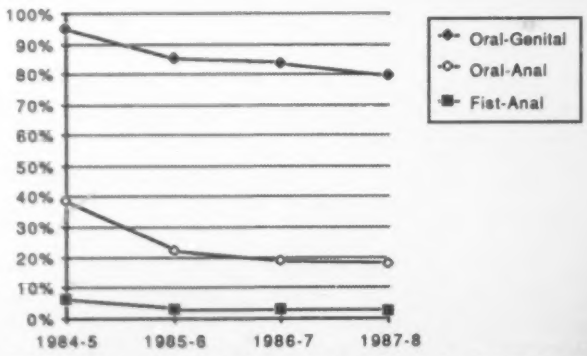

FIGURE 3—Percent Men Who Reported Engaging in Oral-genital, Oral-anal, and Fist-anal Sex Each Year

TABLE 2—Cross-sectional Predictors of Unprotected Anal Intercourse in 1985

Predictors	Adjusted Odds Ratio	(95% CI)
Age (20 year difference)	0.6	(0.5,0.8)
Age at which first thought of self as gay	1.0	(0.9,1.2)
Age at which first socialized as gay	1.1	(0.8,1.3)
Number of sex partners	1.4	(1.2,1.6)
Antibody status (positive)	1.3	(1.1,1.5)
HIV-related symptoms	1.1	(0.9,1.3)
Smoking	1.1	(0.9,1.3)
Alcohol consumption	1.3	(1.1,1.6)
Friends, lover with AIDS	0.9	(0.8,1.1)
Loneliness	1.0	(0.9,1.1)
Amount of social support	1.0	(1.0,1.0)
Social support satisfaction	1.0	(0.9,1.2)
Depression	0.9	(0.8,1.0)

receptive anal practices with and without ejaculation, which decreased from 42 percent and 63 percent to 17 and 17 percent respectively. All behaviors changed significantly from year one to year two. After June 1986, most behavior changes leveled off. Unprotected insertive and receptive anal intercourse and insertive oral-genital contact continued to decrease significantly from 1985–6 to 1986–7. The percent of subjects reporting unprotected insertive and receptive anal sex, insertive and receptive oral-genital contact, sex with anonymous partners, and multiple sexual partners continued to decline until 1987–8.

Maintenance of Behavior Change

Behaviors were remarkably stable: 25 percent and 31 percent of the subjects were classified as stable low risk, and an additional 50 percent and 47 percent fell into the change to low risk category. Only 16 percent and 12 percent of the subjects admitted to relapsing to unprotected insertive and receptive anal sex, respectively, while an additional 10 percent were stable high risk.

Younger men were more likely to engage in unprotected anal intercourse than older men in the sample in both 1985 and 1988. Subjects who reported in 1985 that they had friends or lovers with AIDS were less likely to engage in unprotected anal sex in 1988 than were men who did not have this experience (Table 3).

TABLE 3—Longitudinal Predictors of Unprotected Anal Intercourse in 1988

Predictors	Adjusted Odds Ratio	(95% CI)
Age (20 year difference)	0.6	(0.3,0.9)
Baseline sexual behavior	3.4	(2.1,5.4)
Age at which first thought of self as gay	1.0	(0.8,1.4)
Age at which first socialized as gay	1.0	(0.7,1.6)
Number of sex partners	1.2	(1.1,1.4)
HIV positive, knowing results	0.7	(0.5,1.1)
HIV negative, knowing results	0.9	(0.6,1.3)
HIV positive, not knowing results	0.7	(0.4,1.1)
HIV-related symptoms	0.9	(0.7,1.2)
Smoking	1.3	(1.0,1.6)
Alcohol	0.8	(0.6,1.2)
Friend, lover with AIDS	0.8	(0.6,1.0)
Loneliness	1.1	(0.8,1.2)
Amount of social support	1.0	(1.0,1.0)
Social support satisfaction	1.1	(0.9,1.4)
Depression	1.1	(0.9,1.3)

See appendix for a description of measures

Men who reported engaging in unprotected anal intercourse in 1985 were likely to report doing so in 1988 than were men who had been practicing safer sex only in 1985.

Participants who were HIV positive in 1985 were more likely to engage in unprotected anal sex at that time. However, knowledge of HIV antibody status in 1987 did not predict sexual behavior in 1988. The presence of HIV-related symptoms did not predict report of high-risk behavior.

Men who reported a greater number of partners in 1985 were far more likely to practice unprotected anal sex in 1988 than were men who had been practicing safer sex only in 1985. Subjects who reported consuming a greater volume of alcohol were more likely to report engaging in high-risk sexual behavior than those who reported less alcohol consumption. This relation held for the cross-sectional analysis in 1985, but did not predict risk behavior longitudinally. Subjects who reported smoking regularly in 1985 were somewhat more likely to engage in unprotected anal sex in 1988.

Men who reported greater levels of depression in 1985 were less likely to report having engaged in unprotected anal sex in that year than subjects who reported fewer depressive symptoms. Participants who reported having a large number of people on whom they could rely for social support were less likely to report practicing risky sex. Loneliness and satisfaction with social support were unrelated to sexual behaviors.

Discussion

The results on individual and group behavior change show that San Francisco gay men have greatly reduced their levels of almost all high-risk sexual behaviors from 1985 to 1988. The differences between all anal sex and anal sex with ejaculation may be due to a misconception that anal sex is "safe" as long as one withdraws before ejaculating.†† This fits with our finding that there has been very little change in oral sex practices, which is another behavior considered "safe" by many in the San Francisco gay community.

Approximately three-fourths of the subjects reported that they were in either the stable low or change to low categories. The data further show that occasional relapse is now more prevalent than stable high-risk practices in this community. Prevention efforts need to shift from focusing on the initial adoption of safe sex, to a relapse prevention format, emphasizing the long-term maintenance of safer sex.

Overall, these changes may represent the most profound behavior changes ever observed in the literature on health behavior change.[10] Studies on smoking cessation, alcohol abstinence and dieting show that it is extremely difficult to maintain healthy behaviors over time. The results on prevalence replicate findings from other San Francisco studies using volunteer populations,[11,12] thus providing a consistent picture of gay sexual behavior in San Francisco.

Although results from similar studies in other parts of the country also show a reduction in high-risk behavior in this population, the degree of stability of change observed in this study may be unique to this cohort or to San Francisco. Although this is good news, it is important to place it in context. It has been estimated that more than half of the

††Ekstrand ML, Stall RD, Coates TJ, McKusick L, Morin S: Risky sex relapse: The next challenge for AIDS prevention: The AIDS Behavioral Research Project. Paper presented at the V International Conference on AIDS, Montreal, Canada, 1989.

mainstream gay community in San Francisco has been infected by HIV; any instance of risky sex with a partner of unknown serostatus thus carries an unacceptable risk. It is important to identify characteristics of relapsers and to obtain a better understanding of the reasons for relapse in order to design effective prevention programs focusing on both the acquisition and maintenance of safe sex in this population.

The most consistent predictors of high-risk behavior included demographic and behavioral variables. Younger men were more likely to engage in unprotected anal sex and those who had friends or lovers with AIDS in 1985 were less likely to practice risky sex in 1988. These findings have been replicated both in other samples of gay and bisexual men[12–14] and among heterosexual men.††† The finding that younger gay men seem to be having more difficulty changing their behaviors could be due to a variety of factors, such as greater impulsivity, having less social support for safe sex practices, feeling uncomfortable obtaining or using condoms, perceptions of invulnerability, or having poor "safe sex negotiating skills." Results from a recent study of HIV risk-taking among young gay men[14] showed that those who practiced unprotected anal intercourse reported enjoying it more, perceiving it as less risky, and had poorer communication skills than those men who did not engage in this practice. The finding that men who practice risky sex report having less social support might be related to this issue, since it is possible that younger gay men may be less involved in the network of gay organizations than are older gay men in San Francisco. This may pose a challenge to future campaigns, since it would imply that the men who need to be targeted may be the hardest to reach.

Past sexual behavior appears to be a strong predictor of future sexual behavior. However, by targeting only those who report current unsafe behaviors we run the risk of missing many men at risk for future unsafe sex, since relapse is even more common than stable high-risk practices in this population. We may thus have to develop different targeting and intervention strategies for men who relapse than for men who have never adopted safer sexual practices.

The finding that antibody positive subjects were more likely to practice unprotected anal sex in 1985 may be due to a variety of factors. This was the year the HIV antibody test first became available and it is therefore possible that the subjects had not received the results of their antibody test when engaging in risky sexual practices. It is also possible that some of these people only engaged in unprotected anal sex with other seropositive partners. However, we have no data on partner serostatus. A recent study of the impact of antibody testing on sexual behaviors in this cohort showed that knowledge of being seropositive was associated with a lower prevalence of insertive anal intercourse in 1987.‡ No differences were found in the prevalence of receptive anal intercourse, suggesting that seropositive men perceive insertive practices as the most likely way of infecting others.‡ This distinction was not made in the present study and may explain why knowledge of serostatus failed to predict overall rates of unprotected anal intercourse. In addition, the Coates, *et al*, study followed subjects until 1987, while the present study examined behavior in 1988.

In summary, the data from this study suggest that we need to target younger gay men, and possibly men who are less involved in the social networks of the gay community. The prevailing norm needs to be that any unprotected anal sex, including occasional relapse, is risky and thus unacceptable.

APPENDIX

Selected Measures

- *HIV Status*

Although HIV status has been measured at each wave of this study, only subjects who have requested knowledge of their results have received them. In wave six (January to June 1987) we asked subjects whether they had received these results and, if so, from whom they had received them. The results showed that 33.1 percent and 31.4 percent knew that they were antibody positive and negative, respectively. An additional 17.3 percent and 18.1 percent were antibody positive and negative, but denied having received their test results. In our 1985 cross-sectional analyses, we were thus only able to use HIV status alone as a predictor of sexual behavior. In the longitudinal analyses, we combined actual HIV status with reported knowledge into three predictor variables (HIV positive, knowing results; HIV negative, knowing results; and HIV positive, not knowing results). For each of the three comparisons, the target group was compared to all other subjects.

- *HIV-Related Symptoms*

We used a clinical index that was developed in a previous study of this sample and which was found to predict HIV infection, diminished skin test reactivity, and reduction in Leu 3a T cells.[16] It includes self-report of the following symptoms: new rash, diarrhea, night sweats, weight loss, persistent fever, herpes, shingles, or bullous impetigo. In addition, it was noted whether the subjects had been diagnosed with oral hairy leukoplakia or oral candidiasis during their physical examination. A subject was considered to have HIV-related symptoms if he had any two or more of these symptoms or infections.

- *Health-Related Behaviors*

Quantity and frequency of alcohol consumption were measured during the interview by asking subjects both how often they had consumed an alcoholic drink during the past six months, and how many drinks they typically consumed on these occasions. Amount was scored on a 4-point scale ranging from "1 or 2 drinks" to "7 or more drinks," while frequency was measured on a 9-point scale, ranging from "every day" to "not at all." Information on amount and frequency of alcohol consumption was then combined into a single 7-point scale, where 1 = abstinent, 2 = occasional-light, 3 = occasional-heavy, 4 = infrequent-light, 5 = infrequent-heavy, 6 = frequent-light, and 7 = frequent-heavy consumption.

Cigarette smoking was assessed by asking subjects if they had been smoking during the past month (scored dichotomously).

- *Number of Sexual Partners*

Number of male sexual partners during the previous six months was used as a continuous measure to examine whether men who engage in unprotected anal sex are more likely to have fewer or more sex partners than men who do not engage in this practice.

- *Psychosocial Measures*

Loneliness was measured using the short form of the Revised UCLA Loneliness Scale.[17] This includes those four scale items, which, using optimal subset regression techniques, best predicted scores in the self-labeling loneliness index. (Chronbach's alpha = .75.)

Depression was assessed with the Center of Epidemiological Studies Depression Scale (CESD). The CESD[18] was developed for use in studies examining the epidemiology of depression in general populations. The scale has high reliability (.77–.92) and correlates highly with other measures of depression (.55–.74).

Social Support—The instrument contains 12 questions (four tangible support items, four emotional support items, and four informational support items). Structural support was expressed in the number of people providing each type of support. Perceived support is the tabulation of satisfaction ratings of that support. Satisfaction scores range from 3 (very satisfied) to 0 (not at all satisfied) for each type of support. The present study used an instrument partly derived from the Social Support Questionnaire by Sarason, *et al*,[19] chosen because of its high stability and internal consistency. The framework used to separate the items into distinct categories of emotional, tangible, and informational support comes from the research on the health-related functions of social support provided by Schaefer, Coyne, and Lazarus.[20]

†††Guydish J, Coates T, Ekstrand M: Changes in AIDS-related high-risk behavior among heterosexual men. Presented at the IV International Conference on AIDS, Stockholm, Sweden 1988.

‡Coates T, Ekstrand M, Kegeles S, Stall R: Knowledge of HIV antibody status, behavior change, and psychological distress in two cohorts of gay men in San Francisco. Manuscript under review.

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NIH Consensus Panel Issues Report on Sunlight and UVR

The National Institutes of Health convened a Consensus Development Conference on Sunlight, Ultraviolet Radiation, and the Skin last year, and has now issued a report containing conclusions and recommendations concerning the effects of sunlight and UVR on the skin. The report was prepared by a panel of experts who considered scientific evidence presented at the conference. A two-page summary of the document is also available. To obtain a free copy of the consensus statement on the effects of sunlight and UVR on the skin, contact:

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Longitudinal Predictors of Reductions in Unprotected Anal Intercourse among Gay Men in San Francisco: The AIDS Behavioral Research Project

LEON MCKUSICK, PHD, THOMAS J. COATES, PHD, STEPHEN F. MORIN, PHD, LANCE POLLACK, MA, AND COLLEEN HOFF

Abstract: Predictors of unprotected anal intercourse were examined among 508 gay men in San Francisco. The cohort was recruited in 1983–84 at which time 49.8 percent of non-monogamous men (N = 435) and 71.2 percent of monogamous men (N = 73) reported practicing unprotected anal intercourse. Only 12 percent of non-monogamous and 27.4 percent of monogamous men reported these practices in 1988. The non-monogamous men who practiced unprotected anal intercourse in 1984 were more likely to be younger, to report that unprotected anal intercourse was their favorite sexual activity, to be low in perceived efficacy to change sexual behavior, to report that friends were more likely to engage in high-risk behaviors, to have less knowledge of health guidelines, and to be less depressed at that time. Non-monogamous individuals who in 1984 reported that unprotected anal intercourse was their favorite sexual activity were more likely to practice that behavior in 1988. Those who knew their serostatus as positive were less likely to report unprotected anal intercourse in 1988. These data infer that in order to modify AIDS-related high-risk behaviors, community risk-reduction programs be differentially aimed at young persons so as to increase personal efficacy about risk reduction, challenge peer norms, promote antibody testing, and eroticize safer sexual activities. (*Am J Public Health* 1990; 80:978–983.)

Introduction

The incidence of new human immunodeficiency virus (HIV) infection, spread through male to male sexual contact, has recently been reported to be less than 1 percent per year in San Francisco.[1] This estimated low incidence rate is presumed to be due to behavior change rather than saturation of infection in the population. High-risk sexual behaviors likely to spread the virus have declined dramatically among gay men in many American urban settings.[2] Among the men in the San Francisco Men's Health Study, 44 percent reported unprotected anal intercourse receptive in 1985; this declined to 3 percent in 1988.[a] Rates of sexually transmitted diseases provide corroborative evidence of behavior change. In 1982, 13 percent of gay and bisexual men seroconverted, and there were 4,008 cases of gonococcal proctitis (GCP). In 1986, the HIV incidence among gay and bisexual men was 1 percent and there were 390 cases of GCP.[b]

Opinions vary regarding what influenced the massive changes in behavior observed in this population.[3–9] Some have drawn a close association between changes in behavior and the public education campaigns conducted in a coordinated fashion by agencies such as the San Francisco Health Department and the San Francisco AIDS Foundation.[3,4] Others have speculated that the observable presence of disease and death from HIV was responsible for individual modifications of behavior.[2,8,9] Few data have been presented to date about predictive associations between social or psychological variables and reductions in AIDS (acquired immunodeficiency syndrome) risk behaviors.[4,8] In earlier studies, we found that the number of sexual partners the previous month was positively correlated with use of sex to relieve tension and agreement that sexual freedom was linked to gay identity. Number of sexual partners was negatively correlated with awareness of the visual image of AIDS deterioration.[10] In a follow-up analysis predicting change from November 1983 to May 1984, four variables distinguished those retaining high numbers of sexual partners from those lowering the number: awareness of the visual image of AIDS deterioration, greater age, being in a primary relationship, and greater length of time since the first homosexual experience.[11]

Based on these preliminary findings, several measures were added to the 1984 and subsequent annual assessments to predict changes in high-risk behavior: measures of health belief adapted to HIV sexual behavioral risk reduction, standard measures of psychological distress, health-related variables, and measures of perceived norms for health risk activity. When the HIV antibody test was introduced, respondents were also asked whether they had been tested. Our intention was to establish a universe of possible psychological, social, and behavioral factors which may influence sexual behavioral risk for HIV transmission,[9] and to determine which of these factors predicted change over time.

Methods

Subjects and Procedures

Subjects for the AIDS Behavioral Research Project were recruited initially in 1983 and 1984 at bath houses and bars,and by advertising for individuals who were in committed relationships or who did not use bars or baths. A total of 754 men were enrolled in the sample in 1984, representing 51 percent of those approached to participate. Subjects were mailed a self-administered questionnaire each November, and asked to complete the questionnaire and return it by mail to the investigators. As of wave 5 of data collection (November 1988), 71 percent (n = 575) of the original cohort had responded to the survey. Another 8 percent (n = 61) are known to have died (either through the report of their friends or through matching in the California Death Registry). Data reported here are for 508 (67.4 percent of the original sample) men who returned every questionnaire between 1984 and 1988. Of the 508 men, 435 (57.7 percent) were non-monoga-

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Editor's Note: See also related paper p 963 this issue.

[a] Ekstrand M, Coates TJ: Prevalence and change in AIDS high risk behavior among gay and bisexual men. Stockholm: Paper presented at the IV International Conference on AIDS, 1988.

[b] Evans PE, Rutherford GW, Amory JW, *et al:* Does health education work? Stockholm: Paper presented at the IV International Conference on AIDS, 1988.

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mous and 73 (9.7 percent) reported that they were mutually monogamous with the same person over the entire assessment period, 1984 to 1988. Age of respondents in 1984 ranged from 19 to 63 with a mean of 35.7 (S.D. = 8.37). The majority of the sample in 1984 held professional or white collar occupations (77 percent), were Caucasian (91 percent), and had attended some college (68 percent). Mean annual income was $24,000. The study has been approved annually by the Institutional Review Board of the University of California San Francisco; a Certificate of Confidentiality was also obtained from the National Institute of Mental Health to further protect the respondents.

Measures

Sexual Behavior—Respondents were asked to report whether or not they had engaged in each of 22 sexual behaviors in the previous 30 days separately for primary and secondary partners.

Preference for Unprotected Anal Intercourse—Respondents rank ordered preferences for various sexual behaviors. A subject was scored as preferring this activity if he gave it a ranking of 1 (reported by 59.9 percent or 257 of non-monogamous and 74 percent or 54 of monogamous men). *Lifetime diagnosis of a sexually transmitted disease (STD)* was scored as no or yes (37.5 percent or 148 of non-monogamous and 29.6 percent or 21 of monogamous men in the sample).

AIDS Antibody Testing Status—Respondents were asked if they had obtained AIDS antibody testing and knew their test results. Testing status as of 1988 was used for this analysis: 31.9 percent (N = 136) of the non-monogamous men and 22.9 percent (N = 16) of the monogamous men were seropositive, while 41.3 percent (N = 176) of the non-monogamous men and 35.7 percent (N = 25) of the monogamous men were seronegative. The remainder (31 percent) were either not tested or declined to answer the question.

AIDS Prodromal Symptoms—All study respondents were presented with a checklist of 11 possible symptoms of HIV infection and asked to indicate which they had experienced in the last year. The list included the following: new rash, diarrhea, night sweats, weight loss, persistent fever, herpes, shingles, bullous impetigo, fatigue, oral hairy leukoplakia, or oral candidiasis. This was coded as a dichotomous variable: zero or one symptom indicated no symptoms and two or more indicated the presence of symptoms. In 1984, 15 percent (N = 76) of non-monogamous and 16.4 percent (N = 12) of the monogamous men reported one or more AIDS prodromal symptoms.

AIDS Loss—Participants were asked in 1984 to report how many of their friends and acquaintances either currently carried an AIDS diagnosis or had died of it; 36.3 percent (N = 155) of the non-monogamous and 35.2 percent (N = 25) of the monogamous men reported two or more such individuals.

Health belief variables included the following: *Belief in AIDS Health Guidelines* assessed the respondent's agreement to eight items on a six-point scale with AIDS risk reduction guidelines (Cronbach's alpha = .70, range = 8 to 48, median = 43); *Personal Efficacy* assessed the respondent's perceptions to 13 items on a six-point scale of being able to control risk for HIV infection and perceived self control in high risk sexual situations (Cronbach's alpha = .85, range = 21 to 78, median = 56); *Perceived Susceptibility to AIDS* assessed individual's belief to six items on a six-point scale that they were personally susceptible to contracting AIDS (Cronbach's alpha = .72, range = 6 to 36, median = 25).

Depression (measured by the Brief Symptom Inventory, expressed on a five-point scale using mean scores on six items, range = .17 to 3.67, median = .50).

Two separate scales were used to assess social support for risk reduction. *Social Support-Health Promotion* asked subjects to report how many of their friends engaged in four health-promoting behaviors (Cronbach's alpha = .68, range = 2 to 16, median = 11.0); *Social Support-High Risk Behavior* measured how many friends engaged in each of six potentially health-impairing behaviors (Cronbach's alpha = .72, range = 1 to 21, median = 10).

Statistical Analyses

We chose unprotected anal intercourse (receptive or insertive) as the primary dependent variable of interest because of its proven relationship with seroconversion.[1,12–14,c] Subjects were categorized as having practiced this behavior if they reported one or more episodes of anal intercourse without a condom in the previous month with either a primary or a secondary partner. Bivariate cross-sectional relationships were examined between predictor variables and occurrence of the dependent variables using 2 × 2 contingency tables, from which were calculated odds ratios and 95% confidence intervals using SPSS-X, Version 3.1. All independent variables were scored dichotomously; continuous scale scores were divided at the median. No differences in outcomes were produced with the scales used continuously or dichotomously. Bivariate associations whose 95% CI did not include one were then entered into multiple logistic regressions to calculate adjusted odds ratios with 95% CI; these were calculated using BMDPLR, Version 1987. A logistic regression model using all variables was also calculated. This resulted in a smaller N (308 for cross-sectional analyses; 146 for longitudinal analyses for those at high risk in 1984, 152 for those not at high risk in 1984). We report here the results from both the limited and full logistic models, since the former includes more subjects and the latter may bias variable analyses less.[15] Predictor analyses were completed separately for non-monogamous men who practiced unprotected anal intercourse in 1984 (N = 212) and for those non-monogamous men who did not practice unprotected anal intercourse in 1984 (N = 214).

Analysis of Attrition Bias—Subjects in this analysis did not differ significantly from deceased subjects or those lost to follow-up on any of the sexual behavior variables reported in this paper. Deceased subjects were older (62.1 percent > 36 years; compared to 46.5 percent of the sample and 39.5 percent of lost), more likely to have been diagnosed with a sexually transmitted disease (57.7 vs 36.3 percent of the sample and 40.3 percent of lost), more likely to believe that they had been exposed to HIV (77.2 vs 58.5 percent of the sample and 59.3 percent of lost), more likely to report prodromal symptoms (31.7 vs 15 percent of the sample and 17.1 percent of lost), and more likely to perceive themselves as susceptible to AIDS (61.8 percent above the median vs 46 percent of the sample and 45.4 percent of lost).

Results

AIDS-Related Risk Behavior: 1984–1988—Figure 1 presents the percent of non-monogamous men reporting various

[c] Hessol N, Rutherford G, Lifson A, *et al:* The natural history of human immunodeficiency virus infection in a cohort of homosexual and bisexual men. Stockholm: Paper presented at the IV International Meeting on AIDS, 1988.

sexual practices in the previous month for all five assessments.

Evident in the graph is the dramatic reduction in unprotected anal intercourse (from 49.8 percent in 1984 to 12 percent in 1988), and the increases in protected anal intercourse (from 15.2 percent in 1984 to 23.8 percent in 1988) and celibacy (from 13.3 percent in 1984 to 18.6 percent in 1988). Figure 2 presents the percent of monogamous men reporting various sexual practices in the previous month for all five assessments.

Monogamous men were more likely to practice unprotected anal intercourse in 1984 (odds ratio = 2.44, 95% CI = 1.44, 4.12) and in 1988 (odds ratio = 2.75, 95% CI = 1.52, 4.97) and were less likely to practice protected anal intercourse in 1984 (odds ratio = 0.41, 95% CI = 0.16, 1.08) and in 1988 (odds ratio = 0.44, 95% CI = 0.21, 0.94). The non-monogamous men were more likely to be celibate in 1984 (odds ratio = 0.18, 95% CI = 0.04, 0.77) but not in 1988 (odds ratio = 0.77, 95% CI = 0.39, 1.53). Monogamous men, compared to non-monogamous men, were more likely to report in 1984 that unprotected anal intercourse was their favorite sexual activity (74 vs 59.9 percent, odds ratio = 1.9, 95% CI = 1.08, 3.32), to be above the median in personal efficacy (68 percent above the median vs 46 percent, odds ratio = 2.49, 95% CI = 1.43, 4.34) and less likely to believe that they were exposed to HIV (40.3 vs 61.5 percent, odds ratio = 0.42, 95% CI = 0.24, 0.72). Monogamous men were less apt to be depressed (21. 9 percent above the median vs 47.2 percent, odds ratio = 0.31, 95% CI = 0.17, 0.56), or to believe that they were susceptible to AIDS (31.4 percent above the median vs 48.5 percent, odds ratio = 0.49, 95% CI = 0.28, 0.84).

Cross-Sectional Predictors of Unprotected Anal Intercourse: 1984—Table 1 presents the bivariate relationships between each of the predictor variables and the practice of unprotected anal intercourse for the non-monogamous men. Adjusted odds ratios from the full regressions are also presented. Those who were younger, who reported that unprotected anal intercourse was their favorite sexual activity, who were lower in personal efficacy, and who were less depressed were more likely to practice unprotected anal intercourse. The limited multivariate analysis revealed the following variables to be related to the practice of unprotected anal intercourse in 1984: younger age (adjusted odds ratio = 0.54, 95% CI = 0.34, 0.87), unprotected anal intercourse as a favorite sexual activity (adjusted odds ratio = 6.92, 95% CI = 3.45, 9.18), and personal efficacy (adjusted odds ratio = 0.41, 95% CI = 0.26, 0.66). Monogamous men were more likely to practice unprotected anal intercourse in 1984 if they reported it to be their favorite sexual behavior (odds ratio = 5.97, 95% CI = 1.84, 19.34).

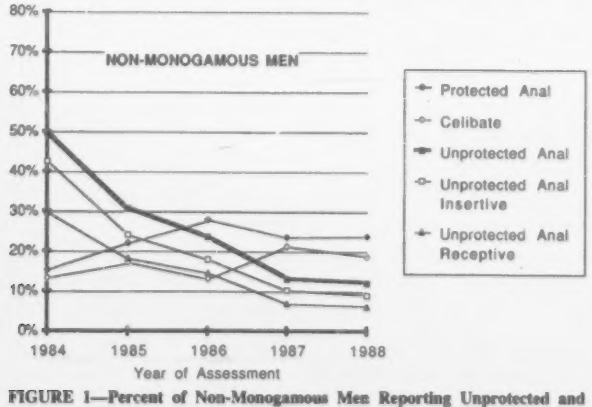

FIGURE 1—Percent of Non-Monogamous Men Reporting Unprotected and Protected Anal Intercourse and Celibacy at Each Annual Assessment

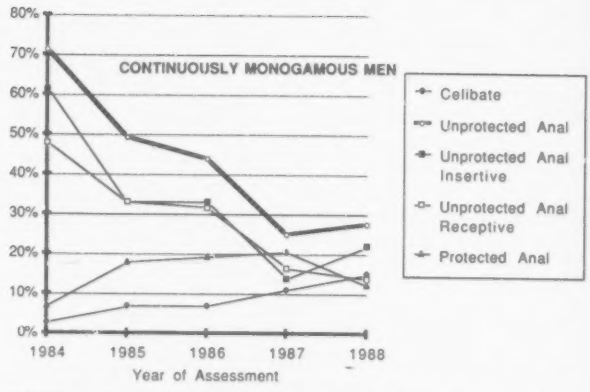

FIGURE 2—Percent of Monogamous Men Reporting Unprotected and Protected Anal Intercourse and Celibacy at Each Annual Assessment

Predictors of Change in Practice of Unprotected Anal Intercourse: 1984–88

Subjects Practicing Unprotected Anal Intercourse in 1984—There were 19.4 percent (N = 41) of the non-monogamous subjects reported practicing unprotected anal intercourse in 1988. Table 2 presents bivariate odds ratios and adjusted odds ratios from the full multivariate model to describe relationships between 1984 predictor variables and the practice of anal intercourse in 1988 for those subjects who reported unprotected anal intercourse in 1984. Unprotected anal intercourse as a favorite sexual activity predicted the practice of anal intercourse four years later. In addition, learning that one was seropositive and experiencing AIDS prodromal symptoms predicted decreased practice of unprotected anal intercourse. The adjusted odds ratios from the limited multivariate analysis were as follows: unprotected anal intercourse as a favorite sexual activity (8.26, 95% CI = 1.88, 36.23) and diagnosed as seropositive (0.37, 95% CI = 0.14, 0.99). Monogamous men who practiced unprotected anal intercourse in 1984 but did not practice it in 1988 were more likely to have been diagnosed as HIV antibody positive (odds ratio = 0.54, 95% CI = .40, .74), while those who tested antibody negative were more likely to continue the practice (odds ratio = 4.44, 95% CI = 1.25, 15.82).

Subjects Not Practicing Unprotected Anal Intercourse in 1984—Among non-monogamous subjects not practicing unprotected anal intercourse in 1984, 5.3 percent (N = 11) reported practicing this behavior in 1988. The only 1984 variable to predict this behavior in 1988 was unprotected anal intercourse as a favorite sexual behavior (odds ratio = 4.21, 95% CI = 1.08, 16.37).

Discussion

Public health campaigns designed to reduce HIV transmission through sexual relations may benefit from the examination of results such as these. Men who initially practiced unprotected anal intercourse outside of a mutually monogamous relationship, even in 1984 when risk information was disseminated widely, preferred unprotected anal intercourse,

TABLE 1—Cross-sectional Predictors of Unprotected Anal Intercourse in 1984 among Non-monogamous Men

Predictor Variables (1984)	Percent (N) Reporting Unprotected Anal Intercourse above Predictor Variable Cutoff	Bivariate Odds Ratio (95% CI)	Adjusted Odds Ratio[b] (95% CI)
Age (>36 years)	37.6 (79)[a]	0.53 (0.36, 0.78)	0.51 (0.29, 0.86)
Unprotected Anal Intercourse-Favorite	79.1 (167)	5.37 (3.49, 8.26)	5.98 (3.46, 10.49)
History of STDs (≥1)	41.5 (80)	1.37 (0.91, 2.07)	1.42 (0.81, 2.46)
Believe Exposed to HIV	65.8 (131)	1.44 (0.96, 2.16)	1.43 (0.79, 2.56)
Prodromal Symptoms (≥1)	16.5 (35)	1.36 (0.79, 2.34)	1.48 (0.66, 3.31)
Personal Efficacy (>mdn)	38.7 (77)	0.42 (0.27, 0.62)	0.36 (0.21, 0.62)
Support-Health Promotion (>mdn)	50.5 (104)	1.10 (0.75, 1.62)	1.38 (0.80, 2.38)
Support-Health Risk (>mdn)	51.9 (108)	1.97 (1.33, 2.92)	1.48 (0.85, 2.57)
Health Guideline Knowledge (>mdn)	42.1 (80)	0.57 (0.38, 0.85)	0.96 (0.60, 1.78)
Personal Susceptibility (>mdn)	46.0 (91)	0.85 (0.57, 1.26)	0.65 (0.37, 1.13)
Depression (>mdn)	44.5 (94)	0.79 (0.54, 1.16)	0.51 (0.29, 0.89)
AIDS Loss (≥1)	32.2 (67)	0.71 (0.48, 1.06)	0.62 (0.36, 1.07)

[a] Total Ns vary from 384 to 421 due to missing data.
[b] n = 308 (152 not practicing unprotected intercourse and 156 practicing unprotected intercourse) due to the necessity of having all cases with complete data on every variable for the complete multivariate model.

TABLE 2—Predictors from 1984 of Unprotected Anal Intercourse in 1988 among Non-monogamous Men Who Practiced Unprotected Anal Intercourse in 1984

Predictor Variables (1984)	Percent (N) Reporting Unprotected Anal Intercourse above Predictor Variable Cutoff	Bivariate Odds Ratio (95% CI)	Adjusted Odds Ratio (95% CI)[b]
Age (≥36 years)	43.9 (18)[a]	0.85 (0.43, 1.69)	1.06 (0.36, 3.12)
Unprotected Anal Intercourse-Favorite	95.1 (39)	6.39 (1.48, 27.64)	8.21 (1.42, 47.28)
History of STDs (≥1)	32.4 (11)	0.62 (0.28, 1.36)	1.09 (0.37, 3.19)
Believe Exposed to HIV	55.0 (22)	0.56 (0.27, 1.14)	0.56 (0.24, 2.11)
Prodromal Symptoms (≥1)	20.0 (7)	1.05 (0.42, 2.61)	5.10 (1.31, 19.84)
Personal Efficacy (>mdn)	60.0 (24)	1.68 (0.83, 3.41)	2.03 (0.73, 5.67)
Support-Health Promotion (>mdn)	15.4 (67)	0.46 (0.18, 1.18)	0.31 (0.09, 1.04)
Support-Health Risk (>mdn)	47.5 (19)	1.36 (0.68, 2.73)	0.70 (0.24, 2.03)
Health Guidelines Knowledge (>mdn)	55.6 (20)	1.46 (0.70, 3.03)	2.11 (0.70, 6.35)
Personal Susceptibility (>mdn)	50.0 (20)	1.22 (0.61, 2.45)	1.34 (0.47, 3.79)
Depression (>mdn)	36.6 (15)	0.66 (0.33, 1.34)	0.38 (0.13, 1.12)
AIDS Loss (≥1)	33.3 (13)	1.06 (0.51, 2.23)	0.72 (0.24, 2.11)
Diagnosed as HIV+	22.0 (9)	0.36 (0.16, 0.82)	0.16 (0.04, 0.59)
Diagnosed as HIV−	48.8 (20)	2.05 (1.02, 4.11)	0.61 (0.19, 1.90)

[a] Total NS vary from 190 to 211 due to missing data.
[b] n = 146 (29 practicing unprotected intercourse and 117 not practicing unprotected intercourse).

had less personal efficacy, were younger, reported that their friends were more likely to engage in high-risk behavior, and had less belief in the importance of the health guidelines. The continued practice of unprotected anal intercourse over time was associated with preference for unprotected anal intercourse and a diagnosis of being seronegative, while a seropositive diagnosis was associated with a reduction in this behavior. Risk reduction programs may need to eroticize alternative sexual practices,[d] promote skills that increase self-efficacy,[16] modify social norms supporting high-risk behavior,[17] and encourage antibody testing so that seropositive individuals are further reinforced to reduce sexual behaviors likely to infect others with HIV.[18–22]

Unprotected anal intercourse as a favorite sexual activity strongly predicted this high-risk behavior both cross-sectionally and longitudinally. Clearly, pre-existent habit patterns and preferences promote continuation of other behaviors that are detrimental to health. Models developed for smoking cessation and alcohol addiction may be adapted to intervene in this new health threat.

Additionally, those who practiced high-risk behavior are supported by the norms they perceive in their social world. Men who initially perceived their friends and social contacts to engage in high-risk behavior were more likely to practice high-risk sex. Norms can influence behavior in two ways. First, individuals may hold norms supporting safer sex, but not share them with peers. In this instance, interpersonal mechanisms are needed for making implicit norms explicit. Secondly, some explicit norms may not support safer sex. In those cases, strategies may be needed that replace these norms with explicit norms for safer sex.[17]

The belief that one is personally capable of implementing

[d] D'Eramo JE, Quadland MD, Shatts, W, Schuman R, and Jacobs R: The 800 men project: A systematic evaluation of AIDS prevention programs demonstrating the efficacy of erotic, sexually explicit safer sex education on gay and bisexual men at risk for AIDS. Stockholm, Sweden: Paper presented at the IV International Conference on AIDS, June 1988.

safe sex practices may be necessary for behavior change. Self-efficacy was associated with lower risk cross-sectionally in 1984. Our self-efficacy items tapped the individual's skill in engaging in safer sexual practices as well as the ability to negotiate safer sex with potential sexual partners. Methods for increasing self-efficacy, especially through skill training, may reinforce HIV sexual behavioral risk reduction.

Knowledge of positive HIV antibody status was associated with reduction of sexual risk. Offering antibody testing and encouraging individuals to act on their knowledge of test results may be regarded as helpful components of an overall community behavioral health program. Receipt of test results took place in the social and legal context of HIV testing in San Francisco. Testing was available under conditions of anonymity (88 percent of the group received test results at Alternative Test Sites which used anonymous procedures) or, at the very least, confidentiality. Legal guarantees of non-discrimination based either on sexual preference or HIV antibody status have been provided in San Francisco and in California. Testing and counseling takes place in a community context of mass public education about AIDS risk reduction. Antibody testing may result in beneficial behavior change, but perhaps only when such additional resources and assurances are provided.[20,*] Knowledge of seronegative status was associated in the bivariate analyses with increased practices of unprotected anal intercourse. It was not possible, however, to determine the meaning of this finding. It might have been the case that seronegative individuals, upon learning their serostatus, sought out other seronegative men to practice their favorite behavior, or were tested upon beginning a relationship. At any rate, the potential for "silent infection" in some seronegative men should suggest that these men exercise great caution in practicing highly risky activities to avoid seroconversion.[23]

Being in a monogamous gay relationship was associated with higher risk sex throughout the entire study. The interaction of sexuality and relationship is a complex one. Other authors have reported a decrease in overall sex activity within longer term primary relationships, both heterosexual and homosexual.[24,25] However, for these respondents, the establishment of trust in a primary bond and mutual support for specific sexual habits and tastes may have led to increased sexual risk activity.

This sample may have limited generalizability in that it includes predominantly White gay males who are educated and living in San Francisco. Clearly, similar research with other groups in other cities is needed to determine if these or other factors are related longitudinally with behavior change. A recent review of behavioral factors in HIV infection[9] found the field confounded by different conceptualizations and operationalizations of concepts. Needed are standardized instruments to be applied to different communities so that the generalizability of elements of behavior change across groups and communities can be determined.[26] Missing from this analysis were data regarding alcohol and drug usage, and their relationship to high-risk sex. These variables have been found to be strongly associated in previous work.[27–29] Regrettably, because they were not included in our baseline questionnaire, they do not appear in this multivariate model. Future studies of high-risk behavior would profit from analysis of the additional contribution of drug and alcohol use in the context of the psychological and social variables already described here. Since these are self-report data, issues of social desirability with regard to reports of sexual behavior need to be considered. When these respondents were recruited they were given thorough reassurances of confidentially, and they have been followed with repeat assessments for many years, giving us less reason to suspect dishonest reporting. Analyses comparing those who dropped out with continuers found few significant differences in reported frequencies of target sex behaviors. Although the magnitude of change in sexual behavior cannot be determined precisely by self-report methods, the trends described by this research closely parallel changes described in other behavioral studies in San Francisco described above.

* Doll L, O'Malley PO, Pershing A, *et al:* High risk behavior and knowledge of HIV-antibody status in the San Francisco City Clinic cohort. Stockholm: Paper presented at the IV International Conference on AIDS, 1988.

ACKNOWLEDGMENTS

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Public Health Briefs

The Mortality Profile of Black Seventh-Day Adventists Residing in Metropolitan Atlanta: A Pilot Study

FREDERICK G. MURPHY, MSPH, MPIA, DANIEL S. BLUMENTHAL, MD, MPH, JOANN DICKSON-SMITH, MSPH, AND RALPH P. PEAY, MPH

Abstract: Mortality information was gathered for 110 Black Seventh-day Adventist members of seven churches in Metropolitan Atlanta, Georgia during the period 1980–87. Seventy-seven percent of the deaths were due to cardiovascular diseases; 8 percent due to cancer, the second leading cause of death. The cancer rate is extremely low in comparison to the proportion of deaths due to cardiovascular diseases. Subsequent research on this population will take into consideration lifestyle factors which could contribute to this finding. (*Am J Public Health* 1990; 80:984–985.)

Introduction

Differences in mortality rates and life expectancy between Black and White Americans are well known,[1–4] as are reduced disease specific mortality rates among certain religious groups whose lifestyles protect them from various risk factors.[5,6] Black Seventh-day Adventists are a group whose race is at high risk for early death but whose lifestyle may reduce that risk substantially. Black Seventh-day Adventists residing in the Metropolitan Atlanta, Georgia area who are members of the South Atlantic Conference of Seventh-day Adventists have not been previously studied.

Seventh-day Adventist doctrine warns its members, for reasons of health and temperance, not to consume caffeine, tobacco, alcohol, illegal drugs, or pork products.[7–9] Although vegetarianism is not required, it is highly recommended. The doctrine also warns against seeking entertainment in establishments where consumption of proscribed substances take place. It further indicates that health risks can result from such attendance.[6,10] This brief investigation examines mortality among Black Seventh-day Adventists living in Metropolitan Atlanta, Georgia during the seven-year period 1980–87.

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Methods

The seven Black Seventh-day Adventist churches included in this study are established members of the South Atlantic Conference of Seventh-day Adventists also based in Atlanta, Georgia. Black Seventh-day Adventist membership in Atlanta grew from 3,366 in 1980 to 6,002 in 1987. Three of the seven churches were established after 1984.

All deaths among members of Adventist churches are reported to regional conferences on a quarterly basis by church clerks. This is part of a conference policy which requires each of its churches to report the names of all individuals dropped from church rolls due to death, apostasy, or lack of attendance. The death portion of these reports contain only the name of the deceased individual, the year of death, and the church with which he/she was affiliated.

We manually retrieved all quarterly reports submitted to the South Atlantic Conference Office between the years 1980 and 1987. Death information and data from each of these were then matched to death certificates at the Georgia Department of Vital Records. Data on the decedents' age at death, sex, and race were obtained from each available death certificate.

Results

Reports of 116 deaths were obtained from the South Atlantic Conference of Seventh-day Adventist. The year of death was not listed for six and could not be matched to death certificates in the State of Georgia Department of Vital Records. We were able to match the remaining 110.

Table 1 shows the distribution and average age at death by sex for the 110 matched death certificates. The female predominance is consistent with the approximately 2:1 (female:male) ratio in Black Seventh-day Adventist church membership in Atlanta.

Table 2 shows the number of deaths attributed to each

TABLE 1—Distribution of Deaths and Average Age at Death by Sex, Metropolitan Atlanta Seventh Day Adventists

Sex	Number of Deaths	Age at Death (years)		
		Average	Median	Range
Male	42	66.4	59	50–96
Female	68	77.4	71	55–104
Total	110	71.5	70	50–104

Metro-Atlanta Minority SDA Mortality Profile

TABLE 2—Total Deaths by Specific Cause and Sex

Cause of Death	Male	Female	ICD Codes A	ICD Codes B	Total
Cardiovascular Disease	29	56	4275	436	85
Lung Cancer	3	3	1629		6
Breast Cancer	—	2	1749		2
Prostate Cancer	1	—	185		1
Pneumonia	2	2	1629		4
Respiratory Failure	—	1	486		1
Homicide	4	—	9654	9554	4
Diabetes	—	1	2500		1
Renal Failure	1	1	1809	7100	2
Accidents	2	1	8120	8140	3
Asthma	—	1	4939		1
Total	42	68			110

Metro-Atlanta Minority SDA Mortality Profile
ICD Code A = Most frequently used code
ICD Code B = Second most frequently used code

specific cause of mortality. Cardiovascular disease accounted for 82.3 percent of the deaths in women and 69 percent in men. All four of the homicide deaths were among males.

Discussion

Although based on relatively small numbers of deaths, the results are suggestive. For example, cancer deaths were relatively rare in this pilot study. This could be attributed, at least in part, to abstinence from use of tobacco and alcohol, and a relatively low-fat diet (due to a high prevalence of vegetarianism) among Seventh-day Adventists.[6,8,9,11] This high proportion of cardiovascular disease deaths among this group could be the result of the large number of elderly persons in the group.

Additional research focused on Black Seventh-day Adventists will be required in larger numbers so that more detailed information can be gathered. Phase II of this investigation is currently being proposed.

ACKNOWLEDGMENTS

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Disaster Medical Assistant Team Formed in Atlanta

An Adult Disaster Medical Assistance Team has been formed in Atlanta, Georgia, as part of the National Disaster Medical System, a joint effort of a number of federal agencies. The system will provide help in the event of a disaster large enough to overwhelm state and local resources.

This team of health volunteer personnel from the greater Atlanta area will function under the supervision of Clayton General Hospital—the hospital designated to help coordinate medical assistance units that travel to disaster sites. This is the second team formed in Atlanta this year. In March, the Henrietta Egleston Hospital for Children formed a Pediatric Disaster Medical Assistance Team.

More information is available from the Office of Emergency Preparedness, HRSA, 5600 Fishers Lane, Rm 14-48, Rockville, MD 20857. Tel: (301) 443-6580.

Playground Hazards in Atlanta Child Care Centers

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Abstract: We identified 684 playground hazards in 66 child care centers despite regulations mandating that the grounds be hazard-free. Of 21 centers with ≤5 hazards, 42.9 percent reported a playground-related injury in the previous year; of 25 centers with 6–11 hazards, 52.0 percent reported a playground-related injury; and of 20 centers with ≥12 hazards, 60.0 percent reported a playground-related injury. Climbing equipment ≥6 feet tall generally had inadequate impact-absorbing undersurfacing and had over twice the rate of fall injuries as climbing equipment <6 feet. (*Am J Public Health* 1990; 80:986–988.)

Introduction

Investigators of medically attended injuries among children attending child care centers found that 47 percent of injuries occurred on the playground.[1] During a 2,000-hour year (40 hours per week × 50 weeks), an estimated 1.64 percent of children in a center will incur a playground-related injury requiring medical evaluation; 0.98 percent will incur a playground equipment-associated injury, and 0.42 percent will incur a climber-related injury.[1] In an attempt to understand why so many injuries occur on playgrounds, we evaluated playground hazards at 66 child care centers.

Methods

The methods of the injury study have been described in detail elsewhere.[1] Briefly, 80 of the 605 licensed centers in Atlanta were randomly selected, and 71 participated in a study of medically attended injuries from June 1987 through May 1988. Of those 71 centers, one had no playground; by the time this study began, one center had replaced its playground, and three centers had closed. Directors of the remaining 66 centers allowed us to evaluate their playgrounds.

Between July and October 1988, two of three evaluators (KWH, LSC, and JMB) visited each participating center. Safety hazards evaluated are listed in Table 1 and included those defined by the Statewide Comprehensive Injury Prevention Program of the Massachusetts Department of Public Health and others.[2–5] Any hazards from playground changes made after March 1, 1988 were excluded from analysis. After the inspection, the director was shown the safety problems identified and given information about playground safety.

Equipment height was measured from the ground to the maximum height that a child's feet can reach. The undersurface (grass, sand, wood chips, etc.) below each piece of equipment was recorded. The undersurface's minimum depth was measured below the point of maximum attainable height.

TABLE 1—Playground Equipment-associated Hazards Identified, Atlanta Child Care Centers, 1988

Type of Equipment-associated Hazard	Number of Times Identified: All Equipment	Number of Times Identified: Climbers Only
Rock, concrete, root or stump in fall zone	171	35
Inadequate clearance*	56	30
Sharp protrusion	42	17
Contaminants in undersurface	38	9
Missing part	31	11
Blunt protrusion	29	14
Trip-and-fall hazard	27	4
Loose parts	21	7
Open "S hook	20	0
Tip-over hazard	18	8
Entrapment hazard	16	12
Hard swing seats	13	0
Broken parts	12	0
Other**	34	10
All equipment-associated hazards	528	157

*Inadequate clearance was defined as an obstruction (fence, wall, etc.) within 4 feet of a climber or within 8 feet of the arc of a swing.
**Other includes all equipment-associated hazards appearing <12 times in surveyed playgrounds.

Results

The 66 centers had 135 playgrounds. At 21 (15.6%) playgrounds we identified no hazards; at 114 playgrounds we identified a median of five hazards (range 1–16). Only one center had no playground hazards identified; the other centers had a median of eight playground hazards (range 1–28).

Hazards Unrelated to Equipment

At 66 (48.9 percent) playgrounds we identified a total of 156 hazards unrelated to equipment (median = 2; range = 1–8). Twenty-eight (20.7 percent) playgrounds had gaps in the fencing wide enough for a child to go through. At 11 playgrounds, children could enter a hazardous area within the playground (e.g., a storage shed). Poison ivy was found on four playgrounds, briars on five, and broken glass on five. Other hazards noted included 33 trip-and-fall hazards, 27 protruding rocks, tree stumps, or concrete pieces; and 23 sharp protrusions.

Equipment-associated Hazards

One hundred and eleven (82.2 percent) playgrounds had an equipment-associated hazard (median = 4; range = 1–13). Although climbers, swings, and slides accounted for 52.2 percent of the equipment (Table 2), they were associated with 65.3 percent of the hazards. Hazards were associated with 36.1 percent of the equipment; by type, swings were the most frequently associated with a hazard, followed by spring riders

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TABLE 2—Playground Equipment: Number of Each Type, Number of Hazards, and Percentage with Hazards, Atlanta Child Care Centers, 1988

Type of Equipment*	Number	Total Number of Hazards Identified	Percentage of Equipment with a Given Number of Hazards**		
			0	1	≥2
Climbers	249	157	55.4	30.5	14.0
Swings***	165	119	47.9	36.3	15.7
Slides	138	69	66.7	22.5	10.8
Barrels	64	6	90.6	9.4	0.0
Seesaws	63	27	65.1	28.6	6.4
Merry-go-rounds	63	33	63.5	22.2	14.3
Spring riders	58	37	51.7	39.7	8.6
Balance beams	47	10	83.0	14.9	2.1
Sand boxes	44	16	75.0	13.6	11.4
Other	166	54	75.3	16.9	7.8
All equipment	1057	528	63.9	25.4	10.7

*Only shown for those types of equipment appearing ≥40 times in surveyed playgrounds.
**Not including inadequacy of undersurface.
***Number of swing seats = 518.

and climbers. The most frequently encountered equipment-associated hazards were hard objects in fall zones (Table 1).

Undersurface below Climbers

The most common surface below climbing equipment was earth or grass (60.6 percent) (Table 3). Impact-absorbing loose-fill materials (e.g., sand or wood chips) were beneath 38.2 percent of climbers, but only 4.2 percent had depths of ≥4 inches. About 27 percent of the climbers were ≥6 feet high and only two (2.9 percent) of those had rubber matting or ≥4 inches of loose-fill material beneath.

Relationship of Hazards to Injuries

During the one-year injury study,[1] of 21 centers with ≤5 hazards, 42.9 percent had reported a playground-related injury; of 25 centers with 6–11 hazards, 52.0 percent reported a playground-related injury; and of 20 centers with ≥12 hazards, 60.0 percent reported a playground-related injury. This same trend was noted for equipment-associated and climber-related hazards and reporting of equipment- and climber-related injuries.

We could not identify two involved climbers for 15 climber-related fall injuries reported in the injury study.[1] Of the 13 pieces of climbing equipment associated with a fall injury, 46.2 percent were ≥6 feet compared to 26.4 percent of climbers unassociated with a fall injury. Climbers ≥6 feet tall had 8.8 medically attended fall injuries per 100 pieces of equipment compared to 3.9 per 100 for climbers <6 feet tall (Table 3).

Six other injuries appeared associated with identified hazards. One head injury occurred from a fall from a broken swing seat. A dental injury resulted from a fall off a bridge with no handrail. One fracture occurred from a fall off a 6.3 foot high slide with an earth undersurface and exposed concrete anchoring. One fracture occurred from a fall off a balance beam 3.3 feet above ½ inch of pea gravel. One fracture and one bruise caused by tripping occurred on playgrounds with tripping hazards.

TABLE 3—Surface and Surface Depth Beneath Climbing Equipment, by Height of Climber and Number and Rate of Medically Attended Fall Injuries, Atlanta Child Care Centers, 1988

Surface	Depth (inches)	Number of Climbers by Height (feet)						
		≤3	4	5	6	7	≥8	Total
Earth/grass	NA	43	40	24	26	12	6	151
Mulch/wood chips/pine straw (inches)								
	≤1	7	12	8	9	2	0	38
	2–3	8	4	3	0	0	0	15
	4–6	1	1	1	1	0	0	4
Sand (inches)								
	≤1	4	3	2	0	0	0	9
	2	0	4	0	3	2	1	10
	3	2	0	1	1	0	0	4
Pea gravel (inches)*								
	≤1	4	2	4	2	1	1	14*
	2	0	0	1	0	0	0	1
Wood Planking	NA	0	1	0	0	0	0	1
Rubber matting	1	0	0	0	1	0	0	1
Total		69	67	44	43	17	8	248*
No. injuries**		1	3	3	3	3	0	13
Fall injuries/100 climbers		1.4	4.5	6.8	7.0	17.6	0.0	

NA = Not applicable.
*Height unknown for one climber with a 1 inch pea gravel undersurface.
**Injuries ascertained from reference 1. Heights of 2 climbers associated with a fall injury were unknown.

Discussion

At child care centers, most injuries that require medical attention occur on playgrounds.[1,6,7] Of the playgrounds we studied, 84 percent contained a hazard. As the number of hazards increased, so did the likelihood a playground-related injury was reported.

Climbers have been associated with a disproportionate number of playground injuries.[1,6,8–10] Although climbers constituted 23.6 percent of study center equipment, they were involved in 42.5 percent of the equipment-associated injuries.[1] Moreover, the rate of fall injuries increased with the height of the climbing equipment.

In our study, 27 percent of climbers were ≥6 feet high and 61 percent had earth or grass underneath. Earth and grass are not considered suitable undersurfaces for climbers ≥4 feet high.[11–12] If loose-fill material is the undersurface, depths of ≥6 inches are recommended[2,11]; yet, the centers we studied rarely had loose-fill materials at recommended depths. Those responsible for school playgrounds have been found legally negligent for failing to provide adequate undersurfacing beneath equipment.[13]

Despite Georgia State regulations for child care centers mandating that climbing equipment be securely stationed, 5 percent of climbers had tip-over hazards. These regulations require that the playground be fenced; yet, 21 percent of the playgrounds studied had fencing gaps large enough to permit a child to pass through. Although prohibited on center grounds, "hazardous conditions" are not defined in the regulations. Although Georgia codes mandate "resilient surfaces" beneath climbers, they do not specify the types and depths of surfaces that satisfy this definition.

We believe that regulations regarding child care center playgrounds should be more specific and better enforced. In addition, regulators, center directors, and parents should be educated about playground hazards and prevention of playground injuries.[3,14,15]

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A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke

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Abstract: We enrolled 15 nonsmoking volunteers to evaluate the feasibility of measuring personal exposure to environmental tobacco smoke (ETS) at work and to characterize workplace exposures. During one workshift, we obtained questionnaires on exposure, saliva and urine for cotinine, and personal air samples for respirable particles and nicotine. The levels of cotinine, respirable particles, and nicotine varied widely with self-reports of exposure to ETS, but on average increased with increasing exposure. (*Am J Public Health* 1990; 80:988–990.)

Introduction

While health effects of passive smoking on children and adults have been identified, the principal location of exposure investigated has been the home.[1,2] Workplace exposure has received less attention, and health effects of environmental tobacco smoke (ETS) in the workplace remain controversial.

We enrolled 15 nonsmoking adults to determine the feasibility of measuring personal exposure to ETS at work and to characterize workplace exposures of this small group of subjects. Indicators of exposure, measured during a workday, included questionnaires, personal samples for respirable particles (RSP) and nicotine, and urinary and salivary cotinine.

Methods

Between October 1986 and May 1987, 15 nonsmoking volunteers (eight men, seven women), 18 years of age and older, were recruited from the Albuquerque, New Mexico area. We obtained exposure questionnaires, saliva, urine, and personal air particle samples during one workshift. The saliva and urine specimens were obtained before and after the workshift. Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone, *et al.*[3] Details of the assay in our laboratory have been reported previously.[4]

During the workshift, each subject wore a personal monitoring pump running at 1.7 l/min with a 10 mm nylon cyclone clipped to the shirt collar.[5] RSP samples were collected on 37 mm Fluropore filters (Millipore Corp). Nicotine was collected on a glass fiber backup filter treated with sodium bisulfate to minimize volatilization; after extraction from the filter, analysis for nicotine was done on a gas chromatograph with a flame ionization detector.[6] The recovery of nicotine by this procedure has been shown to be 98 percent efficient.

From the questionnaires, we derived measures of exposure including the total number of cigarette smokers and total number of hours exposed during the workshift. To describe the relationships among the measures of ETS exposure, Spearman correlations were calculated. Data analysis was performed with standard programs.[7]

Results

Occupations of the subjects were diverse (Table 1); mean age was 44.8 years; average duration of the workshift and of the personal monitoring was 6.5 hours (SD ± 2.0).

Exposure to cigarette smokers at work was reported by 13 of the 15 participants. Of the 13 reporting exposure, two reported exposure to crowds of smokers during their workshift and the remaining 11 encountered a mean of 8.8 smokers (SD ± 6.7). The mean reported hours of exposure was 3.4 (SD ± 2.1).

Respirable particle and nicotine concentrations varied widely with the reported number of smokers and hours of exposure. The mean concentrations for RSP and nicotine were 63.9 $\mu g/m^3$ (SD ± 41.5) and 20.4 $\mu g/m^3$ (SD ± 20.6), respectively. Correlations between the atmospheric markers

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TABLE 1—Description of Participants in a Personal Monitoring Study of Exposure to Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Occupation/Workplace	Workshift Duration/ Exposure Duration (hours)	RSP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)
Males			
Physician/Hospital	8/5	52.3	10.0
Social Worker/Office	8/0	44.0	2.5
Stock Broker/Office	8/2	69.4	7.2
Bus Boy/Restaurant	8/8	145.8	45.0
Maintenance Worker/Retail Store	8/3	85.2	6.9
Barber/Barber Shop	8/0	14.7	4.0
Barber/Barber Shop	8/4	145.8	13.7
Volunteer/Hospital	4/2	80.0	46.0
Females			
Interviewer/Public Transportation	3/2	4.0	0.0
Travel Agent/Office	8/5	85.7	50.0
Travel Agent/Office	6/4	62.1	46.7
Attorney/Office	8/6	63.3	5.9
Volunteer/Hospital	4/3	27.6	6.3
Volunteer/Hospital	4/3	25.2	8.7
Volunteer/Hospital	4/4	53.2	53.2

and the questionnaire measures of exposure to ETS were moderate (Table 2).

As was observed for the atmospheric markers, the post-workshift urinary and salivary cotinine levels varied widely with self-reported exposure. In comparison with pre-workshift levels, post-workshift levels were not consistently increased. The mean pre-workshift urinary and salivary cotinine concentrations were 31.8 ng/mg Cr (SD ± 67.6) and 2.9 ng/ml (SD ± 5.0), respectively. For the post-workshift levels, the corresponding values were 19.7 ng/mg Cr (SD ± 43.2) and 3.5 ng/ml (SD ± 5.9).

Spearman correlation coefficients were calculated to examine the relations among the questionnaire variables, the atmospheric markers, and urinary and salivary cotinine (Table 2). Moderate correlations were obtained for self-reports and cotinine levels, and nicotine levels and cotinine levels. However, RSP levels and cotinine concentrations were not correlated.

TABLE 2—Spearman Correlations between Various Measures of Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Correlated Measures	N	r
RSP ($\mu g/m^3$) with:		
Nicotine	15	0.57*
Total number of smokers	15	0.44
Total hours of exposure	15	0.53*
Postshift urinary cotinine	14	0.05
Postshift salivary cotinine	11	−0.07
Nicotine ($\mu g/m^3$) with:		
Total number of smokers	15	0.62*
Total hours of exposure	15	0.54*
Postshift urinary cotinine	14	0.60*
Postshift salivary cotinine	11	0.46
Postshift urinary cotinine (ng/mg Cr) with:		
Total number of smokers	14	0.39
Total hours of exposure	14	0.57*
Postshift salivary cotinine (ng/ml) with:		
Total number of smokers	11	0.63*
Total hours of exposure	11	0.45

*$p < 0.05$

Discussion

The controversial effects of involuntary smoking in the workplace need further investigation. The conduct of such research would be facilitated by the development of unintrusive and accurate methods of exposure assessment. Alternative approaches include active and passive monitoring, biological markers, and questionnaires. We have shown that personal monitoring for tobacco smoke components can be accomplished in the workplace. However, many employers and employees would not participate in the study because of concern about the wearing of pumps.

Despite the small number of subjects studied in this investigation, objective evidence of exposure to ETS was obtained in various workplaces. The levels of RSP and nicotine were similar to those observed in other investigations.[6,8–10] However, few of these studies included information on the intensity and duration of exposure to ETS.[10]

We observed moderate positive correlations among the questionnaire measures of ETS exposure, the results of personal monitoring for RSP and nicotine, and measurements of urinary cotinine. Each of these types of measures provides a differing index of exposure to ETS.[1] The questionnaire measures that were used assess source strength, but concentrations of ETS are also influenced by room volume and ventilation. Nicotine is a specific marker of exposure to ETS, whereas RSP is nonspecific. Cotinine levels reflect nicotine exposure, but also are determined by timing of specimen collection[10] and uptake and metabolism. Thus, tight concordance among these broad indicators of exposure used in this study would not be anticipated.

Because of the differing characteristics of questionnaires, personal monitoring, and biological markers for assessing ETS exposure, no single method should be considered as optimal for studying the workplace. We recommend that assessment of ETS exposure in indoor environments should utilize multiple approaches to characterize short- and long-term exposures. In population studies, questionnaire measures of exposure offer the simplest approach with personal atmospheric markers and biologic markers providing methods for estimating the potential magnitude of misclassification of self-reported exposure.

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Sexual Histories of Heterosexual Couples with One HIV-Infected Partner

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Abstract: Ninety-eight heterosexual couples enrolled in a HIV transmission study, at least one of whom was HIV-infected, were interviewed about sexual behavior. Although males and females were interviewed separately, there was agreement between them on the number of sexual contacts, the practice or anal intercourse, and condom use. These findings of strong reliability are encouraging, but do not necessarily imply that the data are valid. (*Am J Public Health* 1990; 80:990–991.)

Introduction

The best way to prevent the spread of infection from human immunodeficiency virus (HIV) is voluntary behavioral change.[1] Before recommending strategies to promote such changes, one must first establish the kinds of behavior currently practiced. To do so, AIDS (acquired immunodeficiency syndrome) researchers rely almost exclusively on interviewing techniques, but many question the validity and reliability of this methodology.[2] For example, in a study of the reproductive histories of a group of women, Hornsby and Wilcox[3] established that daily logs of sexual behavior were more accurate than comparable data collected through retrospective interviews. Similar results were found by both Udry and Morris[4] and Kunin and Ames,[5] who determined that recent and frequent reporting of sexual behavior produced the most precise results. However, AIDS epidemiologists may not have the opportunity to collect data on a regular basis and are thus forced to rely on retrospective reports of behavior even though the accuracy of these reports (especially for a variety of sexual practices) has not been established. Here I report on the reliability of such data, comparing reports of sexual behavior between men and women in couples of which at least one partner was HIV seropositive.

Methods

Since 1985 we have been conducting a study of the heterosexual transmission of acquired immunodeficiency syndrome (AIDS) in California in which we enroll the opposite sex partners of individuals infected with HIV. Although the study began by enrolling the female partners of HIV-infected men,[6] in 1987 we began to enroll participants as couples. Individuals who test positive for HIV at a variety of sites and who also have heterosexual partners are referred to the study as part of their post-test counseling. Enrollment is voluntary; study protocol is described elsewhere.[6]

Interviewers are female and are matched to couples by ethnicity. They are trained in group and individual sessions that include role playing and interpretation of interview responses. Results were reliable across interviewers. To ascertain number of sexual contacts, the interviewer asks how many times the couple has sexual intercourse in a typical week or month. Deviations from this number throughout the relationship are noted. The total number of contacts is estimated by multiplying the duration of the relationship by the typical reported amount and weighting this number by changes in sexual activity over time. Individuals in the couple are interviewed separately on the same day.

Behaviors considered in this report are: vaginal intercourse, anal intercourse, and condom use. Continuous responses were compared using Pearson's correlation coefficient, and categorical responses were compared using the Kappa coefficient and its corresponding confidence limits as described by Fleiss.[7] HIV infection was ascertained from serological tests using enzyme immunoassays with Western Blot confirmation.

Results

This report includes data from 98 couples, 68 percent of which were monogamous at entry into the study. Most of the men and women reported only one partner in the six months prior to entry into the study. Median number of partners since 1978 (the beginning of the AIDS epidemic) was five for the women and 10 for the men. The overall male-to-female transmission rate from the study which began in 1985 was 24 percent. Seventy-seven of the couples included infected men and their female partners; 12 (16 percent) of these women were infected. Twenty-one of the couples were infected women and their male partners; none of these men were infected. The reported risk group of the infected partner of the 98 couples varied and is described in Table 1. Sixty-three percent of the couples were White, 20 percent Latino, 7 percent Black, 1 percent Asian, and the remainder were couples of mixed ethnicity. Average income for the couples was $20–29,000 per year; average level of education was 13

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TABLE 1—Risk Group of the HIV-Infected Index Case in 98 Heterosexual Couples

Characteristics	Male N = 77 (%)	Female N = 21 (%)
Bisexual	27 (35)	
Bisexual + Intravenous Drug Use	11 (14)	
Intravenous Drug Use	24 (31)	3 14)
Heterosexual Partner		10 (48)
Hemophiliac or Transfusion-associated	6 (8)	5 (24)
No Identified Risk	9 (12)	3 (14)

school years. The couples had been together from one month (four contacts) to 19 years (greater than 3,000 contacts).

The total number of vaginal intercourse contacts reported by the man and the woman were highly correlated (r = .84, 95% CI = .64, 1). Because the couples knew they were to be interviewed about their sexual behavior, they may have discussed responses to anticipated questions. In fact, some of their responses were identical, but there was still agreement between those partners with non-identical responses (r = .72, 95% CI = .44, 1). Women reported more vaginal contacts than their male partners for the duration of the relationship, but this was not a significant difference (mean estimated number of contacts was 540 by women versus 508 by men). Agreement was stronger for the couples with briefer relationships.

Number of anal intercourse contacts was compared as a continuous variable, with somewhat less strong agreement (r = .44, 95% CI = .13, .74). Responses on anal intercourse as a dichotomous variable (ever versus never) compared using the Kappa coefficient, agreed well (Kappa = .76, 95% CI = .56, .95) (Table 2). Responses on condom use treated as a dichotomous variable (Table 3) also agreed well (Kappa = .74, 95% CI = .54, .94). Two couples who disagreed about the practice of anal intercourse also disagreed about the practice of condom use. The small number of couples and high levels of agreement precluded analyzing differences by risk group of the index case, ethnicity, gender, number of sexual partners, or duration of the relationship.

Discussion

In this group of heterosexual couples with one HIV-infected partner, agreement about sexual practices was observed. Couples agreed somewhat better on the number of contacts of vaginal than anal intercourse. These patterns are similar to those observed by Seage, *et al*[8] in homosexual couples.

Participation was voluntary and the results might not apply to all heterosexual couples with one infected partner. In addition, this evidence of good reliability does not necessarily imply validity. For example, measurements were not independent because couples may have discussed responses prior to the interview.

The occasional disagreement among partners suggests that researchers still must be cautious in relying on the reports of sexual behaviors they are examining. We intend to examine this phenomenon further by assessing willingness to discuss HIV risk and serostatus between partners. Kegeles, *et al*[9] found that HIV-infected homosexual men were not completely forthright in discussing their antibody status with non-primary partners. These results remain to be examined between heterosexual partners.

TABLE 2—Practice of Anal Intercourse Reported by Men and by Women Heterosexual Partners, N (proportion)

		Men		
		Ever	Never	Total
Women	Ever	34 (.35)	5 (.05)	39 (.4)
	Never	5 (.05)	54 (.55)	59 (.6)
	Total	39 (.4)	59 (.6)	98

Kappa = .76 (95% CI = .56, .95)

TABLE 3—Condom Use reported by Men and Women Heterosexual Partners, N (proportion)

		Men		
		Ever	Never	Total
Women	Ever	45 (.46)	6 (.06)	51 (.52)
	Never	7 (.07)	40 (.41)	47 (.48)
	Total	52 (.53)	46 (.47)	98

Kappa = .74 (95% CI = .54, .94)

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Maintenance of Nonsmoking Postpartum by Women Who Stopped Smoking during Pregnancy

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Abstract: This paper describes self-reported maintenance of nonsmoking at six months postpartum by women whose abstinence was verified beginning before the 20th week of pregnancy and continuing through delivery (n = 134). The overall maintenance rate of 37 percent was not related to sociodemographic characteristics, smoking and obstetric history, the time when quitting occurred, or a prenatal smoking cessation program. Although this rate is encouraging, more attention should be directed to sustaining prenatal abstinence from smoking beyond delivery. (*Am J Public Health* 1990; 80:992–994.)

Introduction

Many women smokers refrain from smoking while they are pregnant. Randomized trials of prenatal smoking cessation programs have found net differences of 12–25 percentage points,[1–3] with corresponding positive effects on pregnancy outcomes[1,4–6] and health care costs.[6] Even more women who smoked prior to pregnancy (16–41 percent)[1–3,7,8] stop smoking in response to pregnancy before they begin prenatal care, and 65–80 percent of those "spontaneous quitters" maintain abstinence throughout pregnancy.[1,9] Rates of relapse postpartum, however, may be high.[10–13] This is of concern because of growing evidence that parental smoking compromises infant health[14] in addition to that of the mother. This study addresses the following questions:

- How much prenatal smoking cessation is sustained after delivery?
- Do prenatal intervention programs negatively affect postpartum maintenance, as suggested in previous findings?[14]
- What are the characteristics of women who maintain abstinence?
- How soon do women return to smoking?

Methods

All English-speaking pregnant women entering care by the 18th week of pregnancy between July 1985 and June 1987 in one of five health centers of a large multispecialty medical group practice in Los Angeles affiliated with Maxicare Health Plans, Inc, comprised the study population. Those who reported smoking ≥7 cigarettes per week before becoming pregnant (23 percent) were randomly assigned to the experimental program or usual care. Of these (n = 541), 59 percent were intake smokers; 41 percent were spontaneous quitters. Characteristics of the multi-ethnic study subjects are described elsewhere.[3,9] The study tested the effectiveness of a serialized self-help smoking cessation program, the first part of which was given at the intake visit; and the remainder were mailed weekly thereafter.[3]

This analysis is of women who continued their pregnancies as medical group and health plan enrollees and who achieved optimal prenatal smoking cessation (see Table 1). For intake smokers, optimal cessation was defined as quitting before the 20th week of pregnancy. Maintenance for intake smokers and spontaneous quitters was assessed biochemically over the course of pregnancy (average tests per subject = three). Rates of optimal quitting were as follows: Intake smokers, 22.2 percent in the experimental group versus 8.6 percent among the controls; spontaneous quitters, 83.7 versus 78.6 percent.

Data for the present analysis were from a questionnaire and interview at the intake visit and a telephone interview six months after delivery that embedded questions about smoking and other health-related behaviors in questions assessing satisfaction with health services.[3] Postpartum maintenance was defined as self-reported nonsmoking throughout the six months since the birth of the baby. Subpatterns and their prevalence are summarized in Figure 1.

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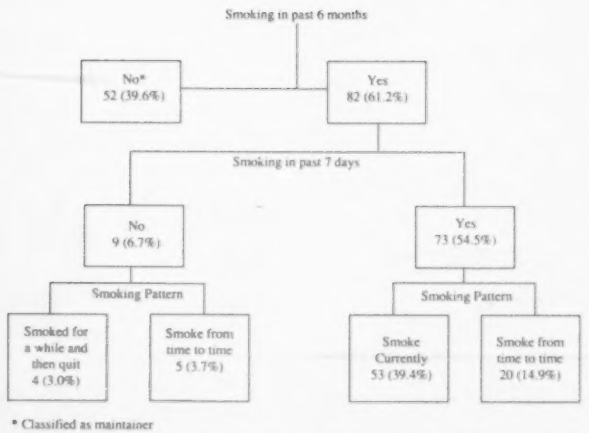
TABLE 1—Sample Attrition by Intake Smoking Status and Experimental Group

	Intake Smokers		Spontaneous Quitters		
	Experimental	Control	Experimental	Control	TOTAL
Randomized for Prenatal Study	165	158	110	108	541
Miscarriage	12	13	9	8	42
Abortion	7	11	4	1	23
Disenrollment/transfer	20	18	10	15	83
Total Sample Size for Analysis in Prenatal Study	126	116	87	84	413
No optimal quit	98	106	15	18	237
Disenrollment/transfer	10	3	12	11	36
Total Eligible for Postpartum Follow-up	18	7	60	55	140
Refused interview	0	1	1	4	6
Total Postpartum Interviews	18	6	59	51	134



* Classified as maintainer

FIGURE 1—Patterns of Postpartum Smoking among Prenatal Quitters (& of total) (n = 134)

Telephone interviews were completed for 134 of the 140 women (96 percent) eligible for follow-up (see Table 1). Comparison of the 134 subjects for whom data were available with the 32 who disenrolled or refused revealed no significant differences in demographic, obstetric history, or smoking history variables (data available on request to author).

Results

Table 2 describes the characteristics of prenatal quitters. The overall maintenance rate was 37.1 percent. Rates did not differ significantly by intake smoking status. Among the spontaneous quitters, experimental status was not related to maintenance. The number of intake smokers was too small to analyze by experimental group status. There were no statistically significant differences ($p > 0.05$) between maintainers and relapsers in sociodemographic characteristics, smoking history, or obstetric history.

Twenty-six percent of the sample reported that they had resumed smoking by the end of the 6th postpartum week (Figure 2). This represented 43 percent of the relapse that occurred. By the end of the third postpartum month, 43 percent had relapsed (71 percent of the six-month total).

TABLE 2—Background and Other Characteristics of Prenatal Quitters at Time of Prenatal Intake

	Percent
Sociodemographic Variables	
Race	
White	57.5
Black	23.1
Other	19.4
Education	
<High school graduate	17.9
High school graduate	41.8
Some college	30.6
>College graduate	9.7
Married	66.4
Age (years)	
18–19	14.2
20–29	67.2
≥30	18.6
Obstetrical Variables	
Primigravida	29.9
Primipara	59.0
Weeks Entering Care	
≤8	26.8
9–13	57.5
≥14	15.7
Smoking History	
Age Smoking Began (years)	
≤16	42.5
17–20	38.8
≥21	18.7
Prepregnancy Smoking Rate	
≤10 cgts/day	66.4
11–19	9.7
≥20	23.9
>60 Minutes to 1st Cigarette	79.9

Discussion

The findings from this study indicate that some prenatal abstinence is sustained postpartum. Although this is based on self-reported smoking status in the postpartum period, two facts offer reassurance: the sample included women whose reported cessation was validated throughout pregnancy, a more sensitive time to report the behavior; and the focus on smoking was masked by questions about other health practices and the health plan services.

The follow-up period in the present study was shorter than the standard of one year in smoking cessation studies. It is likely that much of the relapse was captured, however. In the National Health Interview Study,[7] 95 percent of return to smoking that was reported at 12 months had occurred by six months; this marked leveling off has been observed elsewhere.[10,15,16] Because of the powerful motivation aroused by the pregnancy, the "post-program" period is defined as beginning with the birth. In fact, however, contact with the program in the present study ceased an average of four months earlier, so the measurement period for the intervention group could be viewed as approaching 12 months. Also, return to work and resumption of alcohol and caffeine, potential sources of stress and cues to smoke, already had occurred in most study subjects. Breastfeeding had ceased for all but one woman.

A strength of this study is the presentation of data on rates of return to smoking in a population with verified and continuous abstinence during pregnancy. Further, the definition of maintenance was relatively conservative.[16] Refusers were counted as smokers.

The rate of nonsmoking postpartum was higher in this study compared to findings from other studies. In the same

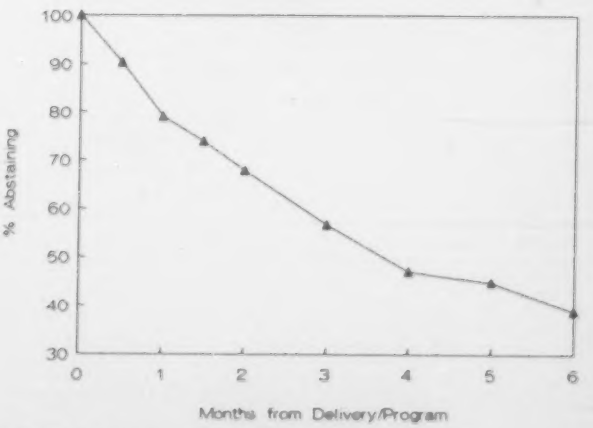


FIGURE 2—Postpartum Smoking Relapse Over Time (n = 134)

TABLE 3—Number and Percent of Prenatal Quitters Who Reported Nonsmoking During the First Six Months Postpartum, by Intake Smoking Status and Experimental Group (n = 140)*

Experimental Group	Intake Smoking Status: Intake Smoker	Intake Smoking Status: Spontaneous Quitter	TOTAL
Experimental	6/18 = 33.3%	23/60 = 38.3%	29/78 = 37.2%
Control	3/7 = 42.8%	20/55 = 36.4%	23/62 = 37.1%
Total	9/25 = 36.0%	43/115 = 37.4%	52/140 = 37.1%

*Includes six women who refused the interview who were considered to be smokers.

population and with a similar intervention but a point prevalence measure of prenatal cessation, the six-week postpartum maintenance rate was 46 percent versus 74 percent in the present study.[12] The three-month postpartum maintenance rate found in an intervention trial in Baltimore[13] was 25 percent for treatment group members versus 57 percent in the present study. The Baltimore population smoked more heavily before pregnancy, had prenatal smoking status assessed only at the eighth month, and had a more intensive intervention including a home visit which may have boosted short-term quitting artificially.

The present results are similar to those reported for six months postpartum in the two other studies that could be located, the National Health Interview followback study[7] and a followup survey of Minneapolis/St. Paul area birth announcements.[17] Both of these groups included spontaneous quitters and used retrospective reports of smoking status during pregnancy. The definitions of prenatal cessation used in these studies did not preclude cessation of shorter duration than the present study.

Within the limitation of small numbers available for sub-analyses, rates of maintenance appeared to be unrelated to smoking status at the first prenatal visit (intake smokers versus spontaneous quitters). Sexton and coworkers[13] found a higher rate of continued abstinence among spontaneous quitters. Because there might be a rebound effect resulting from the emphasis on cessation to protect the fetus, we looked for but did not find any negative effects of the intervention.

These findings suggest that prenatal smoking cessation does carry over to the postpartum period but that exclusive concentration on the prenatal period is not enough. To capitalize further on the opportunity presented to reduce rates of smoking among young women and to protect the health of their offspring, future research should be directed toward the development and testing of intervention programs for late pregnancy and the early postpartum period.

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Changes in Needle Sharing Behavior among Intravenous Drug Users: San Francisco, 1986–88

JOSEPH R. GUYDISH, PHD, AL ABRAMOWITZ, MS, WILLIAM WOODS, PHD, DENNIS M. BLACK, PHD, AND JAMES L. SORENSEN, PHD

Abstract: We analyzed data for San Francisco intravenous drug users entering treatment, April 1986–September 1988 (N = 7,660). The proportion of cases reporting any needle sharing in the month preceding treatment decreased from 50 percent in 1986 to 28 percent in 1988. Similar decreases were reported by two longitudinal cohorts (needle sharing by the same individuals) admitted in 1986 and 1987 (n = 303), and in 1986 and 1988 (n = 205). In a multiple logistic regression model four variables predicted needle sharing: earlier time of admission, cocaine use, younger age, and being White rather than Black. (*Am J Public Health* 1990; 80:995–997.)

Introduction

Successful and sustained risk reduction among intravenous drug users (IVDUs) is vital in slowing the spread of the human immunodeficiency virus (HIV) in the United States. Injectable drug use is implicated in 27 percent of adult acquired immunodeficiency syndrome (AIDS) cases, and this figure is substantially higher for Blacks (45 percent) and Hispanics (47 percent). Well over half of all heterosexually transmitted cases and pediatric cases are among sexual partners and children of IVDUs.[1]

HIV prevention strategies targeting IVDUs, in addition to drug abuse treatment, now include grassroots efforts,[2] clinic-based interventions,[3,4] street outreach projects,[5] community-based information and awareness campaigns,[6] and publicly supported,[7] and unsanctioned needle exchanges.[8] In New York City, where seroprevalence among IVDUs reached about 60 percent,[9] cross-sectional and ethnographic studies report decreased needle sharing, increased needle cleaning, and increased demand for sterile needles on the streets.[10–13]

In San Francisco, one study found no change in needle sharing for cross-sectional samples drawn in 1985 and 1987,[14] while in three other studies IVDUs self-reported some decrease in sharing.[15–17] We report changes in needle sharing behavior for two samples of IVDUs entering drug detoxification programs in San Francisco for successive time periods from 1986 to 1988, and for two retrospective cohorts repeating treatment during the study period. We also evaluated correlates of needle sharing behavior.

Methods

Study respondents were IVDUs consecutively entering outpatient drug detoxification clinics (San Francisco General Hospital and Haight Ashbury Free Medical Clinic) from April 1986 through September 1988 (N = 7,660). Participants were interviewed on admission concerning demographics, drug use, drug treatment, and the number of needle sharing occasions for the 30-day period preceding admission to treatment. To evaluate change over time, the data were partitioned into consecutive six-month calendar periods, giving a total of five time blocks. The proportion of participants reporting any needle sharing was compared across time periods. The two largest cohorts of treatment repeaters within non-adjacent six-month periods were identified in 1986–87 (n = 303), and in 1986–88 (n = 205).

Logistic regression modeling was applied to identify correlates of needle sharing, using only the first admission for each individual during the study period (n = 5,610). The outcome variable was number of needle sharing occasions in the past month, coded dichotomously to reflect *any* sharing versus *no* sharing. Sex, age, education, ethnicity, cocaine use, number of prior admissions to drug treatment (<3 admissions or 3+ admissions), and time period (coded 1–5) were entered as predictors. Since route of administration was not known for cocaine, this variable reflects cocaine administered by smoking, injection, inhalation, and other routes.

Results

Demographic characteristics reported here reflect only the first admission for each individual within each clinic (n = 5,916). For both clinics combined, most admissions (92 percent) were for heroin use. The separate clinic samples differed in terms of age, ethnicity, education, and drug of choice (Table 1).

For all admissions to both clinics (N = 7,660), the proportion of cases reporting any needle sharing decreased at each successive time period, from 50 percent at Time 1 (April-September 1986) to 28 percent at Time 5 (April-

TABLE 1—Demographic Characteristics for Clinic Samples*

Demographics	Clinic	
	San Francisco General Hospital (n = 2,211)	Haight Ashbury Free Medical Clinic (n = 3,705)
Drug of Choice		
% Heroin	100	87
% Speed/Cocaine/Other	—	13
Ethnicity		
% White	49	66
% Black	27	18
% Latino	20	12
% Asian/Indian	4	4
Education		
% High School or Less	78	64
% Some College	22	36
Sex (% Female)	37	34
Mean Age (years)	36	34

*Based on first admission only within each clinic.

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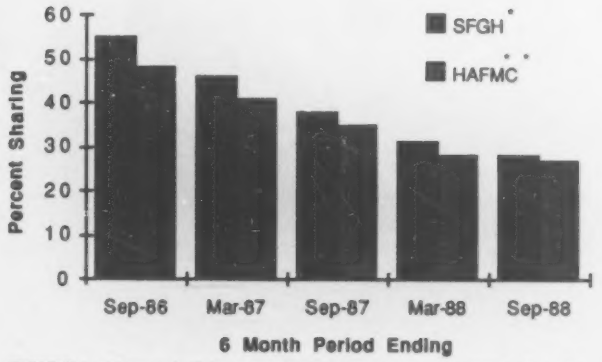

FIGURE 1—Changes in Self-reported Needle Sharing among IVDUs Entering Two San Francisco Outpatient Detoxification Clinics, 1986–88

September 1988). The data for each clinic separately are presented in Figure 1.

Self-reported needle sharing also decreased in individuals seen more than once. For the cohort entering treatment in both 1986 and 1987, the proportion of needle sharers decreased from 52 percent to 35 percent. For the cohort entering treatment in 1986 and 1988, the proportion of sharers decreased from 47 percent to 23 percent.

Frequencies for variables included in the model predicting any needle sharing are given in Table 2, together with a summary of logistic regression results. Four variables predicted needle sharing:

- IVDUs entering treatment at later time periods were less likely to report needle sharing;
- IVDUs reporting any use of cocaine were more likely to report needle sharing;
- older IVDUs were less likely to report needle sharing; and
- Blacks reported less sharing than did Whites.

Discussion

Self-reported needle sharing decreased over time for the total sample (N = 7,660), for each individual clinic sample, for treatment repeaters, and in logistic modeling which considered only first admissions. Consequently, the observed decrease in needle sharing is not likely due to unique characteristics of individual clinic samples, or to the presence of treatment repeaters in samples selected for univariate analyses.

Self-report bias could account for these results. We asked a convenience sample of 20 methadone detoxification participants, after admission to treatment, about underreporting of needle sharing at the time of admission. Most thought that underreporting would occur, but for reasons of social desirability rather than entry into treatment, which implies that these data reflect a lower boundary rather than "true" needle sharing. Collecting data within drug treatment programs also limits generalizability to a large non-treatment population. Of the estimated 16,000 IVDUs in San Francisco,[18] only 5,000 entered treatment in 1987.

The finding of decreased needle sharing behavior over time indicates that many San Francisco IVDUs are reducing risk of HIV infection by modifying needle sharing behavior. These risk-reduction practices, together with increased needle cleaning previously reported among San Francisco IVDUs,[14,19] may account in part for the decrease and leveling of seroconversion in this population from 7 percent per year in 1985 to an estimated 2.4 percent currently.[20] Nevertheless, 30 percent or more of San Francisco IVDUs still admit to the

TABLE 2—Distribution of Predictors and Results of Logistic Regression Analyses (N = 5,610)*

Predictors	Predictor Distributions			Logistic Regression Results+	
	Response Value	n	(Percent Sharing)**	Units++	OR (95% CI)
Age (years)	<30	1240	(43)	+10 years	0.87 (0.80, 0.94)
	30–39	2943	(40)		
	40–49	1070	(40)		
	50+	357	(28)		
Education (highest grade)	<10	458	(45)	+5 years	1.01 (0.99, 1.03)
	10–14	4566	(40)		
	15+	586	(40)		
Prior Treatment	<3	2799	(41)	<3, ≥3	0.98 (0.87, 1.09)
	≥3	2811	(39)		
Sex	Male	3636	(39)	M/F	0.99 (0.89, 1.12)
	Female	1974	(41)		
Cocaine use	No	3805	(38)	No/Yes	1.41 (1.25, 1.59)
	Yes	1805	(43)		
Black	No	4381	(41)	No/Yes	0.73 (0.63, 0.85)
	Yes	1229	(35)		
Latino	No	4784	(40)	No/Yes	0.99 (0.85, 1.17)
	Yes	826	(41)		
Time period	1	1384	(52)	+6 months	0.77 (0.74, 0.80)
	2	1199	(43)		
	3	1034	(38)		
	4	1071	(32)		
	5	922	(29)	-	

*Sample includes only the first admission into either clinic, for each individual, during the study period.
**Percentages reflect proportion of cases in each category who reported needle sharing.
+Adjusted odds ratios.
++For continuous variables, sign gives direction of relationship with needle sharing. For dichotomous variables, odds ratio reflects change in units from left to right.

behavior that most effectively facilitates the spread of HIV, signaling the need for a second wave of innovative and aggressive AIDS prevention to include greater emphasis on cleaning needles with bleach, increased collaboration between members of the IVDU subculture and public health workers, and the use of multiple intervention strategies.[21,22]

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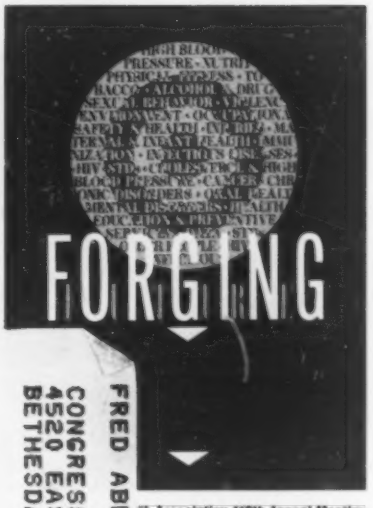

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Meeting the Needs for Health Services of Persons with Mental Retardation Living in the Community

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Abstract: Adequate health services are critical to the success of efforts to maintain persons with mental retardation in the community, yet information concerning the health status of this population is in short supply. This paper presents the results of a survey of 333 mentally retarded persons randomly selected from a population of 1,333 such individuals living in community settings. Almost two-thirds had chronic conditions requiring medical intervention. The top five conditions in terms of prevalence were neurologic, ophthalmologic, dermatologic, psychiatric-emotional, and orthopedic. The majority of conditions were being managed appropriately in the community health system. A substantial proportion can be managed by primary care physicians with limited specialty involvement. For almost 60 percent of clients with conditions requiring home treatments on an ongoing basis, however, service gaps were identified. Other problems included the reluctance of some providers to accept Medicaid, and the inability of some clients to cooperate with medical examinations. (*Am J Public Health* 1990; 80:1043–1048.)

Introduction

Adults and children with mental retardation have the same needs as other individuals for preventive health care, episodic care for acute illness, and ongoing care for chronic medical conditions. They often have chronic conditions, physical disabilities, and sensory deficits that are associated with their mental retardation and require specialized services. Some may have acquired conditions, e.g. contractures, related to suboptimal medical management.[1]

Ensuring adequate health services to manage these needs is critical to the success of efforts to maintain persons with mental retardation in the community, yet information concerning the population living in community settings and their requirements for health services is in short supply. The decentralized nature of the community system and the variety of living arrangements which it supports—ranging from sheltered housing with on-site supervision to assistance to persons living with their families or living independently—make it difficult to conduct survey research which depicts the overall population.

National data are available which describe the population living in staffed residential facilities in the community.[2] Comparable data describing retarded persons living with their families or living independently are not available although this is a larger population.

Published studies of the health needs of persons with mental retardation living in community settings report that these individuals have significant health problems,[3–5] but they may be biased toward individuals with the most complex medical conditions. None are based on the experiences of randomly selected subjects, a factor which is of concern given the heterogeneous nature of this population. Two of the published studies are also based on the experiences of a limited number of subjects, most of whom had been previously institutionalized, a factor generally associated with a poorer health status.

Studies of the prevalence of medical conditions among mentally retarded persons living in institutions also report that these individuals carry a burden of chronic disease which is greater than that in the general population.[6–8] Studies describing the health status of institutionalized populations, however, may not be applicable to the population living in the community, particularly to those who have never been institutionalized.

The present study sought to eliminate these potential sources of bias by surveying the health needs of a large sample randomly selected from a population of persons with mental retardation living in a range of settings in the community. Only one-third had been institutionalized previously. Information is provided concerning the prevalence of chronic conditions among these individuals and their concomitant requirements for physician, allied health, and home health services.

Methods

Sample

The study population universe was composed of 1,333 individuals with mental retardation who were receiving services in the community system administered by the Massachusetts Department of Mental Health/Division of Mental Retardation in southeastern Massachusetts. All clients were receiving active case management services and most were receiving additional services including residential and day program services. A sample of 350, stratified by area, was randomly selected for study. Seventeen individuals who did not meet eligibility criteria were excluded: seven lived in other regions, eight had withdrawn their request for services, one was determined not to be mentally retarded, and one was living in a public institution. The final sample consisted of 333 clients.

Data Collection

Data were collected by a client-specific questionnaire that was completed for each client by his or her designated service coordinator. The survey covered a 12-month period. Following training sessions supplemented by a reference manual, the questionnaire was completed primarily from the clients' record which includes their individual service plans. Regulations require a medical assessment for each client soon after the initiation of services and reviewed annually thereafter, and an annual medical assessment that includes a physical examination with a gynecological examination for women. The record also contains the service coordinator's quarterly progress notes which document events relevant to

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the client's major health and other service needs. If the information requested in the questionnaire was not available in the record, the client's program staff or family was contacted to obtain it. Completed questionnaires were obtained for all of the 333 clients in the sample.

Data concerning the prevalence of chronic conditions were validated as follows: a 16 percent random sample was selected from the group who had been institutionalized. Their medical records in the institution were reviewed and compared with the information in this survey. In addition, for seven clients, medical assessments had been completed by their physicians 18 months earlier in another survey conducted by these authors;[7] this information was similarly compared with the survey information.

Data Analysis

Data related to clients' background, demographic characteristics, extent of need for health services, and health service utilization were analyzed using the Statistical Package for the Social Sciences (SPSS). Responses to open-ended questions were content analyzed according to pre-selected categories. In order to determine the adequacy of service utilization, several measures were taken. The project's advisory committee, an interdisciplinary group of professionals with expertise relative to mental retardation, delineated the constellation of physician and home health services that should be available to mentally retarded persons in the community. The services received by a client were judged to be adequate if the client was being seen by the appropriate type of provider given the nature and severity (i.e. level) of the client's medical condition. The analysis of physician services involved an additional step: a physician review panel—composed of an internist/family practitioner in private practice, a family practitioner in a community health center, and an internist practicing in a state residential facility for the mentally retarded—reviewed the client's medical conditions and noted if the current provider was adequate.

Determining Need for Physician and Allied Health Services—The project's advisory committee decided that each client should have an age-appropriate primary care physician (internists for adults, pediatricians for children, family practitioners for either). Specialists, when needed, would provide supplementary care or care in partnership with the primary care physician. Adequate primary care for women included the provision of routine gynecological examinations and pap smears on a regular basis. To quantify the need for specialty physician services, classifications were developed which categorized the clients' chronic medical conditions by level. Each level corresponded to the type of medical provider most appropriate for managing the condition in the community.

- *Level 1 conditions* are managed solely by the client's primary care physician, or by the primary care physician with specialty back-up on an infrequent basis; i.e. less than once a year. For some Level 1 conditions, the extent of the specialist's role might be a one visit consultation for diagnostic purposes with subsequent management provided by the primary care physician. Level 1 care also includes preventive health care, and care for acute episodes of illness. Examples of Level 1 conditions include: occasional or borderline hypertension, and adult onset diabetes with no medication required.
- *Level 2 conditions* are characterized by increased specialty involvement. They are managed by the primary care physician with specialty back-up at least once a year or by a specialist solely at least once a year. Examples of Level 2 conditions include: recurrent otitis media, and peptic ulcer.
- *Level 3 conditions* are managed by a specialist solely whose involvement is ongoing (i.e. several visits a year). Examples of Level 3 conditions include: brittle diabetes in poor control, and uncontrolled grand mal seizures.

The physician review panel reviewed the data provided concerning chronic conditions and determined the following for each condition: 1) the system (e.g. cardiovascular, neurologic) which was involved; 2) whether the condition described was Level 1, 2, or 3; and 3) if care by a specialist was indicated, whether the client was seeing an appropriate kind of specialist. If a specialist was not being seen when necessary, or if an inappropriate type of specialist was following the condition, the service pattern was judged to be inadequate. The panel also reviewed information concerning services provided by allied health professionals and determined whether the allied health professional being seen was an appropriate provider for the condition described.

Determining Need for Home Health Services—Acknowledging that some clients have medical conditions that require ongoing care and treatment at home, the survey assessed the adequacy of home health services in a similar manner. The advisory committee began with the premise that many home health treatments can be safely provided by lay persons including parents, on the condition that the provider is trained and periodically monitored by a nurse. To quantify the need for home health services, the advisory committee developed a list of 16 home health needs commonly encountered in this population and categorized each need by level; each level carried with it a requirement for a specific degree of nursing involvement.

- *Level I needs* are supervised by a nurse who visits the residence on an as needed but regularly scheduled basis. Examples: prescribed topical skin treatments (e.g. lotions), weight control programs, and prescribed eye drops.
- *Level 2 needs* are supervised by a nurse who visits the residence at least once a day. Examples include: medications which require monitoring because of concerns about large dosages or potential side effects; and brittle diabetes, requiring daily insulin injections, urine tests, and diet precautions.
- *Level 3 needs* are attended to solely by a nurse. Examples include: specific nursing treatments (e.g. shunt care), catheter care, and suctioning.

The home health services section of the questionnaire included the list of home health needs developed by the advisory committee. After designating which of these needs applied to their client, service coordinators were asked to describe the client's current care pattern by indicating who attended to these needs.

Direct care by a nurse was considered an adequate care pattern for needs at all three levels. No care or care by the client alone or by an untrained, unsupervised lay person were considered inadequate patterns of care at all three levels.

Results

Sample Characteristics

As shown in Table 1, the sample closely resembled the study population in terms of sex, age, and prior institution-

TABLE 1—Distribution of Population and Sample by Age, Sex and Prior Institutionalization

Characteristics	Population (N = 1333)	Sample (n = 333)
Sex	%	%
Male	54.0	51.7
Female	45.8	48.4
Unknown	0.2	0
Age (years)		
0–17	5.2	5.4
18–21	11.0	10.8
22–44	61.3	61.6
45–64	19.6	20.4
65–98	2.0	1.8
Unknown	0.9	0
Prior Institutionalization		
Yes	33.8	33.6
No	65.6	66.1
Unknown	0.5	0.3

alization. The largest proportion of the study sample (39.3 percent) lived with their families; 36 percent lived in residential programs supervised by the state Department of Mental Health; 5.7 percent in nursing homes, and 3.6 percent in rest homes. Almost 9 percent lived independently with minimal or no supervision. The remaining clients (6.6 percent) lived in various settings including supervised residences for individuals with psychiatric conditions, and family-arranged foster homes. Sixty-six percent had never been institutionalized in a public facility for the mentally retarded. Sixteen percent lived in residential settings with nurses on site and received all or most of their health services, including physician services, within their facility. The remainder, who lived either in family situations, in residences with non-medical staff, or independently, were expected to obtain their health services from providers serving the general population.

Medicaid was the sole source of payment for health services for 65 percent; an additional 14 percent received both Medicaid and Medicare, and 5 percent were Medicare beneficiaries solely. Almost 11 percent were insured by commercial or private insurers; one percent reported "other" payment sources, and for 3 percent the payment source was unknown. Less than 3 percent reported no health insurance coverage.

Care by Primary Care Physicians—Eighty-seven percent of the sample were reported to have a regular physician (who knows the client, manages overall medical care and refers to specialists as needed). For clients younger than age 22, 49 percent were being seen by a pediatrician and another 30 percent by an internist. For older clients, 47 percent were being seen by an internist and 36 percent by a family or general practitioner. The clients who did not have regular physicians tended to be living with their families (50 percent) or living independently (27 percent).

Only 40 percent of the women ages 18 and older were known to have had a gynecological examination provided by either her primary care physician or by a gynecologist within the three-year period preceding the survey. In written comments, 7 percent of women ages 18 and older were noted to have refused gynecological examinations or to exhibit behaviors that made gynecological examinations impossible.

Care by Specialty Physicians—Sixty-two percent of the clients were judged to have one or more chronic medical conditions serious enough to warrant ongoing medical intervention: 30 percent with one condition, 14 percent with two conditions, 12 percent with three conditions, and 5 percent with four or more conditions. Eight-four percent of the chronic conditions reported were being managed by physicians, and 16 percent were managed by allied health professionals (e.g. podiatrists).

The reported prevalence of chronic conditions by systems for all clients is shown in Table 2. In terms of prevalence, the top five types of conditions were neurologic, ophthalmologic, dermatologic, psychiatric-emotional, and musculoskeletal or orthopedic. The high prevalence of neurologic conditions reflects primarily seizure disorders (16 percent). As shown in Table 3, the distribution of conditions by system and level indicates that while many of the conditions were judged manageable by a primary care physician, a substantial amount of specialty involvement is called for. Among the five most prevalent conditions, dermatologic conditions were primarily Level I conditions, while the majority of the neurologic, ophthalmologic, psychiatric-emotional and orthopedic conditions were Level 2, requiring a partnership between the primary care physician and a specialist, or Level 3, needing the ongoing involvement of a specialist. Looking at all conditions together, nearly half were judged to be Level 1, 14 percent were Level 2 and almost 40 percent were Level 3.

The allied health professionals named most frequently as managing chronic conditions were podiatrists who managed 9 percent of the conditions, and optometrists who managed almost 5 percent.

Among the previously institutionalized clients, who had been living in the community an average of 4.6 years, 20 conditions were reported in their institutional records; 13 of these conditions were reported in this survey. Of the seven conditions not reported, two involved hearing loss, one was a refractive error (good vision with glasses), two were cardiac murmurs, one was mild spastic cerebral palsy and one was hypothyroidism. Six new conditions were reported; of these, two were skin irritations requiring topical treatments; the others were a fungal infection of the nailbed, anxiety treated with medication, high blood pressure, and a heart murmur.

Among seven clients about whom physicians had provided information 18 months earlier,[7] 18 conditions were

TABLE 2—Prevalence of Chronic Medical Conditions by System for All Clients in Rank Order* (n = 333)

Condition Types by System	Percent (SE) of Clients	(n)
Neurologic	20.1 (± 2.2)	(67)
Ophthalmologic	18.6 (± 2.1)	(62)
Dermatologic	15.6 (± 2.0)	(52)
Psychiatric/emotional	10.8 (± 1.7)	(36)
Musculoskeletal (orthopedic)	9.3 (± 1.6)	(31)
Cardiovascular	8.4 (± 1.5)	(28)
Ear/nose/throat	7.5 (± 1.4)	(25)
Endocrine	5.1 (± 1.2)	(17)
Gastrointestinal	4.2 (± 1.1)	(14)
Neuropsychiatric**	3.0 (± 0.9)	(10)
Respiratory/pulmonary	2.4 (± 0.8)	(8)
Genitourinary	2.1 (± 0.8)	(7)
Nutritional	1.2 (± 0.6)	(4)
Hematologic	0.6 (± 0.4)	(2)
Gynecologic***	1.9 (± 0.7)	(3)

*31.2% of clients reported more than one chronic condition.
**Those conditions judged by the physician review panel to require *both* psychiatry and neurology services for appropriate diagnosis and treatment.
***Female clients only (161).

TABLE 3—Distribution of Chronic Medical Conditions Managed by Physicians by System and Level

Type of Condition (n)	Level 1 %	Level 2 %	Level 3 %
Neurologic (70)	40	33	27
Ophthalmologic (43)	9	0	91
Dermatologic (26)	73	0	27
Psychiatric/emotional (32)	41	19	41
Musculoskeletal (32)	34	16	50
Cardiovascular (30)	73	17	10
Ear/nose/throat (26)	46	0	54
Endocrine (17)	88	0	12
Gastrointestinal (14)	93	7	0
Neuropsychiatric (10)	10	20	70
Respiratory/pulmonary (8)	75	13	13
Genitourinary (7)	43	14	43
Nutritional (3)	33	67	0
Hematologic (2)	100	0	0
Gynecologic (3)	67	0	33
Multi-system (2)	0	0	100
100% (325)	47 (152)	14 (46)	39 (127)

reported by their physicians, 14 of which were reported in this survey. Of the unreported conditions, three were ophthalmologic (hyperopia/esotropia, light esotropia, and immature cataracts) and one was dermatologic (acne). Two new conditions were reported: chronic constipation and osteoporosis.

Adequacy of Service—As Table 4 indicates, 92 percent of conditions managed by physicians and 89 percent of conditions managed by allied health professionals were judged to be adequately managed. Information concerning conditions for which service utilization was judged to be inadequate is shown in the Appendix.

Need for Home Health Services—Fifty-seven percent of clients were reported to have a chronic condition which carried with it a need for ongoing care and treatment at home. For all clients, level of care was judged to be Level 1 in 28 percent, Level 2 in 27 percent, and Level 3 in 2 percent. The reported prevalence of home health needs is shown in Table 5. Clients who had been cared for by a nurse were found to be receiving services from a variety of agencies, with the highest proportion (15 percent) receiving services from nurses employed by their departmentally contracted residential or day program vendor. Only 3 percent were receiving services from generic home health agencies such as the Visiting Nurses Association.

Of all clients reported to have home health needs, 30 percent were judged to be receiving adequate care while 61 percent were receiving inadequate care. Another 9 percent were receiving "mixed" nursing services; some of their needs were met via an adequate service pattern while other needs received an inadequate pattern of care. For 59 percent of clients with home health needs, licensed nurses were not involved at all, either as direct care providers or as trainers and supervisors of direct care providers.

TABLE 4—Distribution of Adequacy of Service Utilization for Chronic Medical Conditions by Level

Level	Adequate	Inadequate	Unable to Judge	Total
Physician Services				
% Level I	93.4	0.7	5.9	100.0
(n)	(142)	(1)	(9)	(152)
% Level 2	87.0	4.4	8.7	100.0
(n)	(40)	(2)	(4)	(46)
% Level 3	91.3	3.2	5.5	100.0
(n)	(116)	(4)	(7)	(127)
Allied Health Services				
(%)	88.5	9.8	1.6	100.0
(n)	(54)	(6)	(1)	(61)

TABLE 5—Prevalence of Conditions Requiring Care and Treatment at Home for All Clients in Rank Order*

Conditions	Percent of Clients (SE) (n)
Medications requiring monitoring	26.7 (± 2.4) (89)
Diet education and weight control	21.6 (± 2.3) (72)
Prescribed topical skin treatments	18.0 (± 2.1) (60)
Gum care/periodontal disease	7.5 (± 1.4) (25)
Prescribed eye or ear treatments	6.9 (± 1.4) (23)
Chronic constipation requiring "bowel regimen"	6.0 (± 1.3) (20)
"Other" situations requiring care and treatment	3.0 (± 0.9) (10)
Medication IM	2.1 (± 0.8) (7)
Brittle diabetes requiring medication and diet precautions	1.8 (± 0.7) (6)
Total care and repositioning for skin and pulmonary reasons	1.8 (± 0.7) (6)
Recurrent urinary tract infections requiring monitoring	1.5 (± 0.7) (5)
Specific nursing treatments (e.g. shunt care)	1.2 (± 0.6) (4)
Gastrostomy or colostomy care	0.9 (± 0.6) (3)
Catheter care	0.6 (± 0.4) (2)
Suctioning	0.3 (± 0.3) (1)

*Some clients reported more than one condition requiring home treatment.

Other Problems Obtaining Health Services—The data suggested that some clients faced additional problems in obtaining health services. The most frequently cited barrier was a financial one. Of the 267 clients who were Medicaid recipients, almost one-fourth reported one or more instances during the survey period when a health provider refused or was reluctant to serve them because the source of payment was Medicaid.

Characteristics of the clients themselves on occasion created barriers to care. One such characteristic is the ability to cooperate with medical examinations and treatments. For 20 percent of clients, examinations and treatments were able to be completed only when supportive measures were taken to prepare the client, e.g., pre-medication or pre-visits for desensitization purposes. For eight clients, examinations and treatments were able to be completed only when extraordinary measures, such as physical restraints or general anesthesia, were taken. Some clients, while generally cooperative with medical treatment, were said to require supportive measures for specific examinations, e.g. gynecological.

Discussion

It is widely assumed that individuals with mental retardation carry a heavy burden of chronic disease requiring an extensive and highly specialized array of medical services that may be difficult to obtain in the "generic" community health care system. Several studies support this assumption.[3–8] This study, while confirming the relatively high prevalence of chronic illnesses requiring medical intervention, indicates that the vast majority of these conditions

for the group studied can appropriately be managed and are being managed within the community health care system. Further, the study shows that about half of these conditions do not require the services of specialty physicians but can be managed by primary care physicians. Almost 90 percent of clients were connected with a primary care physician who served as their "regular doctor," indicating that they have obtained a portal of entry to the health care system that eludes many individuals in the general population. The findings concerning primary care for women were not as sanguine. Less than 40 percent of the women were known to have had a routine gynecological examination within the three years prior to the survey, thereby missing an important opportunity for prevention and early detection of disease. Given the lack of agreement within the medical community as to the recommended frequency of preventive gynecological examinations for the general population, Massachusetts regulations calling for yearly gynecological examinations for women appear overly rigid. Nonetheless, the provision of gynecological services for women with mental retardation appears to be poorly implemented.

The major service gap identified by this survey concerned the provision of home health care, a subject that has received little attention relative to the mentally retarded. In the judgment of the advisory committee, the optimum management of home health needs in the community service system calls for the involvement of a nurse, who would function most often as a trainer, supervisor, and monitor of lay care givers, and not as a direct care provider. This approach arose from the belief that any home health system established for this population must address the fact that most of these individuals cannot attend to their own health needs. The system must also be designed to accommodate the range of care givers in the community system who have taken on this responsibility. In particular, it must address the fact that staff in many community residential programs are young, inexperienced, and have a high turnover rate. Almost 60 percent of clients with home health needs in the sample studied had not had a nurse involved in their care at all. The remainder of clients had been seen by a nurse but in many instances the nurse acted as a short-term direct care provider and did not function in the expanded role specified by the advisory committee. Generic home health agencies are a potential source of nursing support. Less than 3 percent of the sample receiving nursing services, however, received their care from such agencies. Current regulations governing the financing of home health care by third parties may restrict the availability of services to the resolution of acute conditions and exclude management of the long-term chronic conditions seen among this population. If generic home health agencies are not a realistic source of services for mentally retarded persons in the community system, serious thought must be given to developing alternative ways to provide this essential health supervision.

The other major area of concern involved financing. At the time of the survey, the Commonwealth of Massachusetts offered generous Medicaid benefits, as compared with other states. Yet almost a fourth of the clients reported instances during the survey period when a provider refused or was reluctant to serve them because the payment source was Medicaid. Currently anticipated deep reductions in publicly financed health benefits in Massachusetts and elsewhere will undoubtedly exacerbate these problems. Such changes could place the health of retarded persons at risk. The heavy dependence of this population on publicly financed health programs in part results from the fact that mentally retarded persons as a group are generally denied access to generic health insurance programs because they are assumed to be in need of expensive and complex health services.

Sterling Garrard suggested in 1983 that the level of consumption of health services by persons with mental retardation living in the community is determined by the proportionate mix of three sub-groups.[9] The first is a low consuming group, probably comprising a sizable minority of the population, whose major service requirements are for primary care with an emphasis on health maintenance. The second is an intermediate consuming group, also estimated as a sizable minority, where increased morbidity necessitates more frequent medical encounters. The third group is a high consuming group estimated to be a small minority of the population. Their increased utilization of health services is attributable to severe chronic medical conditions. Their major service requirements are for primary care frequently supplemented by specialty consultations.

The findings of this study support Garrard's suggestion. A recent study of state Medicaid expenditures, which found that the disabled with mental retardation as their primary disability did not have higher than average expenses in most states, also supports Garrard's contention.[10] It is possible that previous studies reporting significant health problems among persons with mental retardation may have been weighted toward Garrard's high consuming group while the two latter studies included individuals from all three groups. Further research is imperative to confirm or challenge these findings; such data are critical to efforts to advocate that persons with mental retardation have access to the health care organizations and insurance programs available to the general population.

Some underreporting of chronic medical conditions undoubtedly occurred in this study. The need to employ respondents who were comparable for all subjects despite their different living arrangements led to the decision to select the service coordinators as respondents despite the fact that they might not have the same first-hand knowledge of the client's health status as the client's physicians, parents, or residential program staff. To minimize underreporting, service coordinators were instructed to report all medical conditions noted in the client's record which contains the report of the client's most recent physical examination (in all cases completed by a physician within the past year) as well as treatment plans to address the client's major needs. If the record information was incomplete, the client's family or residential program staff were contacted. In addition, all prescribed medications used by the client within the past 12 months were noted and checked to ensure that each was linked to a medical condition reported in the questionnaire. These two checks showed that from 65 to 78 percent of all chronic conditions were reported in the record, and that unreported conditions were predominantly Level 1, the least serious conditions.

A second potential source of underreporting was the study's focus on individuals in the community who were receiving services from the state mental retardation agency. There are mentally retarded individuals in every community who are not known to public agencies, although their numbers are difficult to estimate. It is generally understood, however, that those individuals in the greatest need of services are the most likely to turn to the state agency for assistance. For adults, it is unlikely that individuals who were not being served by the state agency would have a higher

prevalence of conditions than clients in this study. The needs of individuals under age 22, however, may be underrepresented, since in Massachusetts, the Department of Education and not the Department of Mental Health, is the primary provider of services for persons with mental retardation under age 22. The small proportion of children under 18 years of age in our survey reflects this fact.

Finally, it is important to note that good health care comprises more than simply obtaining the services of appropriate physicians, allied health professionals, and home health providers. Quality of care considerations, while very important, were beyond the scope of this survey. Some responses also indicated that many clients had encountered particular types of problems when seeking health care, including, at times, negative attitudes toward or poor interpersonal skills with mentally retarded patients. Future research efforts should attempt to describe more completely these and other barriers to obtaining health services in the community. Preventive health services and the availability of ancillary and rehabilitative services such as physical therapy and adaptive equipment services deserve particular attention.

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APPENDIX

Medical Conditions with Inadequate Service Utilization among Mentally Retarded Clients

Physician Services

- One Level 1 skin condition was managed by a psychiatrist; dermatologist recommended.
- One Level 2 cardiovascular condition was managed by a general practitioner; cardiologist recommended.
- One Level 2 psychiatric/emotional condition was managed by a general practitioner; psychiatrist recommended.
- One Level 3 eye condition was managed by an optometrist; ophthalmologist recommended.
- One Level 3 eye condition was managed by an ENT; ophthalmologist recommended.
- One Level 3 neurological condition was managed by a neurologist; neuropsychiatrist (behavioral neurology) recommended.
- One Level 3 neurological condition was managed by a psychiatrist; neurologist recommended.

Allied Health Services

- Two eye conditions were managed by optometrists; recommended provider types were ophthalmologists.
- Three skin conditions were managed by podiatrists; recommended provider types were dermatologists.
- One gastrointestinal condition was managed by a homeopath; recommended provider type was a gastroenterologist, possibly with consultation from a psychiatrist.

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Some of these data were presented at the 113th annual meeting of the American Public Health Association in Washington, DC 1985.

NIH Consensus Statement on Surgery for Epilepsy now Available

A National Institutes of Health (NIH) consensus development statement on Surgery for Epilepsy has been developed by a panel of experts who considered scientific evidence at a Consensus Development Conference held March 19–21, 1990 at NIH.

Single copies of the report, or the two-page summary, may be obtained from: William H. Hall, Director of Communications, Office of Medical Applications of Research, NIH, Building 1, Room 259, Bethesda, MD 20892. Tel: (301) 496-1143.

Homeless and Housed Families in Los Angeles: A Study Comparing Demographic, Economic, and Family Function Characteristics

David Wood, MD, MPH, R. Burciaga Valdez, PhD, MHSA, Toshi Hayashi, PhD, and Albert Shen, MPH

Abstract: We studied 196 homeless and 194 housed poor families in Los Angeles, California to gain an understanding of events that precipitate family homelessness. Both homeless and housed poor mothers averaged 29 years old and were accompanied by two or three children. Three-fourths of both the homeless and housed families had income below the poverty level, and both groups expended almost two-thirds of their income on housing. Mothers in homeless families more commonly reported spousal abuse (35 vs 16 percent), child abuse (28 vs 10 percent), drug use (43 vs 30 percent), or mental health problems (14 vs 6 percent) and weaker support networks. Homeless mothers more commonly came from homes where their parents abused drugs or alcohol (49 vs 34 percent) or more commonly lived outside the home or in foster care (35 vs 25 percent). Homelessness was reported as due primarily to economic pressures of housing costs, but personal and family problems frequently played a contributing role, especially for single parent families. Burdens of increasing housing costs and family dysfunction among housed poor families place many at risk for homelessness. (*Am J Public Health* 1990; 80:1049–1052.)

Introduction

Families make up over one-third of the homeless nationwide, and in many large cities (e.g., New York, Philadelphia, Portland) families represent the majority of the homeless population.[1] Los Angeles has approximately 35,000 to 50,000 homeless persons, about one-third of whom are members of homeless families.[2] The growth of the homeless family population has been attributed to increasing poverty among families combined with rising housing costs[3] and family dysfunction.[4–6]

A number of studies indicate that changes have occurred in the composition of the homeless family population over time and that important geographic variations exist.[1,7–10] Yet, with the exception of one study in Boston,[4] only descriptive studies of the homeless family population are available. To shed more light on the composition of homeless families and gain a better understanding of the precipitating events of homelessness among families, we surveyed 196 homeless families in shelters and compared them to 194 housed, poor families in Los Angeles, California. The health status of homeless and housed children was also assessed and those comparisons are presented elsewhere.[11]

Methods

Homeless families (n = 196) and a comparison group of stably housed, poor families (n = 194) were interviewed during March 1987 through early 1988 in Los Angeles, California. From a universe of 23 shelters serving families in Los Angeles, homeless families were sampled from the 10 largest and busiest shelters. These 10 shelters received 80 percent of all family shelter referrals in Los Angeles during the three months preceding the study. Interviewers visited each shelter at least weekly during the interview period and sampled families from among those who had come to the shelter within the past week. In the five smaller shelters (less than 10 families) a complete sampling of the new families was attempted. In the five larger shelters every other new family was selected for an interview. If after two attempts we failed to locate a family, we approached the next family on the roster.

The housed, poor families were systematically sampled from families presenting to four welfare offices, selected on the basis of the geographic distribution of the "last stable address" of the homeless families. In the welfare offices we screened all presenting families and requested an interview from those who had lived in their current residence at least six months and had received welfare benefits continuously for at least one year. Of the 240 families we sought to interview, 46 refused to participate. There were no differences in age or ethnicity of the mothers who refused compared to the respondents.

Interview and Questionnaire

Structured interviews were conducted with the mothers by trained interviewers and lasted approximately 45 minutes.

Families were categorized as female, single-parent or two-parent families based on whether the mother reported that she was currently with an adult male partner. The mothers were asked if they had been abused in their current or most recent significant relationship, if they or their partner abused drugs or alcohol, and if they or their partner had a history of hospitalization for mental illness. Mothers also were asked if, in her family of origin, there was a history of family violence, drug or alcohol abuse, or mental illness. The quantity and quality of maternal social supports were assessed by documenting the total number of persons the mother could rely on for support, the ages of the supports, and the mother's relationship to the supports.

Family income was a summation of the total income contributed from all family members over the past year and was further assessed by source. The percent of income expended on housing was derived by dividing reported rent and utility expenditures by the average monthly income. For homeless families, we asked them to report the rent and utility costs at their last stable residence (defined as the family having their own place for at least two months' duration).

Housing histories included the number of moves over the past five years and the types of living situations experienced (doubled up with strangers, living in a hotel or motel, living in a car or on the street). Homeless families were asked to describe the events leading to their loss of housing. For each

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narrative, two raters independently identified a primary precipitating event which were then grouped into five categories: 1) *Economic*—such as job loss or loss of AFDC (aid to families with dependent children) grant income; 2) *Housing-related*—such as eviction, failure to pay rent, conversion, or demolition; 3) *Marriage or family relationship* problems; 4) *Violence* and/or drug related; 5) *Natural disaster*. When multiple events were described, the rater sought to identify the initiating or underlying event. For example, where income loss lead to eviction the primary event was classified as "income loss."

Analysis

The homeless and housed samples were compared using simple comparison of means and proportions and two-tailed t-test and Chi-square analysis. We report 95 percent confidence intervals for the differences in means and proportions. Multiple regression as used to test each comparison while controlling for potentially confounding factors of race, maternal education, child sex, child age, and family structure. The qualitative results remained unchanged, so we report the unadjusted comparisons for simplicity.

Results

Demographics and Family Structure

The majority of homeless and housed mothers in our sample were Black, and averaged 29 years of age (Table 1). A larger proportion of the homeless mothers were White. Homeless mothers reported larger families and were less likely to have graduated from high school. More of the homeless families were headed by two parents and and more of the homeless couples than housed couples had been together for over one year (Table 1).

Family Problems and Maternal Social Supports

Homeless mothers reported more spousal abuse and drug abuse by their mates and were more likely to have had a child abuse report opened on the family and more likely to have personally required hospitalization for mental illness

TABLE 1—Demographic Characteristics of Homeless and Housed Women and Families

Characteristic	Homeless Families (n = 196)	Housed Families (n = 193)	Difference of Homeless-Housed (95% confidence interval)
Maternal ethnicity (%)			
Black	57	70	−13 (−22, −4)
Non-Latino White	30	15	15 (7, 23)
Latino	8	11	−3 (−9, 3)
Other	6	4	2 (0, 4)
Percentage of mothers with high school diploma or some college	57	72	−15 (−24, −6)
Percentage of families with two parents (versus single-parent, female-headed)	47	38	9 (1, 19)
Percentage of two-parent families together more than one year	83	62	21 (12, 30)
Mean number of children per family	2.7	2.3	0.4 (0.1, 0.7)
Mean Maternal age	29.5	29.0	0.5 (−1, 2)

TABLE 2—Percentage of Mothers Reporting Violence, Drug or Alcohol Abuse, Criminal Involvement, and Mental Health Problems

Family or Personal Problem Reported	Homeless Families (n = 192)	Housed Families (n = 190)	Difference of Homeless-Housed (95% confidence interval)
Violence			
Mate abuses mother	34	16	16 (9, 25)
Mate involved in criminal activity	29	20	9 (0.4, 18)
Social service file opened for child abuse or neglect	28	10	18 (14, 22)
Drug Alcohol Abuse			
Mate abuses alcohol or drugs	43	30	13 (3, 23)
Mother abuses drugs	32	26	6 (−3, 15)
Mental Illness			
Mate has history of mental illness	15	11	4 (−3, 11)
Mother has history of hospitalization for mental health problem	14	6	8 (2, 14)

(Table 2). Both the homeless mothers and housed mothers frequently reported using drugs such as crack and heroin (32 percent and 26 percent, respectively).

A history of substance abuse and family violence were more often reported for the homeless mothers' own family of origin (Table 3). Over one-third of the homeless mothers reported being placed with relatives or in foster care as children compared to one-fourth of the housed mothers.

Two-thirds of homeless mothers named only one or no adult supports compared to one-half of housed mothers (Table 4). Fewer homeless mothers could rely on their parents for support, and they were more likely to rely on their own minor children for support than were housed mothers.

The single-parent homeless families reported many more relationship problems, such as drug abuse or violence than did the two-parent homeless families (means: 1.9 vs 1.2; Diff = 0.7, 95% CI = .39, .99). Single-parent homeless mothers also reported a mean of fewer adults in their network of social supports than the two-parent homeless families (1.3 vs 2.2, Diff = −.9, 95% CI = −1.34, −.37); and single-parent homeless mothers more often named their minor children as their primary supports than the two-parent homeless families. In contrast, the two-parent homeless families were very

TABLE 3—Percentage of Homeless and Housed Mothers Reporting Problems in Their Own Families of Origin

Problem Reported by Mother	Homeless Families (n = 195)	Housed Families (n = 189)	Difference of Homeless-Housed (95% confidence interval)
Mother's family of origin headed by single, female	40	47	−7 (−17, 3)
Mother's parents divorced or death of parent(s)	43	36	7 (−3, 17)
Mothers parent(s) abused drugs or alcohol	49	34	15 (5, 25)
As a child, mother lived with a relative or in foster care	35	25	10 (1, 19)
Mother was physically or sexually abused by parent(s)	31	21	10 (1, 19)

TABLE 4—Number and Type of Social Supports Reported by Homeless and Housed Mothers

	Homeless Mothers	Housed Mothers	Difference of Homeless-Housed (95% confidence interval)
Mean number of adult supports named by mother (n = 189, 184)	1.75	2.22	.48 (0.11, 0.85)
Percentage of mothers reporting:			
1 or no adult support	66	48	18 (8, 28)
2 or 3 adult supports	26	36	−10 (−19, −1)
4 or more adult supports (n = 196, 194)	8	16	−8 (−14, −2)
Percentage of mothers naming own mother or father as a support (n = 196, 194)	31	65	−34 (−43, −25)
Percentage of mothers naming own child less than 18 years old as a support (n = 165, 167)	37	13	24 (15, 33)

similar to the two-parent housed families on measures of social support and family relationship problems.

Family Income, Housing Costs and Housing Histories

After adjusting family income for family size, over three-fourths of the families in both groups had incomes below the poverty level (in 1987 the poverty level for a family of three was $8940,[12] (Table 5). The income of the housed families came almost exclusively from welfare, whereas only 61 percent of homeless families were dependent exclusively on welfare (27 percent depended on the mother's or father's income exclusively or as a supplement to welfare, and 12 percent reported no income at all). Although AFDC was a primary source of income for homeless families, 43 percent reported losing or being removed from the welfare rolls during the past year.

Families in both the homeless and housed groups reportedly spent or had spent about two-thirds of their monthly income on rent and utilities (means: 67 percent vs 64 percent), and about one-fourth of each group had rent-to-income ratios above 80 percent (Table 5). Only about 15 percent of families in each group had rent-to-income ratios below 30 percent, the upper limit recommended by the federal housing guidelines.[13]

Homeless families reported long histories of housing instability, averaging 3.7 moves in the past year. Over 40 percent of the homeless families reported more than five moves in the past five years. During the past five years over half of the families had doubled-up with others (56 percent), three-fourths had lived in a hotel or motel (76 percent), and one-third had lived on the street or in a car (32 percent).

Although the housed families were chosen on the basis of their recent housing stability, many had experienced housing instability over the past five years. Almost two-fifths (39 percent) of the housed families reported that, sometime during the past five years, they had doubled up with strangers for housing, and almost one-fifth (19 percent) reported that they had lived in a hotel, motel, a car or a shelter. Thus, during the past five years, two out of five housed, poor families had experienced unstable living situations and one in five had been homeless.

TABLE 5—Reported Family Income, Rent Burden for Homeless and Housed Families

	Homeless (n = 196)	Housed (n = 194)	Difference of Homeless-Housed (95% confidence interval)
Mean family income	9,739	8,150	1589 (677, 2500)
Percentage of Families with income below poverty line	82	88	−6 (−13, 1)
Mean rent to income ratio	0.67	0.64	0.03 (−0.7, 0.13)
Mean number of moves over the past five years	5.9	2.3	3.6 (2.8, 4.4)

Precipitating Causes of Homelessness

The majority of homeless families (57 percent) described economic problems (i.e., job loss, loss of AFDC) or housing problems (i.e., eviction) as the primary precipitating cause of their homelessness; one-fourth described personal or family problems, such as a relationship breakup or estrangement from the extended family. Violence or drug problems were described as the primary causes of homelessness by 11 percent of the families. The distribution of precipitating events, however, was quite different for two-parent compared to single-parent homeless families. Two-parent families were more likely to cite economic or housing problems as precipitating events for homelessness than single-parent families (72 percent vs 48 percent; Table 6). Single-parent families were more apt to cite family problems as primary precipitant of homelessness than two-parent families (31 percent versus 16 percent).

Discussion

Previous surveys and earlier studies in various locales describe the homeless family population as predominantly composed of single-parent, female-headed families.[4,5,8,14] Yet almost half of our sample of homeless families was composed of two-parent families, most of whom had been together for over one year. Recent studies in Seattle, Portland, and other cities have similarly found approximately half of homeless families to be two-parent families.[1,7]

Previous homeless studies may have underestimated the proportion of two-parent families because they questioned only shelter directors or other "key" informants, or because two-parent families tend to falsely report themselves as single-parent families in order to qualify for services, including emergency shelter itself.[15] The high prevalence of two-parent families among homeless families has important implications for our public policy of family assistance. Due to categorical qualification criteria, two-parent families are less frequently eligible for family assistance programs such as

TABLE 6—Distribution of Reported Causes of Homelessness in Single-Parent versus Two-Parent Homeless Families

Percentage of Families Reporting Cause of Homelessness	Two-Parent-Families (n = 90)	Single-Parent Families (n = 97)	Difference of Two Parent-Single Parent (95% confidence interval)
Housing problems	34	28	6 (−7, 19)
Economic problems	38	20	18 (5, 31)
Family/relationship problems	16	31	−15 (−27, −3)
Related to drug use or violence	8	14	−6 (−15, 3)

Medicaid, the Homeless Family Assistance Program in California, and AFDC. These policies promote family separation and actually may precipitate homelessness.

Prior to becoming homeless, families were expending such high proportions of their income on housing that they had few resources remaining to meet other basic necessities such as food and clothing.[11] Our sample of stably housed families also reported equally high rent-to-income ratios of 60 to 80 percent, which puts them at great risk for housing loss; many reported doubling up or even experiencing short periods of homelessness over the past five years.

Millions of families nationally, including over two-thirds of all families on AFDC, expend over 50 percent of their family income on housing, and are severely rent burdened.[13,16–18] In almost all states, AFDC grants are well below the poverty level and in many states the entire AFDC grant does not reach the level of the lowest local Fair Market Rent.[19] AFDC grant levels must at least be raised to the federal poverty level, and should ideally be indexed to local fair market rents in order that families not expend more than 30 percent of their grant on housing.

Homeless families frequently experienced discontinuations of welfare assistance for administrative reasons rather than due to increased family income. This caused the family financial hardship and usually played an important role in their loss of housing. Nationwide, as many as 25 percent of welfare families annually experience administrative discontinuations without necessarily any change in their family income.[20,21] Welfare administrative procedures should be modified to promote rather than undermine the economic stability of fragile families and thereby prevent rather than precipitate their loss of permanent housing.

The stress of homelessness, pre-existing family problems, and weak support networks increase the likelihood that homeless families will not obtain access to our fragmented social service system. Family outreach services and comprehensive case management should be offered to assist homeless families to resolve family problems, secure permanent housing, and prevent future homeless episodes.[20,22] The outreach and case management services should include crisis intervention, assistance with program enrollment and benefit maintenance, family counseling, job training, and rehabilitation for drug dependence.

Several issues regarding our sampling strategy should be noted. We may have overrepresented newly homeless families and underrepresented families who have been homeless in the same shelters for longer periods. Further, due to logistical problems in the field and the limited resources of the study, the sampling in some shelters occasionally was reduced to a convenience sample of families. This limitation represents a common problem among published studies in this field.[23] Lastly, the housed, poor family sample may overrepresent Blacks and underrepresent Latinos compared to the overall AFDC population in Los Angeles; the difference resulted largely from our geographic selection of welfare offices to represent the homeless families' neighborhoods of origin.

In summary, the most common contributing factors to the loss of permanent housing among our sample of homeless families were high housing costs and family poverty. Interpersonal problems and social isolation also played a role in the loss of permanent housing, especially among the single-parent families. Housing costs continue to rise and family poverty is worsening, putting millions of American families at risk for losing permanent housing independent of the presence of interpersonal problems.

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Racial Differences in Serum Cotinine Levels among Smokers in the Coronary Artery Risk Development in (Young) Adults Study

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Abstract: Cotinine was measured in the serum of nearly all 5,115 18–30 year old, Black and White, men and women participating in the Coronary Artery Risk Development in (Young) Adults Study, 30 percent of whom reported current cigarette smoking. Ninety-five percent of the reported smokers had serum cotinine levels indicative of smoking (>13 ng/ml). The median cotinine level was higher in Black than White smokers (221 ng/ml versus 170 ng/ml; 95 percent CI for difference: 34, 65) in spite of the fact that estimated daily nicotine exposure and serum thiocyanate were higher in Whites. The difference persisted after controlling for number of cigarettes, nicotine content, frequency of inhalation, weekly sidestream smoke exposure, age, gender, and education. A reporting bias and nicotine intake were ruled out as explanations for the racial difference suggesting that the metabolism of nicotine or the excretion of cotinine may differ by race. Racial differences in cotinine levels may provide clues to the reasons for the observed lower cessation rates and higher rates of some smoking-related cancers in Blacks. (*Am J Public Health* 1990; 80:1053–1056.)

Introduction

Cotinine, a major metabolite of nicotine, is currently considered the best indicator of tobacco smoke exposure.[1] It is specific for nicotine, has a long half-life (15–40 hours), and its level is thought to be directly proportional to the quantity of absorbed nicotine.[2] The quantity of nicotine absorbed by smokers is quite variable, being dependent upon its concentration in the smoke, the individual's smoking pattern, and the smoke pH.[3] Less is known regarding characteristics which influence the circulating levels of nicotine and cotinine or their maintenance in body fluids.[4–6] Such information may assist in the study of nicotine addiction and illnesses attributable to the toxic effects of this drug.

This paper will assess the differences in cotinine levels in a biracial group of young adults who, as determined by self-report, are cigarette smokers. Possible behavioral and metabolic differences which might account for this observation are evaluated.

Methods

The Coronary Artery Risk Development in (Young) Adults Study (CARDIA) is a longitudinal study of the antecedents and risk factors for cardiovascular disease in a cohort of 5,115 Black and White men and women ages 18–30 years at their initial examination in 1985. The sample was recruited to achieve approximately balanced subgroups of self-reported race, gender, education (high school or less and more than high school) and age (18–24 and 25–30 years) in Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. Data were collected on a variety of risk factors as previously described.[7]

This report focuses on subjects who reported, on an interviewer-administered questionnaire, current, regular cigarette smoking of at least five cigarettes per week almost every week for at least three months. Smokers were queried regarding the frequency of inhalation, the number of cigarettes smoked daily, the total number of years of regular smoking, and current as well as past brand choices including questions about cigarette length, flavor, and filter. Information was also collected on cigar, pipe, smokeless tobacco, and nicotine gum use as well as weekly exposure to sidestream smoke.

The amount of tar, nicotine, and carbon monoxide in the cigarette brands was estimated from the Federal Trade Commission (FTC) Report on January 1985.[8] A crude measure of daily nicotine exposure was estimated as the reported number of cigarettes smoked per day × the nicotine content of the reported cigarette brand.

Blood was collected under a standardized protocol. A 1 ml aliquot of serum was frozen and shipped to the Clinical Biochemistry Laboratories at the American Health Foundation. Cotinine was determined by radioimmunoassay using the method of Haley, *et al*,[9] after a modification of the method described by Langone.[10]

A 3.4 percent sample of randomly selected blind duplicates was submitted by the field centers over the entire study period for an external assessment of quality. Cross-classification of the duplicate samples of both smokers and nonsmokers over five categories of cotinine levels (0–13 ng/ml, 14–99, 100–249, 250–499, ≥ 500) yielded a 91 percent (177/194) exact agreement rate. The agreement declined as the cotinine levels increased. Internal quality control was maintained as well; the interassay coefficient of variation was 7 percent.

Thiocyanate (SCN) was also quantitated by the laboratory according to the automated procedure described by Butts, *et al*.[11] SCN is a metabolite of hydrogen cyanide, a toxic gas present in tobacco smoke. It also occurs naturally in certain foods, making it a less specific marker of cigarette

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smoking.[9] The decision to analyze SCN on a subset of samples was made after the start of the examination. Thus, SCN analyses were performed on samples left from the cotinine assays. Although the laboratory selected the subset, their selection criteria were based on the sample cotinine level and not the demographic characteristics of the subjects. SCN was measured on approximately 75 percent of the smokers.

Confidence intervals and statistical tests for the cotinine values are based on the Wilcoxon rank sum procedure. Similarly, Spearman nonparametric correlations were calculated for cotinine and other variables. Correlation coefficients were compared using the Fisher's Z transformation.[12]

A number of linear models were examined to assess the contribution of each of the independent variables to the cotinine measurement. We have chosen to present the multiple linear regression model of the untransformed cotinine because of its simplicity and because the variance of cotinine was reasonably stable over all levels of nicotine exposure. Other models, including log cotinine as the dependent variable, produced similar results. Separate analyses were performed for each of the four geographic areas as a check of the consistency of the major findings. All analyses were performed using the Statistical Analysis System (SAS).[13]

Results

Current cigarette smoking was reported by 1,545 (30 percent) of the 5,115 subjects; characteristics of these smokers have been described previously.[14] One-hundred-twenty-one participants were excluded from analyses (18 were missing the serum cotinine measure, 68 had uncodable cigarette brands, and 35 were regular users of cigars, pipes, smokeless tobacco, or nicotine gum). This left 1,424 (92 percent) smokers available for analyses.

Self-report appeared to be a satisfactory indicator of cigarette smoking when compared to serum cotinine levels. Overall, 95.0 percent of subjects who reported current smoking had cotinine levels > 13 ng/ml[15] (positive predictive value, PPV) while only 5.4 percent of the reported 3,508 non-smokers had cotinine levels above this cutpoint (false negative rate). PPV and false negative rates were slightly lower in Whites than Blacks (PPV: 92.9 percent vs 96.6 percent; false negative rates: 4.5 percent vs 6.5 percent).

Cotinine levels were higher in Black smokers than in White smokers; however, the estimated daily nicotine exposure and thiocyanate levels were higher in Whites than Blacks (Table 1). Black subjects smoked cigarettes of higher nicotine content and more frequently chose mentholated brands. White subjects smoked a greater number of cigarettes per day and reported more frequent inhalation than Black smokers. Fifty-five percent of Black smokers smoked one of two specific brands of cigarettes while only 10 percent of White smokers smoked these brands.

The highest correlation between cotinine and the other exposure measures (cigarettes smoked per day, daily nicotine exposure, thiocyanate, nicotine content of the cigarette) was found with the reported number of cigarettes smoked per day; the correlation coefficient was lower in Black smokers than White smokers (0.46 versus 0.64, $p < .0001$).

Cotinine levels were higher in Blacks in each category of the number of cigarettes smoked per day (significant for all but 21–40 cigarettes category, Figure 1), yet thiocyanate levels were not different between the races except for the upper category in which White smokers had greater levels (Figure 2). A racial comparison of cotinine and thiocyanate levels by daily nicotine exposure produced similar results.

Cotinine levels were 83.3 ng/ml higher in Black smokers than White smokers after adjusting for the number of cigarettes per day, nicotine content of the cigarette, years of smoking, inhalation frequency, weekly sidestream smoke exposure, age, education, and gender (Table 2). No two-way interaction terms yielded significant regression coefficients. Marijuana use and body mass index were also examined and although both yielded significant negative coefficients, only a modest reduction in the race coefficient was observed. The results of a similar regression on thiocyanate showed no association with race.

The race differential was explored further. In each of the four separate linear regression models representing the CARDIA centers, and including all the factors listed in Table 2, the

TABLE 1—Means and Standard Deviations (or Proportions) and 95% Confidence Intervals (CI) for Racial Differences of Selected Variables Among CARDIA Smokers

	Men			Women		
	White (N = 273)	Black (N = 388)	(95% CI for Difference)	White (N = 329)	Black (N = 434)	(95% CI for Difference)
Age (years)	25.4 ± 3.4	24.6 ± 3.8	(0.3, 1.4)	25.0 ± 3.5	25.0 ± 3.7	(−0.5, 0.5)
Education (years)	13.4 ± 2.3	12.2 ± 1.6	(0.9, 1.5)	13.3 ± 2.1	12.6 ± 1.6	(0.4, 1.0)
Cotinine (ng/ml)	210.2 ± 145.1	244.8 ± 156.2	(11.4, 57.8)	176.4 ± 137.6	251.2 ± 175.6	(52.6, 97.0)
(median)	(205)	(219)	(7, 54)	(152)	(222)	(44, 85)
Thiocyanate (μmol/l)*	160.4 ± 46.5	148.0 ± 46.0	(4.2, 20.6)	162.7 ± 53.7	152.2 ± 52.8	(1.5, 19.5)
Nicotine Content of Cigarette (mg)**	0.84 ± 0.25	1.03 ± 0.21	(.15, .23)	0.80 ± 0.27	1.00 ± 0.24	(.16, .24)
Cigarettes per Day	18.1 ± 10.6	10.8 ± 7.2	(5.9, 8.8)	14.9 ± 9.8	10.6 ± 6.7	(3.1, 5.5)
Daily Nicotine Exposure (mg/day)***	14.9 ± 9.7	11.2 ± 8.0	(2.3, 5.1)	11.9 ± 8.8	10.6 ± 7.2	(0.1, 2.5)
Sidestream Smoke Exposure (hrs/wk)	21.0 ± 24.5	19.5 ± 22.4	(−2.2, 5.2)	23.0 ± 25.4	22.1 ± 25.2	(−2.7, 4.5)
Inhaling "Always" (%)	69	57	(5, 19)	76	51	(18, 32)
"Almost Always" (%)	26	24	(−5, 9)	19	27	(2, 14)
Smoking Mentholated (%)	24	87	(57, 69)	34	91	(51, 63)

*WM n = 198; BM n = 320; WW n = 223; BW n = 347.
**nicotine content of cigarette, coded from 1985 FTC report.
***calculated as nicotine (mg) × cigarettes per day.

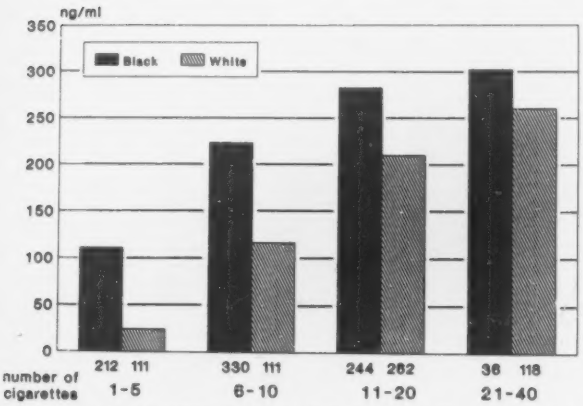

FIGURE 1—Median Cotinine (ng/ml) by Daily Number of Cigarettes Smoked
Numbers directly below bars are sample sizes. Racial comparisons within category of cigarettes smoked are significant ($p < .0001$), except for 21–40 cigarettes category ($p = .34$). Nine White subjects who reported smoking > 40 cigarettes per day were excluded.

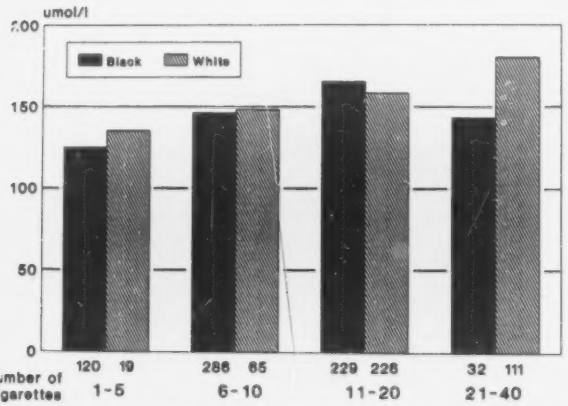

FIGURE 2—Median Thiocyanate (umol/l) by Daily Number of Cigarettes Smoked
Numbers directly below bars are sample sizes. Racial comparisons within category of cigarettes smoked are not significant except for 21–40 cigarettes category ($p < .001$). Nine White subjects who reported smoking > 40 cigarettes per day were excluded. Median thiocyanate is nearly identical to the mean; the median is presented for comparability to Figure 1.

race coefficient was found to be significant and consistent (Birmingham, 72.3; Chicago, 125.6; Minneapolis, 73.5; Oakland, 88.0; all $p < .0001$). The race coefficient was also significant in models representing smokers of mentholated (89.0, $p < .0001$) as well as non-mentholated cigarettes (51.5, $p = .0009$). Highly significant racial differences were also present in all three educational groups (< 12 years, 12 years, > 12 years) examined.

Discussion

In this cohort of smokers, race was strongly and consistently associated with serum cotinine even after adjustment for factors known to affect its concentration. To our knowledge, this finding has only been reported in one other study, as a chance finding[16]; misreport of exposure or metabolic differences were considered as an explanation of this finding in that study. As expected, we found that individual smoking patterns were also important factors in the prediction of cotinine levels in CARDIA smokers, with the quantity smoked most strongly correlated with cotinine in univariate and multivariate analyses.

There are several possible explanations for the higher cotinine levels in Black smokers. First, a reporting bias could account for the difference. A number of studies have shown that smokers tend to underreport their consumption of cigarettes.[17] However, a race differential in reporting appears unlikely (as was also shown in the National Health Interview Survey[18]). Our calculations indicate that all Black smokers would have to smoke nearly two times what they report to eliminate the race differential. Also, thiocyanate levels were equivalent between the races when the quantity smoked is taken into account (except in the 21–40 cigarettes category in which White smokers had higher thiocyanate levels). This also suggests that self-report is comparable between the races. Furthermore, underreporting might be expected to be different in various educational levels: however, we found the race differential in cotinine levels in all educational groups. The race differential in the false negative rate was also examined as a contributor to the cotinine differential. If, among Blacks, a large number of light smokers reported not smoking (false negatives), the average cotinine level reported here for Blacks may be inflated. However, upon examination of all participants considered "smokers" by their cotinine level, similar racial differences in the average cotinine level were observed. None of these findings supports a reporting bias as an explanation for the race differential in cotinine levels.

There are a number of factors in the style of smoking such as depth and frequency of inhalation, unsmoked butt length, and obstruction of filter ventilation which affect exposure.[17] Inhalation differences between the races in CARDIA favored higher nicotine exposure in the White smokers; this was not observed in the cotinine results. The amount of the cigarette which was smoked and the degree of filter obstruction were not ascertained from the CARDIA smokers. Possible differences in these factors could only partially account for higher cotinine levels in Black smokers.

We conclude that neither the number of cigarettes

TABLE 2—Multiple Linear Regression Coefficients of Serum Cotinine on Several Variables in CARDIA Smokers

Variables	β Coefficient (Std Error)
Demographic	
Race	
Black	83.3 (8.9)
White	Reference
Education (years)	−4.9 (2.2)
Age (years)	−0.1 (1.4)
Sex	
Men	−2.9 (7.6)
Women	Reference
Smoking	
Cigarettes per Day	6.9 (0.5)
Number of Years of Regular Smoking	3.8 (1.2)
Nicotine Content of Cigarette (per 0.1 mg)	5.6 (1.6)
Inhalation	
Always	11.3 (11.6)
Almost Always	35.2 (12.7)
Partially, Rarely or Never	Reference
Sidestream Smoke Exposure (hrs/wk)	0.3 (0.2)

$R^2 = 0.23$.

smoked, the nicotine content of the cigarette, nor the frequency of inhalation can account for the observed racial difference in cotinine. It is possible that our adjustment for these or other unknown variables has been inappropriate. For example, since Blacks differed from Whites in the brands they smoked, a particular brand of cigarette may be the culprit. Mentholation might also account for differences. Mentholated cigarettes, smoked by 89 percent of Black smokers but only 30 percent of White smokers, might account for underreporting of inhalation frequency because of the anesthetic effect that menthol provides. A deeper and more frequent inhalation pattern in such smokers might result in a greater nicotine exposure and thus higher cotinine levels. However, cotinine levels, adjusted for the known covariates, were significantly higher in Blacks smoking non-mentholated cigarettes as well as in those smoking mentholated cigarettes. Mentholation does not appear to explain the race differential.

Finally, differences may exist in nicotine absorption or nicotine metabolism between the races. Tolerance to nicotine has been shown to be regulated by genetic factors in mice.[19] In man, ethnic differences are known to exist in the metabolism of drugs and other xenobiotics,[20] of which nicotine is an example. The finding of racial differences in cotinine levels across geographic areas and gender groups are consistent with a genetic mechanism.

Furthermore, the induction of microsomal enzymes might be varied across the two groups or differences in urine elimination[4] of nicotine or cotinine might account for the dissimilarity. Blacks could excrete less unmetabolized nicotine than Whites due to urine pH differences or metabolic induction. Additionally, diet could influence urine output and pH in one racial group. We were unable to address these possible explanations in this study.

The possible association of racial differences in cotinine levels to two public health issues should be addressed. The proposed associations are clearly speculative and suggest avenues for further research. First, cotinine, as an indicator of nicotine exposure, may be a reasonable indicator of nicotine dependence. Consequently, it is intriguing that cessation rates are higher in Whites and Blacks.[21] It is unclear whether Whites are more successful at cessation than Blacks because of a lesser nicotine dependency or other factors such as greater participation in cessation programs. The predictors of these success rates merit further investigation.

Second, higher average cotinine levels in Black smokers correspond with their higher incidence of several tobacco related cancers, particularly among young men.[22] Certainly the overall greater proportion of smokers among Blacks than Whites account for some of this differential, but perhaps not all. The biologic plausibility of such an association remains unclear. Although cocarcinogenic activity has been found for nicotine,[23] it has not been shown for metabolites of nicotine, including cotinine.[24]

In conclusion, considerably higher cotinine levels were observed in Black than in White smokers and this was not explained by reporting bias or nicotine intake differences. The difference in cotinine levels may be due to innate differences between the races in the metabolism or excretion of nicotine or cotinine. The findings may be relevant to understanding racial differences in the risk of nicotine addiction and tobacco-related cancers.

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Apparent Underreporting of Cigarette Consumption among Mexican American Smokers

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Abstract: To determine the accuracy of self-report of cigarette consumption among Mexican American smokers, we compared self-reported cigarette use and serum cotinine concentrations in a sample of 547 participants in the Hispanic Health and Nutrition Examination Survey (HHANES). We defined underreporting of cigarette use as a cotinine to cigarette-per-day ratio of > 0.142 μM/l which represented a substantial discrepancy between self-reported consumption and serum cotinine. Of the 98 men and 97 women who reported smoking one to nine cigarettes/day, 20.4 percent and 24.7 percent, respectively, underreported their cigarette consumption. Underreporting was less common among men and women smoking 10 to 19 cigarettes/day (8.3 percent and 10.8 percent, respectively) and 20 or more cigarettes/day (2.2 percent and 2.9 percent, respectively). Comparison of underreporters to other smokers by demographic characteristics within sex and cigarettes/day categories showed no differences. Differences in cotinine metabolism and extremely efficient smoking are alternative explanations that can not be ruled out with these data. We believe, however, that a proportion of Mexican American light smokers may underreport the quantity of cigarettes smoked per day, and may truly be moderate or heavy smokers. (*Am J Public Health* 1990; 80:1057–1061.)

Introduction

Over the past three decades, epidemiological surveys have relied on self-reported smoking behavior to determine current smoking prevalence, and to quantify tobacco consumption in cigarettes per day.[1–3] Because of possible deception in reporting smoking status, biochemical tests to validate self-reported cessation from cigarette smoking have become necessary measures in evaluating programs to help smokers quit.[4] Studies evaluating the relation between biochemical measurements and self-reported cigarette consumption among known smokers indicate that serum levels of nicotine and of its principal metabolite cotinine plateau above 20 cigarettes per day.[5,6] Behavioral factors related to the way a cigarette is smoked may be more important in maintaining a smoker's nicotine level than the actual nicotine delivery of a given cigarette brand as determined by a cigarette smoking machine and reported by the Federal Trade Commission.[7]

Regional and national surveys have consistently shown that Mexican Americans differ in their smoking behavior when compared to Whites who are not Hispanic.[8–13] Men of both groups smoke in similar proportions and smoking rates are higher among White women, but Mexican American smokers report an average of 10 to 12 cigarettes per day compared to 19 to 23 for Whites and 14 to 15 cigarettes per day for Blacks.[13,14] Because the proportion of light smokers among Mexican Americans is substantially greater than among Whites, we hypothesized that underreporting may partly account for this difference.

To compare self-reported cigarette use with a biological marker of smoking behavior, we evaluated the serum cotinine levels in 547 Mexican American smokers who participated in the Hispanic Health and Nutrition Examination Survey (HHANES) in the Southwestern states.

Methods

Southwestern HHANES

The Mexican American portion of HHANES was conducted in the Southwestern states of Arizona, California, Colorado, New Mexico and Texas between 1982–1983 by the National Center for Health Statistics (NCHS). A stratified probability sample of the civilian, noninstitutionalized Mexican American population living in these states was generated using a four-stage process.[15] HHANES included both a household interview and a medical examination, conducted in either Spanish or English. Data on smoking behavior and demographic information were collected. A total of 4,735 Mexican Americans age 20 to 74 years were selected for the survey, with 3,324 (70.2 percent) completing the physical examination portion where blood was drawn. Details of the sample design, operational plan, and quality control procedures are described elsewhere.[15]

The Cotinine Sample

The study population in this report is a subsample of the examined HHANES Mexican American population. Eligibility was based on current smoking status and the availability of 0.5 ml of frozen serum in 1986. Stratifying by sex (male/female) and the number of self-reported cigarettes smoked (1 to 9, 10 to 19, ≥ 20), persons were randomly selected from those with available serum in order to have 90 to 98 subjects per cell. Due to the limited number of women smoking 20 or more cigarettes per day, only 70 persons could be selected for that cell. Table 1 shows the distribution of the cotinine sample in the six cells with the corresponding total number of examined Mexican American HHANES smokers in each cell. The proportion of persons by cell selected from the total sample ranged from 44 percent to 80 percent.

Cotinine Analysis

Measurement of the concentration of cotinine in the serum of persons who reported smoking more than five cigarettes per day was performed using automated capillary gas chromatographic techniques described in detail in other publications.[16,17] Cotinine, principal metabolite of nicotine,

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TABLE 1—Demographic Characteristics of Sample Selected for Cotinine Measurements from Mexican American Smokers in Southwest HHANES

	Self-Reported Cigarettes per Day					
	1 to 9		10–19		≥ 20	
	Men	Women	Men	Women	Men	Women
HHANES denominator (n)	221	197	153	123	207	87
cotinine sample (n)	98	97	96	93	93	70
% age (years):						
20–39	62	61	55	47	46	51
40–74	38	39	45	53	54	49
mean age (years)	37.9	37.6	39.6	41.3	43.3	41.6
% with education (years):						
0–11	72	64	68	63	66	61
≥ 12	28	36	31	37	34	39
mean education (years)	7.7	8.9	8.6	86	9.0	9.1
% acculturation tertile						
low	45	31	24	20	32	19
middle	35	34	30	38	37	26
high	20	35	46	42	31	56
% with income						
below poverty	36	31	34	33	32	34
at or above poverty	64	69	66	67	68	66
% with blood drawn:						
in morning	58	58	62	58	63	54
in afternoon	42	42	39	42	37	46
mean cigarettes/day	4.1	4.2	12.7	11.0	24.0	24.3

has a half-life averaging 19 hours, compared to a half-life of two hours for nicotine.[18,19] There are no data on possible ethnic or racial differences in the metabolism of nicotine among habitual smokers.

Excess serum on HHANES participants was stored at the Centers for Disease Control laboratory at a temperature of minus 70 degrees Celsius. Samples were shipped to San Francisco for cotinine assessment on dry ice and thawed prior to analysis. Cotinine analyses were completed within five years of serum collection, and concentrations of cotinine in frozen serum have been found to be stable over at least a five-year period (unpublished data, N.L. Benowitz). Serum cotinine concentration from smokers reporting one to five cigarettes per day was assayed by the same capillary column gas chromatography method, but modified with a sample concentration step to increase sensitivity. Standards of 0.001 (0.2), 0.003 (0.5), 0.006 (1.0), 0.028 (5.0), and 0.057 μM/l (10 ng/ml) were used. The limit of detection was about 0.001 μM/l (0.2 ng/ml). Results from these two methods for measuring cotinine on the same sample are identical.

Based on data from studies[20] conducted with heavy smokers (average of 36 cigarettes per day) who were restricted to 15, 10, and 5 cigarettes per day, a maximum serum cotinine level of 0.142 μM/l (25 ng/ml) per cigarette smoked was achieved by these extremely efficient smokers. This corresponds to an intake of nicotine of approximately 3 mg per cigarette. Thus, we have defined underreporting of self-reported cigarette consumption as a serum cotinine per cigarette value exceeding 0.142 μM/l (25 ng/ml) per cigarette. In support of this cutoff are the data of Shiffman, *et al*,[21] who measured cotinine levels in smokers who habitually smoke no more than five cigarettes per day. The average cotinine level per cigarette smoked was 0.08 μM/l (SD = 0.067) (14.0 ng/ml; SD = 11.8), which is nearly identical to the 0.07 μM/l (SD = 0.031) (12.5 ng/ml; (SD = 5.4) value for more dependent smokers reported by Benowitz and colleagues.[19]

Data Analysis

Data were analyzed using SPSSX.[22] Comparisons of proportions were made with chi squares and means were compared by t test and analysis of variance. An acculturation scale modified from Cuellar and colleagues[23] was used to divide the population into low, middle, and high tertiles for the purpose of these analyses. The poverty threshold defined in 1982 dollars by the Bureau of the Census was used to categorize the sample by income.[24]

Results

The demographic characteristics of the 547 Mexican American smokers are shown in Table 1 stratified by sex and self-reported cigarettes per day. This sample of smokers is not representative of Mexican Americans in the Southwest US because of the criteria used for selection. Light smokers (1–9 cigarettes/day) were younger than moderate (10–19 cigarettes/day) and heavy smokers (≥ 20 cigarettes per day). Acculturation scores were lower among men compared to women for each category of smokers and among light smokers compared to moderate and heavy smokers. Distribution by years of education completed, income below or above the poverty level, and the time blood was drawn were similar by sex and category of smokers.

Data from smokers who underreported cigarette consumption, as defined by a cotinine per cigarette ratio exceeding 0.142 μM/l (25 ng/ml) per reported cigarette, as shown in Table 2. Of the 98 men and 97 women light smokers, 20 (20.4 percent) and 24 (24.7 percent), respectively, exceeded 0.142 μM/l per cigarette with a mean cotinine per cigarette almost twice the threshold. Mean cotinine levels for light smokers were 4.3 times greater for men and 4.9 times greater for women who underreported than among the other smokers. Among moderate smokers the proportion underreporting cigarette consumption decreased to 8.3 percent for men and 10.8 percent for women. Heavy smokers were the least likely to be classified as underreporters with only two men (2.2

TABLE 2—Serum Cotinine in Mexican American Smokers: Underreporters and Other Smokers

	Self-Reported Cigarettes/Day					
	1 to 9		10–19		≥ 20	
	men	women	men	women	men	women
total n	98	97	96	93	93	70
Percent underreporters	20.4	24.7	8.3	10.8	2.2	2.9
Mean cotinine (μM/l)	1.018	1.206	2.055	2.039	2.909	3.108
Mean cotinine/cigarette	0.255	0.270	0.185	0.196	0.145	0.155
Percent other smokers	79.6	75.3	91.7	89.2	97.8	97.1
Mean cotinine (μM/l)	0.238	0.242	0.943	0.774	1.349	1.410
Mean cotinine/cigarette	0.049	0.054	0.073	0.069	0.059	0.064

percent) and two women (2.9 percent). Comparison of the demographic characteristics of the 66 smokers classified as underreporting cigarette consumption to the other smokers by smoker category and sex show no differences by any of the variables examined including acculturation score. None of the 66 smokers classified as underreporters used cigars, pipes, or smokeless tobacco.

Among the underreporting light and moderate smokers, 36 indicated that they smoked a greater number of cigarettes in the past than at the time of the survey. If this number is used as an alternative value for self-reported consumption, then the proportion of underreporters among self-reported light smokers would decrease to 6.1 percent for men and to 15.5 percent for women. A similar decrease in underreporters would occur among moderate smokers to 3.1 percent of men and 7.5 percent of women. None of the four underreporting heavy smokers reported smoking a greater number of cigarettes in the past than at the time of the survey.

In order to quantify the number of cigarettes underreported by these smokers, an average minimum increase in serum cotinine of 0.057 μM/l (10 ng/ml) per cigarette smokes was estimated to occur.[20] A range of underreported cigarettes per day was derived from the expected minimum increase (0.057 μM/l) and the expected maximum (0.142 μM/l) increase in serum cotinine. Among the men and women light smokers, we estimated that they were actually smoking from 3 to 14 and from 4 to 17 more cigarettes per day, respectively. Among moderate smokers the estimated range of additional cigarettes smoked per day was 3 to 25 for men and 4 to 26 for women.

The average serum cotinine of the Mexican American smokers in this sample are shown in Table 3 by sex and smoker category. The overall values for moderate and heavy smokers are somewhat lower than the average level of 1.47 μM/l (259 ng/ml) reported among 450 smokers of 10 or more cigarettes per day who were volunteers at the blood banks in Bergen County, New Jersey (ethnicity not stated, but presumably mostly Whites).[6] Pearson correlations between cotinine levels and self-reported cigarettes per day are weak for moderate and heavy smokers and stronger for light smokers. Analysis of variance showed that serum cotinine increases with number of cigarettes smoked per day ($F(2,545) = 104.7$, $p < .0001$), age ($F(1,432) = 8.5$, $p = .004$), and acculturation level ($F(2,545) = 3.5$, $p = .03$) related. Sex, educational level, poverty status, and time blood was drawn did not have a consistent influence on cotinine concentration.

Discussion

The measurement of cotinine concentration in blood, urine, or saliva has been used predominantly to verify self-reported smoking status among persons claiming to have quit using cigarettes.[25–29] Use of cotinine measurements to evaluate underreporting in the quantity of self-reported cigarettes smoked per day has not been previously conducted. Applying this method to analysis of a sample of HHANES Mexican American smokers indicates that at least 20.4 percent of men and 24.7 percent of women who reported smoking less than 10 cigarettes per day may be underreporting their actual consumption.

TABLE 3—Serum Cotinine (μM/l) by Demographic Characteristics and Cigarette Consumption in HHANES Mexican American Smokers

		Self-Reported Cigarettes/Day					
		1 to 9		10–19		≥ 20	
		men	women	men	women	men	women
total n		98	97	96	93	93	70
Mean cotinine (μM/l)		0.40	0.48	1.03	0.91	1.38	1.46
Correlation to cotinine	r	.51	.50	.25	.20	.19	.25
	p	<.01	<.01	<.01	<.03	<.04	<.02
Age (years)							
20–39		0.37	0.37	1.07	0.84	1.27	1.40
40–74		0.44	0.65	0.99	0.98	1.48	1.52
Acculturation							
low		0.48	0.28	0.97	0.83	1.35	1.38
middle		0.32	0.63	1.02	0.93	1.35	1.17
high		0.35	0.52	1.07	0.93	1.46	1.62

We initiated this research to validate biochemically the finding in the Southwest HHANES (and other surveys) that Mexican Americans are mostly light or moderate smokers. Our findings support an overall lower level of dependence on nicotine as compared to another large community sample of non-Hispanic smokers.[6] However, a subset of those Mexican American smokers who report less than 10 cigarettes per day are more dependent on nicotine than the self-reported number of cigarettes would indicate. If a substantially greater proportion of Mexican American smokers are moderate or heavy smokers, then the related health risks for cardiovascular disease and cancer are greater than that determined by self-report. This could also have implications for planning and conducting smoking cessation interventions among Mexican Americans. The need to intensify ongoing community interventions based on culturally appropriate self-help methods[30] or to complement this with individualized or physician-mediated interventions must be considered since many Mexican Americans may not be light smokers.

The proportion of underreporting among light and moderate smokers was reduced by more than half when the maximum number of cigarettes smoked in the past was used in place of the self-reported number at the time of the survey. This may reflect the desire among many smokers to smoke less and the accurate reporting of past history may actually represent current habits. Regardless, the maximum number of cigarettes smoked per day in the past may be an important additional measure of self-reported smoking behavior among Mexican Americans.

Coultas and colleagues recently found discrepancies between self-reported and validated cigarette smoking in a community survey of New Mexico Hispanics.[31] Their study found that among former smokers older than 18 years of age, the proportion with a salivary cotinine greater than 0.11 μM/l (20 ng/ml) (defined as current smoker) was as high as 21.4 percent. Our study expands on these findings in that we are estimating a similar rate of apparent underreporting in self-reported number of cigarettes consumed among Mexican American light smokers. The substantial level of inconsistencies between the number of self-reported cigarettes smoked and the results of cotinine concentrations may also reflect a tendency among Latinos to give socially desirable responses.[32]

The use of a cotinine per cigarette ratio exceeding 0.142 μM/l (25 ng/ml) as the cutoff to define underreporting is conservative. In another study, nine heavy smokers (average 36 cigarettes per day) were restricted to five cigarettes per day and only two were able to achieve cotinine to cigarette ratios exceeding 0.142 μM/l per cigarette.[19] In contrast, people who are carefully documented as smoking five or fewer cigarettes per day have intakes of nicotine and levels of cotinine per cigarette similar to heavy smokers during unrestricted smoking.[20] Considering these data, we conclude that in setting the criterion at 0.142 μM/l per cigarette we are probably underestimating the prevalence of possible underreporting.

It is possible that some underreporters in HHANES were extraordinarily efficient in smoking cigarettes, but this is unlikely to fully explain our results given that heavy smokers with restricted access to cigarettes were unable to achieve comparable cotinine levels. Another possible alternative explanation for our findings involves ethnic or racial differences in the metabolism of nicotine and clearance of cotinine. Wagenknecht and colleagues[33] have proposed that higher serum cotinine levels among Black compared to White smokers (despite fewer self-reported cigarettes consumed per day) were due to racial differences in the metabolism of nicotine and not to a reporting bias. If Mexican Americans metabolize nicotine more slowly, our results would be partly accounted for without underreporting. Alternatively, light and moderate smokers for any ethnic group may have a much slower metabolism of nicotine or a slower clearance of cotinine than the average habitual smoker.

Several limitations in this study need to be considered. Although HHANES results are generalizable to Mexican Americans in the Southwestern states, our study selected smokers based on availability of frozen serum and may not apply to all smokers. Given that 42 percent of HHANES Mexican American smokers reported smoking less than 10 cigarettes per day, the highest estimates of underreporting apply to a substantial proportion of this population. However, it is possible that if smokers had been informed that a biochemical validation test would be conducted, underreporting would be less frequent. The HHANES Puerto Rican and Cuban American samples were not evaluated in this study, but the proportion of light smokers in those Latino subgroups is less than among Mexican Americans, and thus underreporting may be less common. Finally, our observations in Mexican Americans may be a phenomenon of all light cigarette smokers' self-reporting on quantity independent of ethnicity. In fact, Warner[34] has suggested that survey reports of cigarette consumption underestimate consumption estimates based on production and sales. A more recent comparison concluded that there was no apparent increase in underreporting cigarette smoking in surveys since 1975.[35] The magnitude of underreporting is greater among light smokers, and thus studies using cotinine measurements and applying the method described in this paper may be more important in these populations.

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NIH Consensus Development Conference Announced on Hyperparathyroidism

Hyperparathyroidism is increasingly being recognized in asymptomatic patients, as a result of widespread use of multiphasic screening tests that lead to detection of hypercalcemia. Physicians are often uncertain about the diagnosis and management of patients with subtle or absent signs and symptoms but with a clear biochemical diagnosis of hyperparathyroidism. Especially difficult are decisions about indications for surgery and how patients should be monitored to detect silent organ damage, particularly progressive bone loss.

Because of these issues, the National Institute of Diabetes and Digestive and Kidney Diseases and the Office of Medical Applications of Research at the National Institutes of Health will co-sponsor a consensus development conference on Diagnosis and Management of Asymptomatic Primary Hyperparathyroidism. The meeting will be held October 29–31 at NIH, Masur Auditorium, in Bethesda, MD.

To register for this conference or to obtain further details, contact: Conference Registrar, Prospect Associates, Suite 500, 1801 Rockville Pike, Rockville, MD 20852. Tel: (301) 468-MEET.

Demographic Characteristics, Risk Behaviors, and HIV Seroprevalence among Intravenous Drug Users by Site of Contact: Results from a Community-wide HIV Surveillance Project

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Abstract: We investigated differences in behaviors important for human immunodeficiency virus (HIV) transmission and HIV antibody status among 927 recent needle users enrolled in a multi-site HIV surveillance project in Worcester, Massachusetts. Subjects were enrolled at drug abuse treatment centers, other clinical sites, and a men's jail. Subjects at drug treatment centers reported less risky injection practices unexplained by demographic variables. Risky sexual practices were in general reported more frequently by men at the jail than men at other sites. However, HIV status showed little relation to enrollment site. These results have implications both for targeting of acquired immunodeficiency syndrome (AIDS) prevention programs to needle users not in drug abuse treatment and for potential selection bias in studies of intravenous drug users. (*Am J Public Health* 1990; 80:1062–1067.)

Introduction

Intravenous drug users (IVDUs) who share injecting equipment play an important role in the transmission of the human immunodeficiency virus type 1 (HIV-1), the causative agent of the acquired immunodeficiency syndrome (AIDS).[1] There is some evidence that IVDUs are not a homogeneous group, and that sub-populations exist with different risks of infection with HIV. Watters and Cheng described two sub-populations of IVDUs in San Francisco: the first was in somewhat regular contact with drug abuse treatment programs, while the second, contacted on the street and in jail, did not interact routinely with drug treatment.[2] Distinction between sub-populations of IVDUs is important both for program planners who may need to target programs differently for different sub-populations, as well as for epidemiologists and other researchers who need to understand the types of selection bias introduced by studying a particular sub-population.

HIV infection is a function both of risky behavior and of the probability that persons with whom the behavior is practiced are infected. Seroprevalence differences may not reflect differences in risky behavior in areas where HIV has been introduced relatively recently. This report investigates differences in risky behaviors and HIV seroprevalence in IVDUs enrolled at multiple sites in a community-wide AIDS prevention program. The data were collected as part of a surveillance system which monitors HIV infection and related behaviors among IVDUs and their sexual contacts in Worcester, Massachusetts.[3] Worcester has an IVDU population estimated in 1988 at about 4,000 and from 1983–86 was the site of a significant epidemic of delta hepatitis.[4] Since 1987, public health interventions targeted at IVDUs which build upon and are coordinated with the ongoing hepatitis B prevention program have been collaboratively planned by members of the Worcester AIDS Consortium, including the city health department, drug abuse treatment facilities, correctional facilities, community health centers, and support and advocacy programs for persons with HIV infection.

Methods

Study enrollment began at the men's county jail in September 1987, followed by the city health department's hepatitis B and HIV clinic and one community health center in December 1987, the three drug abuse treatment centers in February 1988, and the second community health center and a clinic for sexually transmitted diseases run by the State Department of Public Health in April 1988. Interviews completed by the end of December 1988 are included in this study.

Interviews with a short, standardized encounter form are conducted by project staff who are not involved in the care or treatment of the individual. At the three drug abuse treatment centers (a therapeutic community, an inpatient 21-day methadone detoxification facility, and an outpatient clinic providing methadone services and drug-free counseling), the encounter form is administered routinely at admission. At one of the community health centers it is administered routinely to all high-risk clients who are referred for HIV counseling. At those sites where use of the encounter form is part of the client's record, consent is obtained for release of encounter form data to the study. At all other sites, consent is obtained prior to administration of the encounter form. At the men's jail an AIDS educational session, at which attendance is voluntary, is given once a week by a team from the City Department of Public Health.* At the end of the session, the inmates are informed about the study and asked to participate.

Information collected in the encounter interview includes sociodemographic variables, drug use and social behaviors, and results of previous HIV antibody testing (see appendix). Subjects are asked about relevant behavior during their lifetime and during the most recent period of at least one week and not more than three months of "free living," when not in any institutional setting. The main drug use and sexual behaviors reported in this paper are those reported for this "risk period" and are referred to subsequently as "recent" behaviors.

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*Noone S, Birch F, Sereti, Santana L, Patashnik B, Murphy B, Lewis B, Brady P, Sullivan J: A comprehensive prison program for AIDS risk reduction. Abstracts of the IV International Conference on AIDS, Stockholm, June 1988.

At all sites, voluntary HIV antibody testing in conjunction with pre-test counseling is available following the interview. Initial serological screening is carried out using Food and Drug Administration (FDA)-approved commercial ELISA (enzyme-linked immunosorbent assay) kits. Each sample which tests positive by the ELISA is confirmed using a Western blot test.**[7]

In this report we use only data from the first contact of each individual with the Worcester program. Cross tabulations, stratified by sex, were used to assess between-site differences in demographic variables, drug and sexual behaviors, and HIV status.

In order to assess site-specific differences controlling for demographic variables, logistic regression modeling[6] was performed on HIV antibody status and four recent behaviors: sharing of injection equipment; use of bleach to clean injection equipment by those who reported sharing; sexual contact with two or more partners (for females, male partners only were considered); and use of condoms during sex among those with two or more sexual partners. All behaviors were dichotomized (yes or no).

Interpretation of the current HIV antibody test results is complicated by the tendency for those who had previously been tested not to be retested, particularly if they had a positive test result. Therefore, we chose to use the cumulative test results of those ever tested, derived from the results of testing in the Worcester program, and self-reported results of previous HIV antibody tests for individuals not tested in the Worcester program. Separate regression models were run for males and females. Goodness of fit of the logistic models was assessed using the Hosmer-Lemeshow statistic.[6]

Results

Participation rates were as follows: about 50 percent of those attending the educational sessions at the jail; about 85 percent of clinic patients referred for counseling; and over 90 percent of those admitted to the drug treatment centers. The participation rate at the jail is an underestimate, as many prisoners were observed to attend two or more of the sessions before deciding to participate. In the period ending December 31, 1988, 2,174 individuals had been enrolled in the project, 927 (43 percent) of whom reported recent ("risk period") needle use.

Characteristics of recent needle users by site of initial study enrollment are shown in Table 1 (males) and Table 2 (females). On transsexual individua! has been omitted from analysis. Clinic sites have been grouped: most were enrolled at health department clinics.

Significant differences between the enrollment sites were found for almost all demographic variables, both among males and females. Among males, the youngest needle users were enrolled at the jail. In both sexes the oldest needle users and those who had been injecting for the longest time were enrolled at the outpatient drug treatment center. The drug abuse treatment centers attracted lower proportions of racial and ethnic minority members, and higher proportions of high school graduates. Those enrolled in the residential drug abuse treatment centers were more often resident outside Worcester than those at the outpatient sites.

Drug use behaviors also differed significantly by site of enrollment. Needle users entering drug abuse treatment reported the highest frequency of injection but the lowest proportion of sharing and sharers were more likely to have used bleach recently to disinfect their injection equipment. Among males, several other risky drug behaviors were also reported less frequently at drug treatment centers: visiting a "shooting gallery" and sharing injection equipment in New York City.

Less marked differences were found in sexual behavior by enrollment site. Among males, as the percentage reporting homosexual partners were very low, these have been combined with heterosexual partners for these analyses. Men at the jail were more likely to report multiple sex partners during the "risk period," and those with multiple partners were less likely to have used condoms.

Overall, 60 percent of males and 37 percent of females were tested for HIV antibody in the program, with lower percentages tested at the drug treatment centers. An additional 20 percent of males and 33 percent of females had been tested prior to enrollment in the Worcester program. The cumulative percentage ever reporting a positive test result was slightly lower at the inpatient and residential drug abuse treatment sites than at the other sites.

Logistic regression modeling controlling for demographic variables indicated that the odds of not sharing injection equipment were higher at all drug abuse treatment sites in comparison with the clinics (Table 3). In comparison with men at the jail, men at all three drug abuse treatment sites were less likely to share and confidence intervals (now shown) excluded unity. In both sexes, bleach use among needle sharers was higher in all drug abuse treatment center groups than the clinic group.

Sexual behaviors in the drug treatment groups differed from those at the clinics but the confidence intervals included unity (Table 3). However, men at the jail were more likely than men entering drug abuse treatment to report two or more sexual partners; among men with multiple partners, those at the detoxification center were more likely to use condoms than men at the jail and these confidence intervals (not shown) excluded unity.

In contrast with the between-site differences in drug and sexual behaviors, cumulative HIV status differed relatively little by enrollment site (Table 3). In an effort to explain the lack of congruence between the serological data and the risk behaviors, we investigated whether there were differences in risk behaviors between those ever and never tested at the drug abuse treatment sites (at the other sites too few had never been tested). We also examined the relation between risk behaviors and serological status among those who had been tested, stratifying by site of enrollment. No significant associations or consistent trends were found, although small numbers of seropositives at some sites limited the power of these analyses.

Discussion

We found significant differences by enrollment site in demographic characteristics and drug use behaviors of recent needle users. Needle users entering drug abuse treatment tend to be of higher socioeconomic status, and include fewer racial-ethnic minority members than those at other sites. Among the drug abuse treatment facilities, those offering

**Alexander SS, Chang-Chi T, Ting RL, Bodman AJ: Western blot analysis of human T-cell lymphotropic virus III proteins using an avidinbiotin system. Abstracts of the Annual Meeting of the American Society for Microbiology, 1985 (March): 297.

TABLE 1—Demographic Variables, Behaviors, and HIV Antibody Status of Male Recent Needle Users (n = 678), by Enrollment Site

Variables	Drug Treatment Centers			Other Sites		
	TC (n = 66) %	Detox (n = 211) %	OP (n = 113) %	Clinics (n = 33) %	Jail (n = 255) %	Total (n = 678) %
Age 30 years or over	64	62	80	70	44	59
Race						
Hispanic	5	5	19	18	29	17
Black	9	8	4	18	8	8
Other	86	87	77	64	63	75
High school graduate	84	74	75	64	53	67
Married	18	31	48	45	56	43
Worcester resident	19	17	64	76	64	46
Prior drug treatment	44	38	28	27	32	34
Prior jail	64	44	55	67	83	64
Injected >5 years	67	61	89	67	56	64
Drugs injected (ever)						
heroin	94	96	100	82	84	91
cocaine	94	89	81	97	93	90
Recent Behaviors						
Daily needle use	75	90	95	50	67	79
Shared equipment	70	61	65	81	85	73
Total sex partners						
None	24	16	14	27	11	15
One	41	48	65	36	43	48
2 or more	35	36	21	36	46	37
Recent Drug Behaviors Among Sharers Only	(n = 46)	(n = 129)	(n = 73)	(n = 26)	(n = 215)	(n = 489)
Frequency of sharing						
daily	13	27	38	33	55	40
1–6/week	52	33	48	44	25	34
<1/week	35	40	14	22	21	26
Cleaned with bleach	59	67	66	46	43	55
Shared in NYC	13	2	4	15	16	10
Visited a shooting gallery	19	20	17	38	32	26
Recent Sexual Behaviors if Multiple Partners	(n = 22)	(n = 75)	(n = 24)	(n = 12)	(n = 112)	(n = 245)
Used condoms	5	32	25	27	12	19
Sex with prostitute	32	29	27	25	25	27
HIV Antibody Tests	(n = 66)	(n = 211)	(n = 113)	(n = 33)	(n = 255)	(n = 678)
Currently tested	26	49	6	88	98	60
Positive result	0	9	29	21	23	18
Ever tested	74	74	51	97	98	80
Positive result	6	15	24	22	23	19

TC = Therapeutic Community;
Detox = Detoxification Facility;
OP = Outpatient Services

residential and inpatient services attract more young, unmarried clients from outside Worcester than the outpatient clinic. Clients entering outpatient drug abuse treatment are older and have longer injection histories than those entering residential drug abuse treatment, in part because most receive methadone services which require the client to have used heroin for at least one year prior to entry into treatment.

Our results suggest that needle users entering treatment have significantly lower levels of risky drug behaviors, including sharing of injection equipment and not using bleach for disinfection, than those at other sites. In spite of more frequent needle use among those entering drug abuse treatment, safer practices appear to be followed.

Among men, those at the jail report multiple sexual partners more frequently than those at the drug abuse treatment centers, and are less likely to use condoms than those at two of the drug treatment sites. In spite of these behavioral differences, there is little difference in HIV status, but testing was not universal.

We were not able to explain the lack of congruence between the serological data and the risk behaviors. Selection bias (selective testing at the drug abuse treatment sites of those with more risky behaviors) did not explain this finding. It seems more likely that the explanation lies in the cross-sectional study design and the use of prevalent rather than incident infection. For many study subjects, HIV infection may have occurred prior to the recent "risk period" for which risk behaviors were measured. For example, in a study of homosexually active males reported by our group, both prior and recent frequently of risky behavior were independently associated with HIV seroprevalence.[7] We were not able to carry out similar analyses on the present study population, as data were not collected on previous risk behaviors.

TABLE 2—Demographic Variables, Behaviors, and HIV Antibody Status of Female Recent Needle Users (n = 248), by Enrollment Site

Variables	Drug treatment centers			Other sites	
	TC (n = 25) %	Detox (n = 103) %	OP (n = 92) %	Clinics (n = 28) %	Total (n = 248) %
Age 30 years or over	40	40	61	36	47
Race					
Hispanic	16	8	10	21	11
Black	8	2	1	18	4
Other	76	90	89	61	85
High school graduate	72	65	68	39	64
Married	8	36	29	32	30
Worcester resident	20	28	68	79	52
Prior drug treatment	40	42	37	36	39
Prior jail	77	34	48	50	45
Injected >5 years	57	61	79	43	65
Drugs injected (ever)					
heroin	100	96	99	89	97
cocaine	100	87	84	86	87
Recent behaviors					
Daily needle use	80	96	94	68	90
Shared equipment	68	58	65	86	65
Male sex partners					
None	20	16	15	22	17
One	32	44	47	44	44
2 or more	48	39	38	33	39
Recent Drug Behaviors among Sharers	(n = 17)	(n = 59)	(n = 60)	(n = 24)	(n = 160)
Frequency of sharing					
daily	25	55	41	54	47
1–6/week	56	17	32	25	28
<1/week	19	28	27	21	25
Cleaned with bleach	71	74	71	50	69
Shared in NYC	18	8	8	0	8
Visited a shooting gallery	29	24	27	21	25
Recent Sexual Behaviors if Multiple Partners	(n = 12)	(n = 38)	(n = 35)	(n = 9)	(n = 94)
Used condoms	58	62	56	22	55
Prostitution	100	68	79	33	73
HIV antibody tests	(n = 25)	(n = 103)	(n = 92)	(n = 28)	(n = 248)
Currently tested	8	51	9	89	37
Positive result	50	17	13	32	22
Ever tested	56	79	55	96	70
Positive result	21	25	37	33	29

TC = Therapeutic Community;
Detox = Detoxification Facility;
OP = Outpatient Services

A second possibility is that previous HIV antibody testing may have affected recent risk behavior. Again, without information on previous behaviors, we were not able to assess this hypothesis.

A third possibility is that needle users who enter treatment represent a sub-population which is particularly motivated to reduce their risk of AIDS. Although they report a high frequency of needle use, they may have modified risk behaviors prior to entering treatment.

Reports having highlighted the significance of the recent epidemic of "crack" cocaine use for HIV transmission, in which sexual practices rather than IV drug use appear to be responsible for HIV transmission.[1] In March 1989, we began to collect information on "crack" use; about one-fifth of recent needle users have smoked "crack" although we have no information on frequency. Drug use rather than sexual behaviors appear to be associated with HIV seroprevalence in this study population, although these behaviors tend to be associated (unpublished data).

Demographic and behavioral differences in sub-populations of needle users have been reported by other researchers, although none have information on drug use and sexual behaviors from as many different enrollment sites as were possible in this study. Several studies from the United Kingdom have found that drug users in treatment were older, had used drugs for longer, and showed greater reductions in risk behavior.[8–11,***] Results of studies from different parts of the US, however, show inconsistent differences by site of enrollment.[2,12†,††,†††]

Several limitations of this research should be considered, including selection and measurement biases. Although the multi-site enrollment in this study allows some examination of possible selection bias, it does not eliminate such bias. Of particular concern is selection bias at the non-drug clinics and at the jail; at these sites, the HIV antibody testing may have attracted many of the participants and others have found that higher risk needle users tend to self-select for HIV testing in prisons.‡,‡‡

Measurement bias was avoided to some extent by having the interviews done by staff who were not involved in the individual's care or treatment. Nevertheless, study participants may have misrepresented their drug behaviors in efforts to impress the staff, or to obtain higher doses of methadone at drug abuse treatment sites. Factors such as social desirability and prior drug use by the interviewer can affect validity.[13,14] Self-reports of drug use may differ from "official" records, such as addict registers or hospital charts.[15–17] Self-reports of sexual behavior are of undetermined reliability and validity; this problem has been examined in homosexual males,[18] but has received little attention among IV drug users.

The demographic differences between needle users by enrollment site are cause for concern; Blacks and Hispanics who are at highest risk of HIV infection[19] are underrepresented in the sub-populations entering treatment.[20] Female needle users are also underrepresented at the inpatient and residential drug abuse treatment centers. Black women of

***Stimson GV, Alldritt L, Dolan L, Donoghoe M, Lart RA: Injecting equipment exchange schemes. Final Report. Monitoring Research Group, Sociology Department, Goldsmith's College, London, 1988.

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††McGough JP, Wood R, Harris N, Kleyn J, Carlisle M. HIV prevalence among different groups of intravenous drug users in Seattle, Washington. Abstract No. T.A.P.57, V International Conference on AIDS, Montreal, Quebec, Canada, June 4–9, 1989.

†††Friedman SR, Rosenblum A, Goldsmith D, Des Jarlais DC, Sufian M, et al. Risk factors for HIV-1 infection among street-recruited intravenous drug users in New York City. Abstract No. T.A.O.12, V International Conference on AIDS, Montreal, Quebec, Canada, June 4–9, 1989.

‡Andrus JK, Fleming D, Knox C, McAllister R, Skeels, Foster L, HIV antibody testing in prisons: assessment of voluntary vs. mandatory testing. Abstract No. 4201, IV International Conference on AIDS, Stockholm, Sweden, June 12–16, 1988.

‡‡Davis JP, Vergeront JM, Frisby HF, Hoxie NJ, Golubjatnikov R, Pfister J. HIV seroprevalence and the efficacy of voluntary HIV testing among male prison inmates in Wisconsin. Abstract No. M.A.P.22, V International Conference on AIDS, Montreal, Quebec, Canada, June 4–9, 1989.

TABLE 3—Multivariate Odds Ratios* (OR) and 95% Confidence Intervals (CI) for Drug and Sexual Behaviors and HIV Antibody Status by Enrollment Site among Male (M) and Female (F) Recent Needle Users

Variables	n	Drug Treatment Centers			Other sites		Goodness-of-Fit p-value
		TC	Detox	OP	Clinics	Jail	
Drug behavior							
Never share							
M OR	653	1.66	2.64	2.44	1.00	0.73	
CI		0.56–4.91	0.999–6.95	0.91–6.53		0.28–1.91	0.55
F OR	242	3.57	6.71	5.24	1.00	—	
CI		0.81–15.77	1.84–24.49	1.44–19.07			0.82
Clean with bleach**							
M OR	470	3.17	5.07	2.71	1.00	1.32	
CI		1.08–9.33	1.96–13.12	1.03–7.12		0.55–3.13	0.66
F OR	155	4.21	4.67	3.38	1.00	—	
CI		0.95–18.67	1.43–15.25	1.14–10.08			0.99
Sexual behavior							
<2 partners							
M OR	638	1.37	1.10	2.07	1.00	0.66	
CI		0.54–3.52	0.49–2.47	0.88–4.89		0.3–1.43	0.65
F OR	236	0.51	0.66	0.84	1.00	—	
CI		0.15–1.76	0.24–1.83	0.31–2.26			0.93
Use condoms***							
M OR	236	0.14	1.23	0.86	1.00	0.31	
CI		0.01–1.7	0.26–5.7	0.16–4.45		0.07–1.43	0.47
F OR	89	3.74	4.40	4.26	1.00	—	
CI		0.44–31.8	0.7–27.8	0.71–25.5			0.24
HIV positive							
M OR	530	0.43	1.10	1.31	1.00	1.22	
CI		0.09–1.94	0.39–3.15	0.44–3.93		0.48–3.15	0.47
F OR	169	0.55	0.58	1.32	1.00	—	
CI		0.11–2.81	0.19–1.77	0.44–3.96			0.55

*Controlling for age, race, marital status, education, and residence.
**Among subjects who share.
***Among subjects with multiple partners.

lower socioeconomic status are least likely to enter drug abuse treatment programs, and may only do so after exhausting informal resources.[21] Outpatient programs which offer child care are needed; methadone maintenance programs can be very effective, but may need to adopt a more lenient approach in order to keep clients in treatment.[22]

Our study has implications both for the conduct of AIDS prevention programs and for research. AIDS prevention programs need to target sub-populations of needle users with the most risky behaviors, particularly those not currently in treatment.‡‡‡ Incarcerated needle users are an important and very problematic group for prevention programs.[23,24]

Continued monitoring of IVDUs for HIV seroprevalence is important to identify high-risk sub-populations, to appropriately target prevention programs, and to evaluate the effectiveness of those programs. Samples of IVDUs entering drug treatment are often used for monitoring HIV exposure, partly because of their accessibility, and partly because the period before entering drug treatment is said to be characterized by a relatively high frequency of drug injection, and unsafe injection practices.[25] Although our results support higher frequency of drug injection in this sub-population, they do not support less safe injection practices. Use of data from drug abuse treatment programs for monitoring HIV exposure should not be adopted without further critical examination of the potential biases involved; data from drug users enrolled at different sites should not be pooled unless they have first been examined separately by site.

‡‡‡Sweha A, Flynn N, Nassar N, Jain S, Webb D, Acuna G. More than one type of HIV education and testing program is necessary to reach selected target populations in large numbers. Abstract No. W.A.P.118, V International Conference on AIDS, Montreal, Quebec, Canada, June 4–9, 1989.

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APPENDIX
Description of Study Variables

Demographics
- Age (years)
- Sex (male; female; transsexual)
- Race (Hispanic; black, non-Hispanic; white, non-Hispanic; Asian or Pacific islander; Indian/native American; other).
- Education: high school graduate or general education diploma (yes; no)
- Residence in city of Worcester (yes; no).

Prior experience
- Previously in jail (yes; no).
- Previously in drug abuse treatment, inpatient or outpatient (yes; no).

Drug use history
- Ever injected drugs, intravenously or subcutaneously (yes; no).
- Ever injected heroin (yes; no).
- Ever injected cocaine (yes; no).
- Injected drugs during the "risk period" (yes; no).

Drug use during "risk period"
- Frequency of injection (more than once/day; once/day; 3–6 times/week; 1–2 times/week; 1–3 times/month; less than once/month).
- Frequency of sharing needles, i.e., injecting with needles someone else has already used (once/day; 3–6 times a week; 1–2 times/week; 1–3 times/month; less than once/month).
- If sharing reported, was bleach ever used to disinfect "works" (yes; no).
- Sharing in a shooting gallery, i.e., a place where you pay with money or drugs to get in or to get high (yes; no).
- Sharing needles in New York City (yes; no).

Sexual behavior during "risk period"
- Number of female sexual partners (none; one; 2–9, 10–19; 20–49; 50 or more).
- Number of male sexual partners (none; one; 2–9; 10–19; 20–49; 50 or more).
- Sexual contact with someone you knew to have used needles since 1981 (yes; no).
- Received money or drugs in exchange for sex (yes; no).
- Sexual contact with a prostitute, i.e., someone who gets paid for sex with money or drugs (yes; no).
- Ever used condom during sex (yes; no).

HIV antibody testing
- Ever tested for HIV antibody, including current testing (yes; no).
- If ever tested, result (positive; negative; equivocal; unknown).
- Currently tested for HIV antibody, i.e., within one day of encounter interview at non-drug treatment sites, within 30 days of interview at the detoxification and the outpatient drug treatment facility, and within 45 days of interview at the residential drug treatment facility (yes; no). If currently tested, result (positive; negative; equivocal; unknown).

Joint US–Canadian Conference to Focus on Diabetes among Native Indians

"What Makes a Model Community Diabetes Program for American Indians Successful?" That will be the theme of a joint US—Canadian conference titled Diabetes and Native Peoples: International Issues in Education, Treatment, and Prevention, to be held November 7–9, in Minneapolis, MN. Sponsors include the Native American Research and Training Center (NARTC), Indian Health Service, Medical Services Branch—Health and Welfare Canada, American Diabetes Association, and the Canadian Diabetes Association.

This international conference will feature speakers from reservation communities and hospitals throughout Canada and the US, and will address common problems in establishing effective programs and providing care to Indian peoples with diabetes. Conferees will discuss development and implementation of model community diabetes programs. Standards of care and of health education recommended for Native peoples with diabetes also will be reviewed and discussed at the conference.

Continuing Education credit will be offered for physicians, nurses, and pharmacists. A $50 registration fee is required and covers the cost of the banquet dinner, Pow-Wow, and continuing education units. For further information, write or call: (USA), Dr. Robert Young, NARTC, 1642 E. Heler St., Tucson, AZ 87519, (602) 621-5560. (Canada) Virginia Toulouse, Assembly of First Nations—Diabetes Conference, 47 Clarence St., 3rd Floor, Atrium Building, Ottawa, Ontario K1N 9K1, (613) 236-0673.

Death Resulting from Motor Vehicle Immersions: The Nature of the Injuries, Personal and Environmental Contributing Factors, and Potential Interventions

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Abstract: We present an epidemiologic characterization of deaths from motor vehicle immersions, based on 77 deaths in 63 motor vehicle immersions in Sacramento County, CA, during 1974–85. All persons were autopsied; all but one of the deaths were due to drowning. Average annual mortality rates per million person years were 12 for males, four for females, 30 for Hispanics, six for White non-Hispanics, Blacks and Asians. Seventy-one percent of drivers and 60 percent of passengers had a blood alcohol concentration ≥22 mmol/L.

Most cases (57 percent) had an Injury Severity Score of 1 (minor injury) or 0 (no injury). Alcohol use was associated with higher Injury Severity Scores. Road curvature of 20° or greater was far more common at crash sites than at matched control sites one mile away(OR = 6.57, 95% CI = 2.93, 14.71). Guard rail placement along highly curved sections of roadway may be an effective preventive measure. (*Am J Public Health* 1990; 80:1068–1070.)

Introduction

Each year there are approximately 350 deaths in the United States resulting from motor vehicle immersions (unpublished data, Fatal Accident Reporting System, 1988). High motor vehicle-related drowning rates have been noted previously in places with many inland waterways such as Florida and Louisiana.[1] In Sacramento County, California, site of a large river delta and the location of this study, autopsy-confirmed drownings from motor vehicle immersions are more common than drownings from boating incidents (unpublished data, The Sacramento Drowning Study, 1987). To our knowledge, the nature and epidemiology of these deaths have not been described.

This study therefore sought answers to the following questions. Are deaths due to motor vehicle immersion actually drownings, or are they due to biomechanical trauma resulting from the crash event itself? By convention all such deaths have been classified as motor vehicle deaths, rather than drownings, since the adoption of the fifth revision of the International List of Causes of Death in 1939. What are the personal and environmental contributing factors for these deaths? What countermeasures are available and likely to be effective?

Sacramento County had 783,381 residents in 1980. The southwestern portion of the county is a river delta. Roads in this area are generally built on levees, some 30 feet above river level. These roads are two lanes wide, with lane widths of 10–12 feet, and have narrow shoulders. The roads are level except for the short distances needed to mount a levee; curves are generally not banked. We observed that speeds of travel commonly exceed 55 mph on major levee roads. In other rural portions of the county, roads may be built next to irrigation canals or other waterways.

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Methods

The study period was 1974–85. Cases were identified through a register maintained by the coroner's office, which lists both the anatomical and external causes of death. Cases were defined as persons, regardless of their cause of death at autopsy, who were motor vehicle occupants and who entered the water by means of a motor vehicle immersion. We reviewed coroner's and police reports and interviewed investigative personnel. We also reviewed coroner's records for all motor vehicle deaths occurring in the county for 1980–85, but found no additional cases.

All cases were autopsied. Causes of death were abstracted directly from the autopsy report. Analyses pertaining to blood alcohol concentration (BAC) were restricted to cases in which death occurred within six hours of immersion, and blood sampling occurred within 24 hours of death. These criteria avoid spuriously low values resulting from post mortem sampling after prolonged survival, and spuriously high values resulting from putrefaction.[2]

Traumatic injuries were scored for severity using the Abbreviated Injury Scale (AIS), which assigns to individual injuries a numerical score between 1 (mild) and 6 (unsurvivable).[3] Injury Severity Scores (ISS) were computed.[4] As used in this article, the term "head injury" includes injury to the external soft tissues of the head and face, such as lacerations and abrasions. In calculating Injury Severity Scores, however, these superficial head injuries were coded as "External" injuries as required by standard scoring procedures.

Following the case-control method developed by Wright and Robertson,[5] each crash site (defined as the point at which the vehicle left the roadway) was compared with a point one mile distant, on the same road except as noted below, in the direction from which the vehicle had come. When the path of travel could not be reconstructed with certainty because of an intersection within one mile of the crash site, preference was given first to arterial rather than tributary roads; when the choice involved two roads of the same type, preference was given to the road adjacent to the water. Eleven of 63 immersion events were excluded from this analysis (path of travel uncertain, three cases; vehicle traveled less than one mile prior to immersion, three cases; crash occurred on private or restricted roadway and the investigators could not gain access, two cases; other, three cases). We limited the

crash site—control site comparison to road curvature, determined by the middle ordinate method.[5] Curvature was measured at 100 foot intervals for 500 feet in either direction from the point at which the vehicle left the roadway. Gradient and banking, evaluated in Wright and Robertson's earlier work, were each present in only two of the sites we studied.

Using our case identification methods as a "gold standard" procedure for crash site identification, we calculated the sensitivity and specificity of various minimum-curvature criteria as screening tests for identifying known crash sites. Sensitivity was defined as the number of known crash sites identified divided by the total number of known crash sites. Specificity was derived in an analogous manner.

We also inventoried risk-reduction features at crash sites, such as warning signs and guard rails, to learn whether post-crash modifications had been made. We used each crash site as its own historical control, comparing the detailed written and pictorial description in the earlier police reports with our observations in 1987–88. This analysis was limited to the 34 cases for which detailed police reports were available.

Average annual mortality rates were calculated based on place of occurrence, using Sacramento County data from the 1980 US Census to approximate a mid-interval population. All denominators are age-, race-, and gender-specific as appropriate. Rates are rounded.

Confidence intervals were calculated by the exact binomial method using Confidence Interval Analysis software.[7] Other analyses were performed with PC-SAS.

Results

There were 78 deaths identified, but one was excluded as the coroner's record could not be located. The 77 remaining deaths resulted from 63 motor vehicle immersions, all single vehicle events, of which seven led to multiple fatalities. Twenty-five immersions also involved a total of 37 vehicle occupants who survived. (Further information on survivors was not available.)

A car was involved in 65 deaths, a truck in 11, and a motorcycle in one. Fifty persons were drivers, and 27 were passengers. Three deaths were specified as job-related. Seventy-five deaths were classified by the coroner as unintentional, one as suicidal, and one as of undetermined intent.

In all but one fatality, drowning was reported as the sole cause of death, or drowning and one or more traumatic injuries were listed as multiple causes of death. The cause of death for the remaining case was "craniocerebral trauma with possible drowning."

The average mortality rate was eight per million person years. Cases ranged in age from seven months to 73 years; 38 were age 15–34; 57 cases were male. Mortality rates were 12 for males and four for females, per million person years.

The mortality rate for Hispanics was five times those for White non-Hispanics, Blacks, and Asians (30 for Hispanics, six for the other groups, per million person years). Hispanics accounted for 36 percent of these deaths, but made up only 9 percent of the county's population in 1980. This difference was not due to a disproportionate number of nonresidents among Hispanic cases. Sixty-three percent of Hispanic cases, and of all cases, lived in the county. While immersions involving any Hispanic deaths were more likely than others to result in multiple fatalities (OR = 7.5, 95% CI = 1.6, 35.3), this difference did not fully explain the high Hispanic mortality rate. The fatal immersion event rate was also higher for Hispanics than for non-Hispanics (20 and six per million person years, respectively).

Forty deaths occurred on Saturday or Sunday, twice the number predicted by the hypothesis of equal occurrence throughout the week ($\chi^2 = 17.19$, df = 6, $p < .01$). There were no significant differences in incidence by time of day.

A blood alcohol concentration (BAC) was determined in 75 cases; the specified eligibility criteria were met in 56. Five cases were children under age 15, none of whom had a positive BAC. Among older persons, 74 percent of drivers and 73 percent of passengers had a positive blood alcohol concentration; 71 percent of drivers and 60 percent of passengers had a BAC ≥ 22 mmol/L.

Serious biomechanical trauma was unusual (Table 1). Most persons had an Injury Severity Score of 1 (minor injury) or 0 (no injury). Fifty-one cases sustained a head injury, but 26 of these were AIS 1 injuries. Alcohol use was associated with higher Injury Severity Scores. Among cases meeting the alcohol sampling criteria, 15 of 18 cases with a score ≥ 10 had a BAC ≥ 11 mmol/L, as opposed to 23 of 36 cases with an ISS of 0 or 1 (OR = 2.83, 95% CI 2.05, 3.89).

Post-event interventions were uncommon. Only four cases involved immersion times of 30 minutes or less. Three of these four people, and 11 of all 77 cases, were given emergency care after retrieval from the water. One person survived long enough to be admitted to a hospital.

There were large differences in road curvature between crash and control sites. Crash sites had a median curvature of 19° over 1000 feet; the median curvature for control sites was 8° (difference = 11°, approx 95% CI = 2°, 18°). The differences in mean curvature were greatest over the 500 feet approaching the point where the vehicle left the roadway (Figure 1). Crash sites were far more likely than control sites to involve a curvature of 20° or greater (OR = 6.57; 95% CI = 2.93, 14.71). Conversely, crash sites were less likely than control sites to have curvatures of less than 5° (OR = .42; 95% CI = .24, .73).

The sensitivity and specificity of several minimum curvature criteria for identifying crash sites varied inversely (Table 2). None of the criteria evaluated was highly sensitive, but it was possible to identify approximately 50 percent of crash sites with a specificity of 75 percent or greater.

There were four multiple-crash sites, each of which had two fatal motor vehicle immersions during the study period. Three of these sites had curvatures of greater than 30° (34°, 66°, 84°) over 1000 feet. The fourth had a curvature of 4° (less than its control) and no other explanatory features.

At two of 34 crash sites for which complete police reports were available, guard rails existed between the road and water at the time the crash occurred. Guard rails had been added to

TABLE 1—Incidence and Severity of Head and Other Injuries in 77 Motor Vehicle Immersion Deaths, Sacramento County, California 1974–85

Overall Injury Severity		Head Injury Severity	
Injury Severity Score	N (%) of Cases	Head Injury AIS*	N (%) of Cases
0	16 (21)	0	26 (34)
1	28 (36)	1	26 (34)
2–9	4 (5)	2	9 (12)
10–19	19 (25)	3	9 (12)
20+	10 (13)	4	7 (9)
Total	77	Total	77

*Includes external injuries to the head and face.

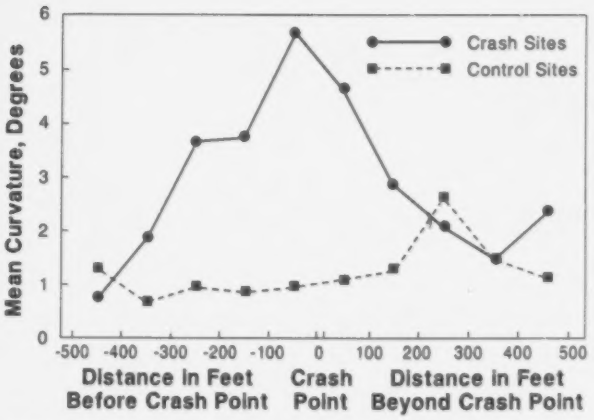

FIGURE 1—Mean Road Curvature over 1,000 Feet at 52 Fatal Motor Vehicle Immersion Sites and Matched Controls, Sacramento County, California 1974–85

five other sites, and extended at one of the first two locations, by the time our field investigation was conducted.

Discussion

These deaths represent an unusual subset of motor vehicle-related deaths, in that traumatic injury was generally minor if present at all. Death, generally by drowning, appeared to result more directly from the hostile environment in which the crash occurred. This may be particularly true for those cases involving minor head injury; transient disorientation or loss of consciousness may well result in death given that immersion has occurred. From the pathophysiologic viewpoint, these deaths are more properly considered drownings. From the prevention standpoint, their current classification as motor vehicle deaths seems appropriate.

The mortality rate for Hispanics was approximately five times that for other groups. These rates were calculated based on place of occurrence, but the observed difference did not result from a high proportion of out-of-county residents among Hispanic fatalities. A number of other factors may be involved. First, fatal immersions involving Hispanic vehicle occupants were more likely than others to result in multiple deaths. Second, the high risk roadways are in agricultural areas of the county. Since Hispanics in the Sacramento area are heavily involved in agriculture, their higher fatal immersion rate may result from a higher exposure to these roadways than that of other groups. Differences in alcohol use and driving experience may also play a role. A controlled study will be necessary to evaluate these possibilities.

TABLE 2—Sensitivity and Specificity of Various Minimum Curvature Criteria for Identifying Sites of 52 Fatal Motor Vehicle Immersions Occurring in Sacramento County, California 1974–85

Minimum Curvature over 1,000 feet (degrees)	Sensitivity (95% CI)	Specificity (95% CI)
10	.58 (.43, .71)	.58 (.43, .71)
15	.50 (.36, .64)	.75 (.61, .86)
20	.46 (.32, .61)	.88 (.76, .96)
30	.37 (.24, .51)	.94 (.84, .99)

Alcohol was a major contributing factor. Seventy-four percent of drivers meeting our sampling criteria had a positive BAC, as did 69 percent of drivers killed in single vehicle crashes nationwide in 1985.[8] Alcohol use was associated with increased injury severity. It is likely that alcohol use contributes to these deaths in two ways: by increasing the likelihood that an immersion event will occur, and by impairing the ability to escape a sinking vehicle.

Finally, we have demonstrated that roadway curvature is an important environmental contributing factor for fatal motor vehicle immersions. Similar results have previously been described for fatal crashes involving roadside fixed objects[5] and single vehicle fatal rollover crashes.[9,10] Sites at high risk for future crash involvement can therefore be identified for environmental modification.

Placing guard rails along waterside roadways would substantially decrease the incidence of immersions. The guardrail now in use in the study area is a w-beam, blocked-out design. Field studies have demonstrated that this design is highly efficient at redirecting impacting vehicles, with a penetration rate of less than 15 percent.[11] The cost of installing this guard rail averaged $14.50 per linear foot in 1988,* or $76,560 to protect one side of a mile of roadway.

It is probably not cost-effective to install guard rails throughout an entire system of waterside roadways. Using a curvature criterion would permit resources to be focused on the most hazardous locations. Nearly half of high-risk locations would be protected by installing guard rails along sections of roadway with curves of 20° or greater over 1000 feet, while minimizing installation in low-risk areas.

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Drownings in Minnesota, 1980–85: A Population-Based Study

KATRINA HEDBERG, MD, PAUL D. GUNDERSON, PHD, CAROL VARGAS, MICHAEL T. OSTERHOLM, PHD, MPH, AND KRISTINE L. MACDONALD, MD

Abstract: We conducted a population-based study of drownings in Minnesota from 1980 through 1985. Five hundred and forty-one drownings (2.1 per 100,000 person-years) were identified from death certificates and from incident reports filed with the Minnesota Department of Natural Resources. Most drownings (334, 62 percent) occurred during summer months (May–August) and involved boating (42 percent) and swimming (35 percent) events. However, 62 drownings (11 percent) occurred during winter months (December–February) and primarily involved snowmobiles and motor vehicles (71 percent) breaking through ice on lakes and waterways. The risk of drowning, estimated by the ratio of drownings to number of water-related activities, was highest during March and April, when the ice on lakes and waterway surfaces is melting, and during October and November, when lake and waterway surfaces are starting to freeze. Drowning rates were highest for males (3.7 per 100,000 person-years), persons 15 to 25 years of age (3.3 per 100,000 person-years), and children less than 5 years of age (2.5 per 100,000 person-years). These data can be used to target prevention strategies, particularly in northern climates. (*Am J Public Health* 1990; 80:1071–1074.)

Introduction

Drowning is the third most common cause of fatal, unintentional injury in the United States; among children and young adults it is the second most common cause.[1] In 1984, drowning accounted for 4,444 deaths nationwide.* Yet detailed epidemiologic data are lacking for outdoor and recreational drownings in northern climates, where surface water is frozen for several months of the year.[2]

Minnesota is known as "The Land of 10,000 Lakes." Water covers 7,353 square miles or 9.2 percent of the state's surface area. Lakes and waterways provide the 4.2 million people who live in Minnesota with numerous recreational opportunities throughout the year, including swimming and boating in the summer and ice-fishing in the winter. The lakes and waterways are usually frozen from December through March.

We conducted a population-based study of drownings in Minnesota, with an emphasis on outdoor and recreational drownings. In addition to characterizing drowning victims, we examined data by season-specific activities associated with drowning.

Methods

A case of drowning was defined as an unintentional death due to submersion in water, that occurred to a Minnesota resident during 1980 through 1985. Deaths involving motor vehicles operated in or on a body of water were considered to be drownings. However, deaths involving motor vehicles that ran off the road into water were considered to be motor vehicle fatalities.

Data on drownings were obtained from two sources: death certificates maintained by the Minnesota Center for Health Statistics, and fatal injury reports filed with the Minnesota Department of Natural Resources. All death certificates in Minnesota are assigned an ICD-9 cause of death code by a certified nosologist. Death certificates assigned an ICD-9 code for drowning were reviewed. This included both boating (E830-E832) and non-boating fatalities (E910).

Law enforcement officials are required to investigate and file a report with the Department of Natural Resources on all fatal injuries that occur on Minnesota lakes and waterways (Minnesota Statute 361.24). These officials often use coroner reports in preparing their reports. These reports were reviewed, and information was obtained on the age and sex of the victim; the date, time, and location of the incident; the victim's activity at the time of drowning; the probable cause of the drowning; and history of alcohol use. Death certificates and Department of Natural Resources reports were manually matched by name, age, and address.

Drowning rates were calculated for the general population of Minnesota residents using population data extrapolated from the 1980 census. To estimate rates of outdoor and recreational drownings for the specific population-at-risk, we calculated the ratio of drownings to the estimated number of water-related activities. The annual number of water-related activities in which Minnesota residents participated was extrapolated from a random telephone survey of 6,000 households conducted by the Department of Natural Resources during a 12-month period, beginning in September 1985.** Each week, a sample of households was contacted and asked to report the age and sex of household members and the number of water-related activities in which each member had participated during the preceding week. (Information was collected on a number of activities, but not on hours of activity.) In addition, we reviewed records that the Department of Natural Resources has maintained since 1944 on the number of drowning deaths that have occurred in Minnesota.

Data were analyzed using standard univariate methods.[3] Years of potential life lost prior to age 65 were calculated.[4]

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*Division of Vital Statistics, National Center for Health Statistics (NCHS), Hyattsville, MD, (unpublished data).

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**Minnesota Center for Survey Research, University of Minnesota. A continuous survey of participation and expenditures in outdoor recreation by Minnesota residents—Year end summary report, February 1987, (unpublished document).

Results

Five hundred and forty-one drownings occurred to Minnesota residents during 1980 through 1985; 458 drownings (85 percent) were identified from death certificate review (ICD-9 codes E830-32 and E910) and 83 drownings (15 percent) were identified only from Department of Natural Resources reports (death certificates were retrieved for these drownings). These 83 deaths were coded as follows: 35 (42 percent) were assigned the ICD-9 code for drownings which reflects accidental or purposely inflicted cause undetermined (E984); 32 (39 percent) were assigned the code for motor vehicle fatalities (E820-25); and 16 (19 percent) were assigned other ICD-9 codes including cardiac arrests, diving accidents, falls and hypothermia.

Four hundred and ninety-one drownings (91 percent) occurred in Minnesota and 50 (9 percent) occurred out-of-state. Forty-three (86 percent) of the out-of-state drownings occurred in the states immediately adjacent to Minnesota or in areas with northern climates (Montana, Alaska, and Canada). The seasonal distribution for the out-of-state drownings was similar to the distribution of drownings which occurred in Minnesota. Five hundred drownings (92 percent) occurred outdoors or were related to recreation and 41 drownings (8 percent) occurred in bathtubs. Department of Natural Resources reports were completed on 404 (90 percent) of the 450 outdoor and recreational drownings that occurred in Minnesota.

Death Certificate Review

The 541 drownings identified represent an overall mean annual incidence of 2.1 drownings per 100,000 person-years. The annual incidence declined over the study period from 2.5 per 100,000 person-years in 1980 to 1.9 per 100,000 person-years in 1985. This finding is consistent with a decreasing trend in the annual incidence of drowning observed in Minnesota since 1944. The drownings that occurred during the study period represent 17,853 years of potential life lost before age 65 (33 years lost for each death).

The majority of drownings (334, 62 percent) occurred during the summer months, May through August, when outdoor and recreational activities peak (Figure 1). However, 62 drownings (11 percent) occurred during the winter months, December through February, when lakes and waterways are frozen, and water-related activities are low. The risk of drowning, estimated by the ratio of drownings to number of water-related activities, was highest during spring and fall months (Figure 1).

Overall, 447 drownings (83 percent) occurred in males (Figure 2). The mean annual incidence for males was 5.3 times higher than that for females (3.7 versus 0.7 per 100,000 person-years, respectively). Incidence rates were highest for persons 15 to 30 years of age and for person less than 5 years of age (3.3 and 2.5 per 100,000 person-years, respectively). For the age group 15 to 30 years, 91 percent of drownings occurred in males. For this age group, the rate for males was 10 times higher than the rate for females (5.2 versus 0.5 per 100,000 person-years, respectively). The ratio of number of drownings to number of water-related activities for males was 3.3 times higher than the ratio for females (13.7 versus 4.2 per 1,000,000 activities, respectively).

The drowning rate for persons 15 to 49 years of age decreased significantly over the six-year study period ($p < 0.001$, chi-square test for trend). However, rates for persons less than 15 years of age and for persons greater than or equal to 50 years of age did not show a similar decrease ($p = 0.22$, and 0.82, respectively, chi-square test for trend).

Department of Natural Resources (DNR) Incident Report Review

DNR report forms were completed for 404 of the outdoor and recreational events. Of these, 382 (95 percent) occurred in a lake, pond, river, or stream, and 18 (4 percent) occurred in a swimming pool.

Table 1 shows the type of activity in which victims were engaged at the time of drowning. Swimming and boating were the most common activities during the summer, driving a vehicle on ice was the most common activity during the winter, while boating and falling from a bank into a body of water were common activities during the fall and spring.

Type of activity also varied by age group. For all ages combined, 66 drownings (16 percent) were due to falling from land, pool-side, or through ice into a body of water. However, for the 29 drownings that occurred in children <5 years of age, 22 (76 percent) were due to falling into a natural body of water or through ice, and three (10 percent) were due to falling into swimming pools. For the 21 drownings that occurred in persons ≥60 years of age, all were due to falling into water from land, pool-side, or through ice.

One hundred and thirty-five of the 404 drownings (33 percent) involved boating incidents. Table 2 shows the probable cause of boating-related drownings and the use of a

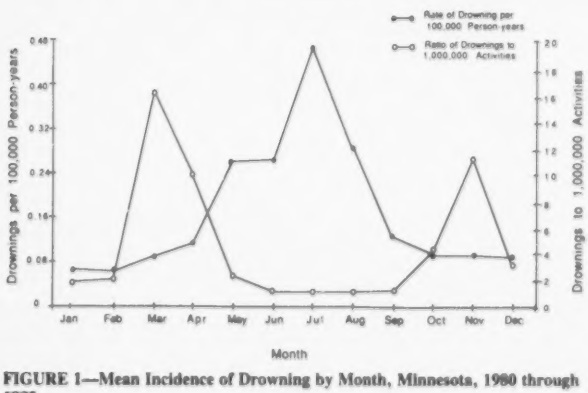

FIGURE 1—Mean Incidence of Drowning by Month, Minnesota, 1980 through 1985

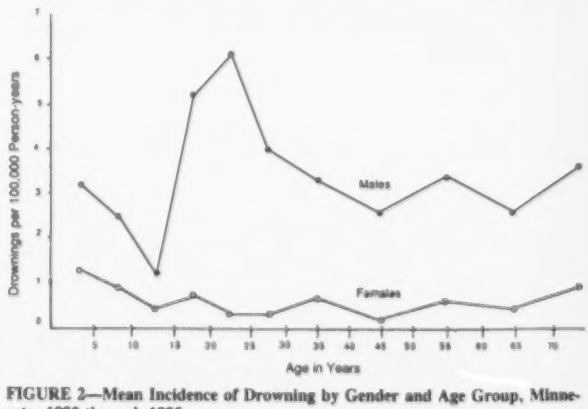

FIGURE 2—Mean Incidence of Drowning by Gender and Age Group, Minnesota, 1980 through 1985

TABLE 1—Type of Activity Reported at the Time of Drowning by Season, Minnesota, 1980 through 1985

Activity	Summer (May–Sept) No. (%)	Fall (Oct–Nov) No. (%)	Winter (Dec–Feb) No. (%)	Spring (Mar–Apr) No. (%)
Swimming/Wading	120 (42)	—	1 (2)	3 (8)
Boating	100 (35)	23 (60)	1 (2)	11 (28)
Fall from bank	30 (10)	7 (18)	—	11 (28)
Inner-tubing	10 (3)	—	—	—
Diving	7 (2)	—	—	—
Vehicle on ice	—	4 (11)	29 (71)	7 (18)
Walking on ice	—	4 (11)	10 (25)	4 (10)
Rescue attempt	2 (1)	—	—	—
Other	2 (1)	—	—	3 (8)
Unknown	15 (5)	—	—	—
Total (N = 404)	286 (100)	38 (100)	41 (100)	39 (100)

TABLE 2—Boating-Related Drownings by Cause and Use of Personal Flotation Device, Minnesota, 1980 through 1985

Cause	No.	(%)
Capsize/Sink	66	(49)
Fall Overboard	38	(28)
Collision—Boat/Dock	16	(12)
Other/Unknown	15	(11)
Total	135	(100)
Use of PFD*		
Used	13	(10)
Available/Not Used	66	(49)
Not available	54	(40)
Unknown	2	(1)
Total	135	(100)

*Personal flotation device

personal flotation device. Only 13 drownings (10 percent) occurred while such a device was being used.

History of alcohol use was noted on 242 reports (60 percent); three (6 percent) of 54 persons less than 15 years of age and 109 (58 percent) of 188 persons more than 15 years of age were reported as having consumed alcohol at the time of the event.

Discussion

The mean annual incidence of drowning in Minnesota during 1980 through 1985 (2.1 per 100,000 person-years) was lower than the 1984 incidence of drowning nationally (2.3 per 100,000 person-years),[5] and lower than the incidence in Georgia from 1981 through 1983 (3.2 per 100,000 person-years),[6] New Mexico from 1975 through 1980 (4.3 per 100,000 person-years),[7] North Carolina from 1980 through 1984 (3.2 per 100,000 person-years),[8] and Los Angeles County from 1976 through 1984 (2.4 per 100,000 person-years).[9] This probably reflects the fact that the peak season when people are participating in outdoor water activities is shorter in Minnesota where lakes and waterways are usually frozen for several months of the year.

We believe that a population-based incidence rate provides an accurate estimate of the risk of drowning and can be compared to population-based rates in other states. Yet, population-based rates assume that every resident in the state is at equal risk of drowning. The ratio of drownings to the number of water-related activities provides an estimate of the risk of drowning for the specific population that participates in water-related activities. The importance of this estimate is illustrated by the seasonal trends in drowning rates.

Previous investigators have noted that the incidence of drowning has marked seasonal variation.[1,10] In Minnesota, as in other states, most drownings occurred during the summer. However, if the risk of drowning is estimated by the ratio of drownings to number of water-related activities, the risk was highest during the spring and fall months, when the ice on the lakes is starting to freeze or thaw and likely to be unsafe. In addition, a submersion incident may be more likely to result in a fatality during the non-summer months due to the increased risk of hypothermia. Thus, this increased risk is probably specific to areas where surface water freezes during the winter.

Investigators in Hawaii and California found that the majority of drownings in these areas occurred in swimming pools.[9,11,12] However, we found that 95 percent of drownings in Minnesota occurred in lakes and waterways and only 4 percent occurred in swimming pools. Even in children less than 5 years of age, only three drownings occurred as a result of falling into swimming pools. Minnesota residents may use swimming pools less than residents of more southern climates because most people have access to natural bodies of water and because of the relatively short season in which people can swim outdoors. Therefore, prevention measures such as requiring all swimming pools to be fenced in, which have proved to be effective in other areas,[13] may have only limited impact in areas similar to Minnesota.

Our data showed that males, children, and young adults had the highest rates of drowning, a finding consistent with several past studies.[6–12,14–16] Comparing population-based rates, the incidence in males was 5.3 times the incidence in females. However, if the risk of drowning is estimated by the ratio of drownings to the number of water-related activities, the risk for males was only 3.3 times the risk for females. Thus, some of the excess risk seen in males may be due to increased time spent participating in water-related activities.

Behavioral factors which may put people at increased risk of drowning include alcohol consumption[16–19] and the nonuse of a personal flotation device. Although more than half of the Minnesota Department of Natural Resources reports completed for persons over 15 years of age listed alcohol consumption as contributing to the drowning, we do not know the number of people consuming alcohol that did not drown. Also, although only 10 percent of boat-related drownings occurred while a flotation device was being used, we do not know how many people who were wearing such devices did not drown, or how many drownings were avoided by people wearing such devices. Therefore, although our data suggest that alcohol consumption and the nonuse of a flotation device may increase the risk of drowning, we are unable to evaluate the exact contribution of these possible risk factors.

Although the overall rate of drowning in Minnesota decreased during the study period, only the rate in persons 15 to 49 years of age showed a significant decline. Risk-taking behavior may have contributed more to drownings in this age group than in other age groups, and may have been more amenable to current intervention strategies. The decrease in the incidence of drowning deaths may also have been a result of improved medical treatment. However, we were unable to evaluate this due to the lack of data on near-drownings.

To design, implement and evaluate injury intervention strategies, accurate surveillance data on injuries are needed.

Previous investigators have questioned the accuracy and usefulness of death certificates for obtaining selected mortality statistics.[20] In our study, 15 percent of the drownings were identified from the Department of Natural Resources reports and would have been missed if we had relied solely on death certificate review. We did not include death certificates assigned ICD-9 code E-984 in our initial review because we wanted to exclude suicides and homicides from the study. However, by examining information from the Department of Natural Resources reports, we were able to include those deaths which were unintentional. In addition, one-third of the drownings identified from the Department of Natural Resources reports had been assigned an ICD-9 code for a motor vehicle fatality. We chose to include only those deaths in which the vehicle was operated on an ice-covered body of water and exclude those in which the vehicle had run off a road into water, assuming that different risk factors may have been involved. Such information on the circumstances surrounding the events was available from the Department of Natural Resources reports but was lacking on most death certificates. This information is vital to understanding the causes of drowning and can be used to target further prevention strategies.

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Society for Disability Studies Issues Call for Papers

The Society for Disability Studies has issued a call for papers for its fourth annual convention, to be held June 19–22, 1991, in San Francisco, California. Possible themes for paper presentations may include research issues of policy, history, sexuality, law, culture, statistics, methods, politics, media, literature, gender, self-image, cross-cultural, family, and related topics. Organized panels are encouraged, but they must be multidisciplinary and cross disability. Proposals for roundtables are also encouraged, and they should attempt to bring together participants with similar research interests.

The deadline for submission of abstracts is January 15, 1991. Please send two copies of a two-to-three page abstract to: Professor David Pfeiffer, Department of Public Management, School of Management, Suffolk University, Boston, MA 02108. Tel: (617) 573-8317.

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A Multifocal Outbreak of Hepatitis A Traced to Commercially Distributed Lettuce

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Abstract: From February 1 through March 20, 1988, 202 cases of hepatitis A were reported in and around Jefferson County, Kentucky. The epidemic curve indicated a common-source exposure. However, there was no apparent single source of exposure from a restaurant, or community gathering; nor was there a geographic clustering by residence. Cases were mainly adults 20–59 years old (89 percent); 51 percent were female. A case-control study using neighborhood controls found that factors associated with hepatitis A were: having eaten downtown (odds ratio [OR] = 4.0) and having dined at any one of three restaurants (OR = 21.0). Case-control studies of patrons of two of these restaurants found that eating green salad was strongly associated with acquiring hepatitis A: OR = 11.6 and OR = 4.4. The three implicated restaurants accounted for 71 percent of the cases. All three restaurants were supplied by the same fresh produce distributor; however, investigation suggested that contamination most likely occurred prior to local distribution. This outbreak of hepatitis A is the first in the United States apparently associated with fresh produce contaminated before distribution to restaurants, and raises important public health issues regarding the regulation of fresh produce. (*Am J Public Health* 1990; 80:1075–1080.)

Introduction

In 1988, 26,600 cases of hepatitis A were reported in the United States.[1] During 1983–88 the incidence of reported cases of hepatitis A increased from 9.2 to 10.9 per 100,000 population, representing the first increase in hepatitis A in more than a decade.[1–3] An estimated 1,000 cases are associated with suspected food- or water-borne outbreaks of hepatitis A each year.[3]

Previous investigations of common-source food-borne outbreaks of hepatitis A have described unifocal outbreaks in which a single location of food consumption (such as a restaurant) was the site of the outbreak; generally, a food handler, recently infected with hepatitis A virus (HAV) and working at that site, was implicated as the source of contamination of the food.[4–6] There have also been reports of outbreaks involving more than one restaurant; however, a food handler was implicated as the source of contamination at each restaurant.[4,7] The major multifocal food-borne outbreaks of hepatitis A due to the contamination of food before distribution to a restaurant have involved shellfish harvested from contaminated bays.[8,9] Reports from the United Kingdom have suggested that certain commercially processed foods (such as frozen raspberries) may have been associated with the transmission of HAV.[10,11]

This is the first report of a hepatitis A outbreak in the United States associated with unprocessed fresh produce contaminated before distribution to restaurants.

Methods

From February 1 through March 20, 1988, a widespread outbreak of hepatitis A affected 202 residents of Jefferson County, Kentucky (1986 intercensal population estimate, 681,066) and the surrounding area. The Louisville-Jefferson County Board of Health, the Kentucky Department of Health, and the Centers for Disease Control initiated a series of investigations to determine the risk factors for the transmission of HAV.

Case Identification

A case was defined as a person who had onset of an illness compatible with viral hepatitis from February 1 through March 20, 1988, and either a positive serologic test for immunoglobulin M (IgM) antibody to HAV (IgM anti-HAV) or a physician diagnosis of hepatitis A. Cases were ascertained through reports by physicians to the Louisville City-Jefferson County Health Department. Serologic testing for IgM anti-HAV was performed by private and hospital-based laboratories in Louisville. All cases were interviewed by telephone to obtain demographic information, date of onset and clinical course of illness, work location, and place of residence.

Community Food and Water Exposure Study

Because the epidemic curve indicated a common source of exposure, a case-control study using neighborhood controls was performed to identify the location of the common source of food or water in the community. A random sample of cases who resided in Jefferson County were matched with controls by neighborhood and by age within 10 years. One control who lived within 10 houses in either direction from each case was selected randomly by using a reverse telephone directory. Eligible controls had no history of hepatitis; no undiagnosed illness during February or March, with any two of the following symptoms: jaundice, nausea, vomiting, and abdominal discomfort; no receipt of immune globulins within the past six months; and no household or sexual contact with a person with hepatitis A since January 15, 1988. Cases and controls were interviewed by telephone to obtain demographic information, history of food and water exposures at home and work, and exposures at restaurants, stores, parties, and community gatherings during January 15–31.

Restaurant Food Exposure Studies

Because the neighborhood study found that eating at any one of three restaurants was associated with acquiring hepatitis A, two of the restaurants (A and B) were selected as sites for the investigation of the source of HAV. Restaurants

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A and B were selected because they accounted for most of the cases, and no case had patronized both restaurants. Restaurant A, located in a downtown professional district, was primarily a lunch business, serving sandwiches, chef salads, and soup. Restaurant B, located in the outskirts of the city, was primarily a dinner restaurant, with standard American fare and a salad bar.

The period of risk of exposure at these restaurants was considered to be January 15–31, one month (the average incubation period for HAV) preceding the period of peak case incidence (February 15–28). Evaluation of the dates of exposure of patrons who ate only once at one of these restaurants (A or B) and ate at no other restaurants in Jefferson County during the month preceding their illness confirmed that January 15–23 was the period of high risk.

For each restaurant study, eligible cases and controls had dined at the designated restaurant (A or B) during January 15–31; controls also had to meet the four eligibility criteria described for controls in the neighborhood study. For the study of restaurant A patrons, unmatched controls were randomly selected from a list of coworkers of each case. For restaurant B, controls were meal companions of the cases. Telephone interviews of cases and controls obtained demographic information and history of food consumption, including the frequency of eating particular foods.

Investigating the Source of Contamination

Based on the results of the restaurant studies, which implicated fresh produce obtained from a single local distributor, the Food and Drug Administration traced the source of shipments of food received by the distributor. In collaboration with the Louisville-Jefferson County Board of Health, the Louisville Water Company evaluated the water supply system.

Statistical Methods

In the community case-control study, exact confidence limits for the odds ratios were calculated by using standard procedures for the binomial distribution.[12] MCSTRAT (a procedure in Statistical Analysis Systems), which is designed for matched case-control data, was used to estimate the variances of the odds ratios in multivariate analysis.[13] For the restaurant case-control studies, variances for the odds ratios were estimated by using the method described by Cornfield.[12]

Results

Outbreak

From February 1 through March 20, 1988, 202 reported cases of hepatitis A met the case definition; 99 percent were tested and confirmed as IgM anti-HAV positive. Cases included 172 Jefferson County residents, and 30 others who had histories of exposures in Jefferson County during the last two weeks in January.

During the 13 months preceding the epidemic, only 12 cases of hepatitis A had been reported in Jefferson County. During the epidemic, there was no increase in the number of cases reported in any of the surrounding counties in Kentucky or Indiana, except for the cases who had known exposures in Jefferson County.

The epidemic curve, with a sharp peak and most of the cases occurring within the two-week period, suggested a common-source exposure (Figure 1). There was no apparent single source of exposure from a restaurant or community gathering and no geographic cluster by location of residence; only seven families had more than one case. Cases clustered

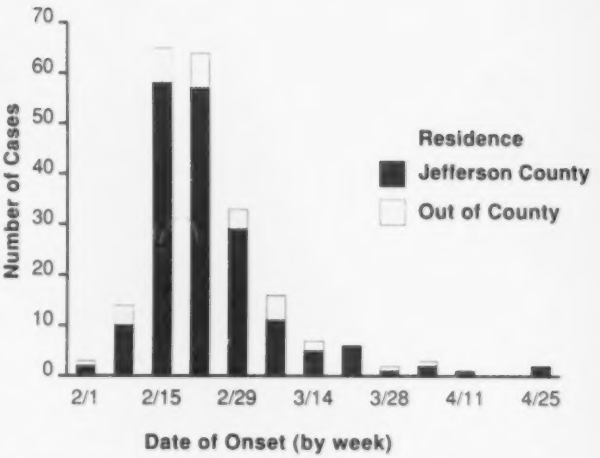

FIGURE 1—Hepatitis A Cases by Date of Onset of Symptoms, Jefferson County, Kentucky, February 1–April 30, 1988

by work location, with 33 percent of the cases working in the downtown area.

Of the cases residing in Jefferson County, the mean age was 38 years (range: 1 to 75 years), 89 percent were 20 to 59 years old, and 51 percent were female. The overall attack rate was 25 cases per 100,000 residents of Jefferson County. The highest attack rate was for adults 20 to 59 years old (40 cases per 100,000 persons), with the highest age-specific attack rate occurring among persons 40 to 49 years old (62 cases per 100,000). Of the 172 cases, 91 percent were jaundiced and 23 percent were hospitalized. HAV infection may have contributed to two deaths (for a case-fatality ratio of 1 percent), including a 60-year-old man who required a liver transplant five months after the onset of illness and died two months later, and a 53-year-old man with a history of alcohol abuse, who died three weeks after onset of illness.

Community Food and Water Exposures

In the study of community food and water exposures, 30 neighborhood case-control pairs were interviewed. A comparison of cases and their matched controls indicated that 70 percent of the pairs had similar levels of income and education, and all were White and non-Hispanic.

Exposures associated with acquiring hepatitis A in univariate analysis included (Table 1) working downtown (odds ratio [OR] = 5.0), having eaten downtown (OR = 4.0), having dined at any one of three (A, B, or C) restaurants (OR = 21.0), and having eaten salad at any restaurant (OR = 6.0). Two of the three implicated retaurants were located downtown. The association between acquiring hepatitis A and having dined at these three restaurants persisted when results were adjusted separately for history of work downtown, and for potential confounders (e.g., income and education).

Restaurant Food Exposures

Restaurant A: In the study of patrons of restaurant A, 38 (100 percent) of the eligible cases were interviewed, and 36 fellow office workers were interviewed as controls. The study found that eating green salad was associated with an 11.6-fold increased risk of acquiring hepatitis A (Table 2). Consumption of other foods was not associated with an increased risk of disease. The risk of acquiring hepatitis A increased as the frequency of consumption of green salads increased from a

TABLE 1—Neighborhood Study of Community Food and Water Exposures

Exposure	% Cases exposed (n = 30)	% Controls exposed (n = 30)	Matched odds ratio	95% confidence interval
Patronize restaurants				
A, B, or C	77	10	21.0	3.4, 832.3
A	30	0	∞	2.0, ∞
B	33	7	9.0	1.2, 399.0
C	23	3	∞	1.1, ∞
Work downtown	40	13	5.0	1.1, 46.6
Eat downtown	47	17	4.0	1.1, 21.7
Eat salad at any restaurant	83	50	6.0	1.3, 49.0
On public water supply	100	97	∞	—
Patronize grocery store 1	70	73	0.8	0.1, 4.2
Patronize grocery store 2	30	23	1.4	0.4, 5.3
Drink water at workplace	77	70	1.5	0.4, 7.2
Use ice machine at work	37	23	1.7	0.6, 5.6

TABLE 2—Restaurant A: Study of Food Exposures

Food Consumed	(%) Cases exposed	(%) Controls exposed	Odds ratio	95% confidence interval
Green salads	32/38 (84)	11/35 (31)	11.6	3.4, 42.8
Soup	26/38 (68)	20/36 (56)	1.7	0.6, 5.0
Prepared salads	16/38 (42)	13/36 (36)	1.3	0.5, 3.7
Iced drinks	9/38 (24)	11/36 (31)	0.7	0.2, 2.2
Sandwich	22/38 (58)	32/36 (89)	0.2	0.04, 0.7
Dressing (if ate salad)	26/32 (81)	11/11 (100)	0.0	0.0, 2.7
Garnish on sandwich (among nonsalad eaters)	6/6 (100)	13/24 (54)	∞	0.8, ∞
Green salads, or garnish on sandwich	38/38 (100)	24/36 (67)	∞	0.0, ∞

1.0-fold risk for those never ordering salad, to an 8.0-fold risk for those sometimes ordering salad, to an 18.0-fold risk for those always ordering salad ($p < .001$, Mantel-Haenszel trend test).

Among those not eating salad, there was an association between disease and consumption of a sandwich with a lettuce or a tomato garnish ($p = .046$). All cases ordered either a green salad or a sandwich with a garnish. The high correlation between ordering a lettuce and a tomato garnish on a sandwich (.74) precluded analysis of the risk of disease associated with a single vegetable.

Restaurant B: In the study of patrons of restaurant B, 48 (100 percent) of the eligible cases and 43 controls were interviewed. Results indicated that eating salad from the salad bar was associated with a 4.4-fold risk of infection and that ordering either the salad bar or a tossed salad with a main dish was associated with a 6.7-fold risk of disease (Table 3). No other menu item was associated with increased risk.

Patrons of restaurant B were interviewed about the consumption of specific food items from the salad bar; however, no single item could be implicated. The high degree of correlation between ordering two given items from the salad bar precluded further analysis of the risk associated with a single salad bar item. Among those eating salad, salad dressing was not associated with increased risk of infection.

Tracing the Source of Contaminated Fresh Produce

During January 15–30, 71 percent of the cases reported exposures at any one of the three implicated restaurants. The peak of the epidemic curve associated with each of these restaurants occurred the same week. Three of the reported cases were food handlers at restaurant A, and one was a food handler at restaurant B; however, the dates of onset of illness among these employees were within the main part of the epidemic curve, not consistent with transmission to their customers, and the employees did not work at other restau-

TABLE 3—Restaurant B: Study of Food Exposures

Food consumed	Cases exposed* n (%)	Controls exposed* n (%)	Odds ratio	95% confidence interval
Salad bar	40/48 (83)	23/43 (53)	4.4	1.5, 12.8
Salad bar or tossed salad	43/46 (93)	28/41 (68)	6.7	1.6, 32.6
Fish	4/45 (9)	3/40 (8)	1.2	0.2, 7.4
Seafood	3/44 (7)	4/41 (9)	0.7	0.1, 3.9
Meat	25/44 (57)	25/37 (68)	0.6	0.2, 1.7

*If a respondent did not answer a question, the respondent was excluded from analysis for that exposure.

rants. The remainder of the employees at restaurant A were tested and were negative for IgM anti-HAV.

The epidemiologic studies implicated salad as the vehicle of transmission, and results of the study at restaurant A suggested that either lettuce or tomatoes were the contaminated produce. Lettuce was considered the most probable vehicle of transmission because all the implicated types of salads contained lettuce, and because lettuce, as compared with other produce such as tomatoes, is harder to clean thoroughly by rinsing. Since all three of the implicated restaurants served iceberg lettuce, and two of the three restaurants served only iceberg lettuce, the suspected vehicle was iceberg lettuce.

All three restaurants were supplied by the same local fresh produce distributor. (Less than 5 percent of food service establishments in Jefferson County were supplied by the implicated distributor.) At the implicated distributor, no hepatitis A cases were identified among the workers, and the handling of produce was minimal. Produce was generally not unpacked from the crates in which it was shipped and not washed or processed. Therefore, contamination most likely occurred before local distribution.

Shipments of iceberg lettuce that would have been received by the local distributor in Louisville January 11–16 were traced to four domestic suppliers; however, no information was available regarding the farm source of lettuce provided by these suppliers. Just before the outbreak in January of 1988, the importation of Mexican iceberg lettuce increased 10-fold[14] because of drought in the Summer of 1987 in the United States (Figure 2). Mexican iceberg lettuce was observed in Louisville by local United States Department of Agriculture (USDA) inspectors during the last two weeks in January (J. Sullivan, Louisville-Jefferson County Board of Health, personal communication, March 1988). However, there is no information available to determine if Mexican imported produce was received by the local distributor or its suppliers.

Investigation of the Water System

The results of bacteriologic sampling of the water distribution system were within normal limits during the months of January and February 1988. No relationship was found between the geographic distribution of cases and the water distribution system or events such as water company construction, repair, and unusual flow disturbances.

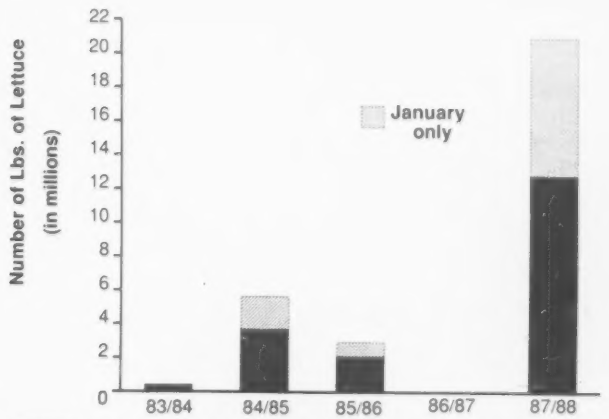

FIGURE 2—Iceberg Lettuce Imported from Mexico, Winter (November–February: 1983–88
SOURCE: Agricultural Marketing Service, US Department of Agriculture

Discussion

This common-source outbreak of hepatitis A was unusual in that there was no single source of exposure from a restaurant or community gathering, nor was there geographic clustering by neighborhood. The epidemic curve, with a sharp peak and most of the cases occurring within a two-week period, indicated a common source of exposure. The age distribution (88 percent of the cases in adults 20 to 59 years old) was atypical of community person-to-person spread and suggested that the persons primarily affected by the epidemic were a "working" cohort.

Case-control studies found that the outbreak was due to contaminated salad served by several restaurants. Because serum specimens of controls were not tested, it is possible that some controls may have been immune or asymptomatic cases. For this possibility to have accounted for the observed associations, there would have to have been an association between exposure, and either immunity or expression of illness. However, without such an assumption (for which there is no basis), the presence of immune controls or asymptomatic cases would tend to produce a conservative estimate of the associations observed.

This is the first report of an outbreak of hepatitis A in the United States associated with a vegetable product which was contaminated prior to distribution to restaurants. At least three restaurants were involved in the outbreak, each accounting for no more than one-third of the cases. Foodhandlers at the restaurants were unlikely to have been the source of the epidemic for two reasons. First, no index case was identified at any of the restaurants. Although serosurveys were not performed at all restaurants, most (76–97 percent) of HAV infections among adults are symptomatic.[14] Second, because the epidemic curve was unimodal, it was unlikely that foodhandlers at each restaurant could have accounted for transmission of HAV to their clientele. Contamination of the produce most likely occurred before local distribution to the restaurants, as there was no hepatitis A case identified among the workers, and there was minimal handling of produce at the local distributor which supplied the three restaurants.

The most likely vehicle of transmission was iceberg lettuce; however, the exact farm source of contaminated lettuce could not be determined. There was no evidence of concurrent outbreaks in the United States associated with lettuce contaminated before local distribution, and the focal nature of the outbreak suggested that either a limited supply of lettuce grown in the United States or imported lettuce had been contaminated. During the peak period of importation in January 1988, Mexican lettuce accounted for only 1 percent of the total US consumption,[15] with any single grower accounting for a small fraction of the total. Contamination of the produce may have occurred in the fields from contaminated water used for growing or irrigation, or from the use of night soil (excrement removed from a cesspool or privy and used as fertilizer). Alternatively, contamination may have occurred during the shipping process if riders were transported in the back of the truck with the produce.

Lettuce contaminated before distribution to food-service facilities also has been associated with two recent large epidemics of shigellosis.[16,17] In these outbreaks, contamination was believed to have occurred in the field or a

warehouse,[16] and in a food-processing plant.[17] Laboratory studies have demonstrated the survival of HAV (in feces) and *Shigella* (in a variety of nonacidic foods) for several weeks at 25 °C.[18,19] These outbreaks underscore the importance of proper sanitation facilities, and good hygiene of workers at each step of the food handling chain.

The timing of the marked increase in imported lettuce in relation to this outbreak, which was associated with fresh produce contaminated before local distribution, raises public health concerns regarding the regulation of imported fresh produce. The purity of water supply used in growing, availability of sanitation facilities to workers, and possible use of night soil by foreign lettuce growers, have not been determined. Imported lettuce is not routinely examined for evidence of fecal contamination at the port of entry to the United States. Imported unprocessed lettuce is exempt from US Customs requirements to label the country of origin on the imported article itself.[20] Although the country of origin must be marked on the outermost container of lettuce that reaches the ultimate purchaser,[20] containers such as supermarket bins and rubberbands holding fresh produce are not subject to marking; if produce should reach the ultimate purchaser without a container, there is no requirement to display the country of origin (M. Amernick, Esq., US Customs, personal communication, October 1989).

Moreover, because there are no federal requirements to label the name of the farm on either imported or domestic lettuce that reaches the ultimate purchaser, and because more than one farm can supply any supplier, it may be impossible to trace lettuce to the growing site during an outbreak, as was the case in this epidemic.

During 1983–88, for the first time in over a decade, there has been a nationwide increase in reported cases of hepatitis A[1–3]; the cause of the increase is not known. This multifocal epidemic of hepatitis A is the first report of an outbreak in the United States associated with a vegetable contaminated before distribution to restaurants, and raises important public health concerns regarding the regulation of both domestic and imported fresh produce.

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Impact of the Human Immunodeficiency Virus Epidemic on Mortality Trends in Young Men, United States

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Abstract: Following a long-term decline, death rates in men 25–44 years of age increased from 212 deaths/100,000 in 1983 to 236 deaths/100,000 in 1987. To assess the impact of human immunodeficiency virus (HIV) infections on this trend and to identify causes that are increasing in association with the HIV epidemic, we analyzed national mortality statistics and compared death rates in states with high and low incidence of acquired immunodeficiency syndrome (AIDS). In 1987, there were 10,248 deaths with HIV infection, AIDS, or conditions in the AIDS surveillance definition assigned as the underlying cause, representing 11 percent of deaths for men in this age group compared to less than 1 percent in 1980. In addition, deaths with other underlying causes, such as other infections, drug abuse, and unknown/unspecified causes, had diverging and higher rates in states with high versus low AIDS incidence. In the absence of deaths due to HIV/AIDS and excess deaths due to these associated conditions, we estimate that death rates for men 25–44 years of age would have been 201–209/100,000 in 1987. For 1987, approximately 70–90 percent of HIV-related deaths were reported through national AIDS surveillance. The HIV epidemic has led to a reversal in mortality trends and to increases in various causes of death for young men. (*Am J Public Health* 1990; 80:1080–1086.)

Introduction

Following a long-term decline, death rates in men 25–44 years of age have begun to increase in the 1980s in the United States (Figure 1). This reversal of mortality trends has coincided with the epidemic of human immunodeficiency virus (HIV) infections and acquired immunodeficiency syndrome (AIDS), which has predominantly affected young men.[1] Deaths due to HIV infection have emerged as a leading cause of years of potential life lost,[2,3] and provisional mortality data from the national vital statistics system indicated that HIV infection ranked fifteenth in 1988 among all causes of death in the United States.[4]

To measure the impact of the HIV epidemic on mortality in young men, we used national mortality data to identify causes of death that are increasing in association with the HIV epidemic and to estimate the number of HIV-related deaths. In these analyses, we examined death rates for a broad spectrum of causes. We focused not only on HIV infection and diseases in the surveillance definition for AIDS but also on other infections and malignancies which have had a suggested link to HIV. In addition, to assess the completeness of national AIDS surveillance, we compared our estimate of HIV-related deaths from vital statistics to mortality data from AIDS case reporting.

Methods

Death Rates 1980–1987

We calculated death rates (deaths/100,000 population) using computer tapes prepared by the National Center for Health Statistics (underlying and multiple causes of death) and the Bureau of the Census.[5,6] We limited our analyses to men, ages 25–44 years, who were United States residents (excluding territories). To identify causes of death associated with HIV infection, we compared mortality trends for four groups of states ranked by AIDS incidence rates, using data from national AIDS surveillance.[7] The highest group (California, Florida, New Jersey, New York, and the District of Columbia) accounts for 59 percent of all AIDS cases reported in men 25–44 years of age, while the lowest group (21 states) accounts for 5 percent of cases. We calculated crude, race-, and age-adjusted death rates. Adjusting for race did not affect the identification of causes that increased more rapidly in states with highest AIDS incidence, and adjusting for age had virtually no effect on comparisons by year or state group. Thus, we present only death rates unadjusted by race or age.

We grouped cause-of-death codes from the Ninth Revision of the International Classification of Disease (ICD-9)[8] into the following categories: HIV infection or AIDS (ICD-9 No. 279.1 for 1980–86, category *042–*044 for 1987*, see below); diseases included in the national surveillance case definition for AIDS (*Pneumocystis carinii* pneumonia, other AIDS-defining infections, Kaposi's sarcoma, non-Hodgkin's lymphomas, encephalopathy, wasting syndrome)[9,10]; other immune deficiency (excludes conditions assigned ICD-9 No. 279.1 in 1980–86, see below); infections not in the AIDS surveillance definition (pneumonia and influenza, septicemia, pulmonary tuberculosis, heart, central nervous system, all other infections); blood disorders (congenital coagulation defects, others); selected cancers with a postulated link to HIV infection (oral-pharyngeal, anal-rectal, other gastroin-

*The notation "category *042–*044" indicates that these are supplemental codes and not part of ICD-9.[9]

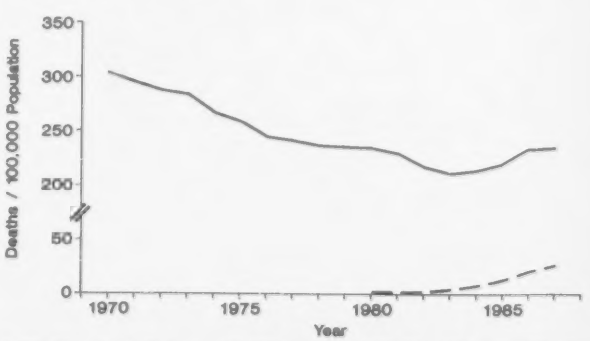

FIGURE 1—Death Rates per 100,000 Population 1970–87 (solid line), and Death Rates per 100,000 for HIV/AIDS and Conditions in AIDS Surveillance Definition, 1980–87 (broken line), Men 25–44 Years of Age, United States

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testinal cancers, Hodgkin's lymphoma)[11–19]; lung cancer, other cancers; injuries (suicide, homicide, unintentional injuries); drug abuse; other system disorders exclusive of above causes (endocrine/metabolic, mental/nervous, cardiovascular, respiratory, digestive, genitourinary, others); and unknown/unspecified causes.**

Beginning in 1983, a pre-existing ICD-9 code (ICD-9 No. 279.1, "deficiency of cell-mediated immunity") was adapted for HIV/AIDS and used through 1986.[20,21] In 1987, a more detailed set of codes (categories *042–*044) for HIV/AIDS was introduced, along with new procedures for assigning HIV/AIDS as an underlying cause.[9,22] In 1986, 52 percent of deaths among men 25–44 years of age which had any listing of HIV/AIDS on death certificates had HIV/AIDS assigned as the underlying cause; in 1987 this increased to 90 percent. For example, a death certificate that mentioned both HIV/AIDS and other diagnoses would be more likely to have HIV/AIDS classified as the underlying cause in 1987 than in 1986. We sought to minimize this discontinuity by considering HIV/AIDS and conditions in the AIDS definition as a single category.

We used the Taylor series method to calculate 95 percent confidence intervals (CIs) for ratios of death rates in 1987 compared to 1980.[23]

Estimate of the Number of HIV-related Deaths, 1987

To estimate the number of HIV-related deaths in 1987, we first determined the number of deaths with certificates that mentioned HIV/AIDS on multiple-cause listings. Among all other deaths, we next considered those with underlying causes identified in the above analyses as increasing in association with the HIV epidemic. For each of these causes, we defined baseline trends by fitting a least median of squares line to the logarithm of death rates for 1970–80.[24] Using this exponential decay model, we then estimated by extrapolation death rates and the number of deaths that would have occurred in 1987 (expected deaths) if baseline trends had remained unchanged. Because data for causes of death were based on a 50 percent sample for some states in 1972, we excluded 1972 from our calculations. Because a new version of the ICD was introduced in 1979, our model included a shift parameter representing the discontinuity that occurred from 1978 to 1979. For some causes, baseline rates during the 1970s were stable or essentially zero or had no consistent trend. When this occurred, we used rates in 1980 as the baseline for estimating expected deaths in 1987 (Table 3). We then calculated excess deaths as the difference between observed deaths (excluding those with certificates that mentioned HIV/AIDS) and expected deaths.

**A listing of codes is available on request from the lead author.

Results

Overall Trends

Deaths with HIV/AIDS or one of the conditions in the AIDS definition listed as the underlying cause increased from 583 (1.9 per 100,000 population) in 1980 to 10,248 (26.6/100,000) in 1987 for men ages 25–44 years in the United States (Figure 1). Such deaths represented 0.8 percent of 73,652 deaths to men ages 25–44 years in 1980 and 11.3 percent of 91,082 deaths in 1987. From 1980 to 1983, total death rates for young men declined from 235.8/100,000 to 212.1/100,000, respectively, and then rose 10 percent to 236.1/100,000 in 1987, returning to a level similar to the rate in 1980.

Among deaths due to HIV/AIDS or conditions in the AIDS definition, the distribution of causes shifted from 1980 to 1986, reflecting the emergence of HIV (Table 1). For example, non-Hodgkin's lymphomas accounted for over two-thirds of such deaths in 1980 but less than 10 percent in 1986. The distribution shifted again in 1987, reflecting implementation of new categories for HIV/AIDS (Table 1). For example, the number of deaths with conditions in the AIDS definition (e.g., *Pneumocystis carinii* pneumonia, Kaposi's sarcoma) assigned as the underlying cause declined from 1986 to 1987, while deaths assigned HIV/AIDS as an underlying cause increased sharply.

Of the 1,381 deaths with conditions in the AIDS definition (but not HIV/AIDS) assigned as the underlying cause in 1987 (Table 1), 136 (10 percent) also mentioned HIV/AIDS on multiple cause listings. In addition to the 8,867 deaths assigned HIV/AIDS as the underlying cause in 1987, there were 979 deaths with HIV/AIDS listed on death certificates but not assigned as the underlying cause. Among these 979 deaths, leading underlying causes were: infections not included in the AIDS definition, 25 percent; conditions in the AIDS definition, 14 percent; drug abuse, 9 percent; cardiovascular disorders (excluding infections and "cardiac arrest"), 8 percent; and lung cancer, 6 percent.

TABLE 1—Deaths with underlying Cause Including HIV/AIDS or Conditions in the AIDS Surveillance Definition, Men 25–44 Years of Age, United States, 1980, 1986, and 1987

Underlying Cause of Death	Number of Deaths			Percent		
	1980	1986	1987	1980	1986	1987
Deficiency of cell-mediated immunity/HIV/AIDS[a]	2	3,850	8,867	0.3	49.4	86.5
PCP[b]	9	1,632	219	1.5	20.9	2.1
Other AIDS Infections[c]	75	1,038	325	12.9	13.3	3.2
Kaposi's sarcoma	44	401	86	7.5	5.1	0.8
Non-Hodgkin's lymphoma	399	770	670	68.4	9.9	6.5
Encephalopathy	54	103	80	9.3	1.3	0.8
Wasting	0	0	1	0.0	0.0	0.0
Total	583	7,794	10,248	100.0	100.0	100.0
Deaths per 100,000 population (for Total)	1.9	20.5	26.3			

[a]ICD-9 No. 279.1 for 1980-1986 (adapted for HIV/AIDS during 1983-1986) and category *042–*044 for 1987 (see text).
[b]*Pneumocystis carinii* pneumonia.
[c]Includes other infections in national surveillance definition for AIDS.[10]

Death Rates and State AIDS Incidence

As expected, death rates for HIV/AIDS or for conditions included in the AIDS surveillance definition increased more in states with the highest AIDS incidence compared with other state groups, and by 1987 ranged from 56.7/100,000 (5,922 deaths) in the five states with the highest AIDS incidence to 8.8/100,000 (701 deaths) in the 21 states with the lowest AIDS incidence. In addition to conditions in the AIDS definition, five other categories for underlying cause-of-death had a divergence in rates from 1980 through 1987 for different state groups: infections not included in the AIDS definition, unknown/unspecified causes, drug abuse, blood disorders other than congenital coagulation defects, and other immune deficiency (Table 2). Mortality trends for the first three of these causes are shown in Figure 2 for states with the highest and lowest AIDS incidence (rates in states with intermediate AIDS incidence were intermediate). For one of these underlying causes, drug abuse, the death rate declined slightly (8.2 to 7.4/100,000) from 1986 to 1987 in the states with the highest AIDS incidence (Figure 2C); however, when multiple cause-of-death listings were used to include deaths with any mention of drug abuse, death rates for drug abuse increased in these states from 1986 to 1987 (19.0 to 22.4/100,000). This difference in trends between underlying and multiple causes-of-death appears to reflect a shift from 1986 to 1987 in the selection of HIV/AIDS or drug abuse as the underlying cause when both are listed on death certificates. For one other category, respiratory conditions (excluding respiratory infections), rates increased more in the highest AIDS incidence states by 1987 (Table 2), but the pattern of divergence for the years between 1980 and 1987 was less clear.

Among infections not included in the AIDS definition, death rates increased from 1980 to 1987 in each subcategory in states with the highest AIDS incidence, while no change, declines, or smaller increases were observed in states with the lowest AIDS incidence. Pneumonia and influenza was the subcategory with the highest death rate (predominantly "bronchopneumonia, organism unspecified" or "pneumonia, organism unspecified"), while the greatest relative increases were seen for septicemia and pulmonary tuberculosis (Table 2).

Death rates for drug abuse had a relative increase that was similar in the lowest AIDS incidence states as in the highest AIDS incidence states (Table 2); however, the absolute difference in rates between the highest and lowest AIDS incidence states widened from 2.7/100,000 in 1980 to 4.9/100,000 in 1987.

Among deaths due to blood disorders other than congenital coagulation defects, the increase in the highest compared with lowest AIDS incidence states was due primarily to unspecified coagulation defects, thrombocytopenia, and agranulocytosis. Among deaths due to non-infectious respiratory conditions, this increase was observed for deaths due to "pulmonary congestion" and "alveolar pneumonopathies."

The pattern of increasing death rates in states with the highest compared with lowest AIDS incidence was not observed for the other cause-of-death groups we examined. To determine whether causes of death not recognized using these categories had such a pattern, we further reviewed numbers of deaths in 1980 and 1986 using ICD-9 codes at the major subheading level (e.g., the first three digits of the four-digit ICD-9 codes), after excluding conditions which accounted for <25 deaths in the two years combined. This review did not identify additional underlying causes with this pattern, with the possible exception of diabetes mellitus, which increased slightly from 2.2/100,000 in 1980 to 2.9/100,000 in 1987 in the highest AIDS incidence states, while remaining stable during this period in the lowest AIDS incidence states.

Race-specific Trends

For both White and Black men, lowest overall death rates for the 25–44 year age group were in 1983. For White men, death rates declined from 204.8/100,000 in 1980 to 187.3/100,000 in 1983, then rose 9 percent to 204.1/100,000 in 1987. For Black men, death rates declined from 510.3/100,000 in 1980 to 431.6 in 1983, then rose 19 percent to 513.5/100,000 in 1987. In 1987, death rates for HIV/AIDS and conditions in the AIDS definition for men in this age group were 22.2/100,000 for

TABLE 2—Deaths Rates for Causes Increasing in Association with the HIV Epidemic, by State Group (excluding HIV/AIDS and conditions in AIDS definition), Men 25–44 Years of Age, United States, 1980 and 1987

	Highest AIDS Incidence States				Lowest AIDS Incidence States			
	Death Rate[a]				Death Rate[a]			
	1980	1987	Rate Ratio[b]	(95% CI)[c]	1980	1987	Rate Ratio[b]	(95% CI)[c]
Infections not in AIDS definition	7.63	11.74	1.5	(1.4, 1.7)	5.20	5.13	1.0	(0.9, 1.1)
Septicemia	0.46	1.65	3.6	(2.5, 5.1)	0.63	0.87	1.4	(0.9, 2.0)
Pneumonia, influenza	3.85	5.42	1.4	(1.2, 1.6)	2.53	2.27	0.9	(0.7, 1.1)
Pulmonary tuberculosis	0.32	0.85	2.7	(1.7, 4.2)	0.19	0.10	0.5	(0.2, 1.3)
Central nervous system	0.36	0.68	1.9	(1.2, 2.9)	0.22	0.23	1.0	(0.5, 2.0)
Heart	0.44	0.62	1.4	(0.9, 2.1)	0.28	0.31	1.1	(0.5, 2.0)
Others	2.20	2.52	1.1	(0.9, 1.4)	1.34	1.35	1.0	(0.8, 1.3)
Drug abuse	4.05	7.41	1.8	(1.6, 2.1)	1.33	2.53	1.9	(1.5, 2.5)
Blood disorders[d]	0.60	1.18	2.0	(1.4, 2.8)	0.60	0.83	1.4	(0.9, 2.1)
Other immune defects	0.05	0.94	19.3	(7.1, 52.5)	0.07	0.19	2.5	(0.9, 7.0)
Unknown/unspecified	4.40	13.66	3.1	(2.8, 3.5)	3.34	2.17	0.6	(0.5, 0.8)
Respiratory[e]	2.20	3.11	1.4	(1.2, 1.7)	2.10	2.19	1.0	(0.8, 1.3)

[a](Deaths/100,000 population).
[b]Rate ratio = (Rate in 1987)/(Rate in 1980).
[c]95% Confidence Intervals.
[d]Excludes congenital clotting defects.
[e]Excludes infections.

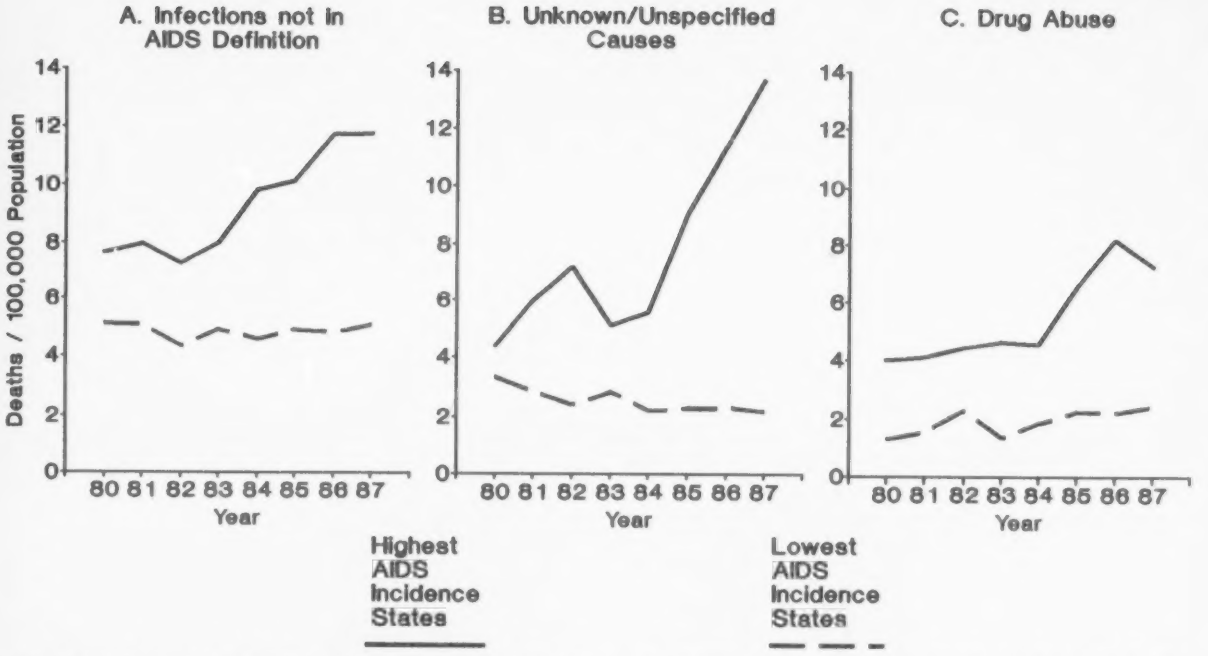

FIGURE 2—Death Rates per 100,000 Population, Men 25–44 Years of Age, Deaths due to Selected Causes with Rates that Diverged from 1980 to 1987 in the Highest Compared with Lowest AIDS Incidence States, United States, 1980–87

White men and 67.1/100,000 for Black men. For infections not in the AIDS definition, death rates increased from 4.2/100,000 in 1980 to 5.3/100,000 in 1987 for White men, and from 20.7 to 26.1/100,000, respectively, for Black men.

Estimate of HIV-related Deaths

In 1987, the estimate of HIV-related deaths in men 25–44 years of age ranged from 10,290 to 13,350, based on the number of excess deaths for those causes identified above as increasing in association with the HIV epidemic (Table 3). The model used for calculating excess deaths is illustrated in Figure 3 for pneumonia and influenza. The minimum estimate of HIV-related deaths (10,290 deaths) represents the number with mention of HIV/AIDS on death certificates and excess deaths due to conditions in the AIDS definition (Table 3). An intermediate estimate of HIV-related deaths, adding those due to infections not in the AIDS definition and other immune defects, is 11,470. The maximum estimate (13,350) includes excess deaths for all causes increasing in association with the epidemic. The 9,838 deaths with certificates that mentioned HIV/AIDS on multiple-cause listings represented 74–96 percent of estimated HIV-related deaths. This compares to 9,430 deaths occurring in 1987 among men 25–44 years of age and reported to national AIDS surveillance through December 1989, representing 71–92 percent of estimated HIV-related deaths for men in this age group.

In the absence of deaths due to HIV/AIDS and excess deaths due to these associated causes, the death rate in 1987 for men 25–44 years of age would have been 201 to 209/100,000, a decline of 11–15 percent from 1980 using the range of estimates of HIV-related deaths. Instead, death rates showed no net decline from 1980 to 1987.

TABLE 3—Estimate of HIV-related Deaths Men 25–44 Years of Age, United States, 1987

	Number of Deaths	Cumulative Total[a]
HIV/AIDS listed on death certificate	9,838	
HIV/AIDS not listed on death certificate, excess deaths with underlying cause[a]		
Other conditions in AIDS definition		
Pneumocystis carinii pneumonia[b]	210	
Other definition infections[c]	200	
Kaposi's sarcoma[b]	30	
Non-Hodgkin's lymphoma[c]	250	
Encephalopathy[d]	0	10,290
Infections not in AIDS definition		
Septicemia[c]	140	
Pneumonia & influenza[c]	680	
Pulmonary tuberculosis[c]	80	
Other infections[c]	250	
Other immune defects[b]	30	11,470
Drug abuse[d]	560	
Blood disorders[d,e]	80	
Unknown/Unspecified[d]	1,010	
Respiratory[b,f]	230	13,350

[a]Rounded to nearest 10 deaths.
[b]Death rate stable or near zero during 1970s, excess deaths estimated using rate in 1980 as baseline.
[c]Excess deaths estimated using trend during 1970s as baseline.
[d]Death rate had no consistent trend during 1970s, excess deaths estimated using rate in 1980 as baseline.
[e]Excludes congenital coagulation defects.
[f]Excludes infections.

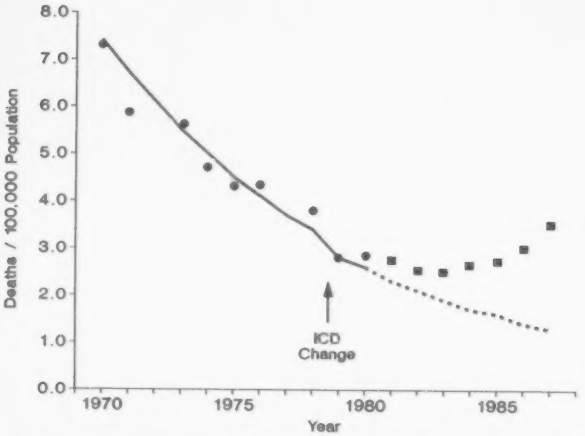

FIGURE 3—Death Rates per 100,000 for Pneumonia and Influenza, Men 25–44 Years of Age, United States, 1970–87
Circles represent observed rates for 1970–80. Squares represent observed rates for 1981–87 (excluding deaths with diagnoses coded as "deficiency of cell-mediated immunity, *Pneumocystis carinii* pneumonia or Kaposi's sarcoma in 1980–86 or as HIV/AIDS in 1987). Solid line represents baseline for rates in 1970–80 and dashed lines predicted rates for 1981–87.

Discussion

The HIV/AIDS epidemic has led to an increase in death rates for young men in the United States. Our findings are similar to those in England and Wales, where death rates have also recently increased in young men, primarily because of causes associated with HIV.[25] By 1987, deaths due to HIV/AIDS and conditions in the surveillance definition for AIDS accounted for more than 10 percent of deaths among men 25–44 years of age. Among Black men, death rates for these conditions were three times the rates for White men in 1987, reflecting disparities in AIDS incidence.[26] In addition, by comparing cause-specific mortality trends in states with high versus low incidence of AIDS, we have identified other causes of death that are increasing in association with the HIV epidemic.

Most notable is an increase in deaths due to infections that are not part of the surveillance definition for AIDS, such as pneumonia, septicemia, and pulmonary tuberculosis. This is consistent with clinical and epidemiologic observations that persons with HIV-related immune dysfunction are susceptible to a broad spectrum of infectious complications in addition to the "opportunistic" infections included in the AIDS surveillance definition.[27] Increases in morbidity and mortality due to these conditions have been noted most prominently in association with intravenous drug use,[28–31] although the risk of severe bacterial infections among HIV-infected persons is not limited to intravenous drug users.[32] The increase in deaths due to pulmonary tuberculosis is consistent with increases in tuberculosis noted in association with HIV and AIDS.[33–37]

The observation that deaths due to blood disorders such as agranulocytosis, thrombocytopenia, and unspecified coagulation defects increased in states with the highest AIDS incidence is consistent with recognized hematologic complications of HIV infection.[38] The finding that immune deficiencies other than cell-mediated immune deficiency (ICD-9 No. 279.1) are increasing is not surprising, given the spectrum of immune defects caused by HIV infection[39] and possible variations in the reporting and coding of HIV-related immune deficiencies.

The divergence in trends for deaths due to drug abuse in states with the high and low AIDS incidence probably reflects differences in both levels of illicit drug use and HIV infection among drug users, particularly in New York and New Jersey.[40] We cannot discern, however, to what extent the upward trend in death rates for drug abuse reflects trends in illicit drug use independent of the HIV epidemic. This possibility was considered but discounted by Stoneburner, *et al*, who studied mortality trends in New York City. While deaths in narcotic users increased from 1979 to 1986, overdose deaths did not increase, which should have occurred if narcotic use had increased.[28]

Similarly, for all causes that are increasing in association with the HIV epidemic, it is not possible to measure by death certificates alone what proportion of the increase is attributable to HIV infection. In some cases HIV infection may be unrecognized, and in others physicians may be unwilling to specify AIDS or HIV on death certificates. For example, the increase in deaths classified as due to "pneumonia" or non-infectious respiratory conditions may reflect the use of euphemisms for AIDS or *Pneumocystis carinii* pneumonia, or the lack of a full diagnostic evaluation. Likewise, the marked increase in deaths with unspecified cause in highest AIDS incidence states may reflect a failure to diagnose HIV infection or, alternatively, incomplete postmortem testing at the time of death certificate coding. Conversely, the categories we used for AIDS indicator diseases may capture some deaths that are unrelated to HIV infection. In 1980, over 500 deaths were due to conditions in the AIDS definition. While a few of these deaths may have been due to HIV infection, the majority most likely reflect the background occurrence of these conditions.

In addition to cancers in the AIDS definition, other cancers have had a suggested link to HIV infection.[11–19] We examined trends in deaths due to several of these cancers (Hodgkin's lymphoma, oral-pharyngeal cancer, anal-rectal cancer) but did not observe an increase in death rates or a divergence in trends for states with high and low AIDS incidence. This does not exclude an etiologic link between HIV and these conditions but suggests that if there is a link, it has not had a substantial effect on mortality trends. Similarly, population-based studies have not found statistically significant increases in the incidence of Hodgkin's lymphoma when high and low AIDS incidence areas were compared or when trends were examined for married and unmarried men.[41–44]

Increased rates of suicide among men with AIDS compared to other men have been described in New York and California.[45,46] Nationally, we observed that suicide and HIV/AIDS were listed together for 33 deaths in 1987, but we did not observe a difference in suicide trends for states with a high versus low AIDS incidence. This suggests that suicides among men with HIV infection has not had a substantial effect on suicide trends.

We have estimated that up to 13,350 HIV-related deaths occurred in 1987 among men 25–44 years of age, based on the number of deaths with HIV/AIDS listed on death certificates and on excess deaths for conditions that are increasing in association with the HIV epidemic. This estimate should be considered approximate for several reasons. We assumed that mortality trends during the 1970s would have continued unchanged during the 1980s in the absence of the HIV epidemic; for some causes we were unable to define a baseline trend in the 1970s; and some HIV-related deaths may be attributed to causes not included in our estimate.

New codes introduced in 1987 improve the classification of HIV-related deaths in national vital statistics. Nonetheless, our findings indicate that death certificates listing HIV/AIDS do not describe the full extent of HIV-related mortality. Similarly, our estimate of HIV-related deaths exceeds the number of deaths reported through national AIDS surveillance. Underreporting will affect both systems if HIV/AIDS is not diagnosed or if physicians are unwilling to report the diagnosis to health agencies. Also, HIV/AIDS may not be listed on death certificates in instances where physicians judge the cause of death to be unrelated to HIV infection. AIDS surveillance undercounts HIV-related deaths to the extent that AIDS cases and subsequent deaths are underreported and that persons with HIV-related disease do not meet surveillance criteria for AIDS. Thus, our estimate that 70–90 percent of HIV-related deaths in 1987 among men 25–44 years of age were detected by AIDS surveillance (which is somewhat higher than a comparable estimate for England and Wales[47]) indicates that reporting for those with disease meeting surveillance criteria must be at least this complete.

The concept of surveillance is increasingly being expanded beyond case reporting to encompass analyses of other data resources, such as vital statistics.[48] Comparison of mortality trends in areas with varying AIDS incidence provides a useful complement to AIDS surveillance in describing both the spectrum of conditions associated with HIV infection and the impact of the HIV epidemic on mortality trends.

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AAA Pedestrian Protection Program Names Four Safest Communities in 1989

The American Automobile Association recently released the results of its 51st annual Pedestrian Protection Program which recognizes communities and states that have made outstanding achievement in pedestrian safety. The four locations in America judged to be the safest places to be a pedestrian are: the State of Wisconsin; Henrico County, Virginia; San Jose, California; and Rocklin, California. According to Bill Bowes, AAA manager of community programs, "these communities have made strong efforts in traffic safety programs, including education at all levels of their population. They work hard at making sure their streets are safe."

The AAA Pedestrian Protection Program is designed to encourage cities and states to teach pedestrian safety and to strictly enforce related laws. Communities also work with AAA to improve their programs. The participating cities are grouped by population size and graded on safety programs, enforcement levels, traffic engineering, pedestrian deaths, and injury rates.

San Jose had a pedestrian death rate of 2.3 per 100,000 population and an injury rate of 54/100,000 in 1989. Comparable rates for the other winners were: Wisconsin, 1.4 deaths and 43 injuries/100,000; Henrico County, 1.9 deaths and 17 injuries/100,000; Rocklin, no deaths and 17 injuries/100,000.

Educational programs in schools and senior centers teach children, adults and senior citizens pedestrian safety. San Jose's active enforcement program led to 355 tickets issued to pedestrians crossing against traffic signals. Most pedestrians killed in the United States either violated traffic laws or behaved carelessly, AAA said. Since the Pedestrian Protection Program began in 1937, pedestrian fatalities have dropped from 15,500 per year to 8,800 in 1989. AAA also distributed a fact sheet about pedestrians, which included the following:

- The first recorded traffic fatality in the US occurred in 1899 in New York City. The victim was a pedestrian.
- The economic loss from pedestrian accidents in the US is estimated at more than $1 billion annually.
- At least 90 percent of all children injured enroute to or from school were struck where no type of special school crossing protection (safety patrols, adult guards, police officers) was in use.
- Pedestrians under age 15 and over age 64 account for 41 percent of all pedestrian fatalities; 17 percent of pedestrian deaths and 37 percent of pedestrian injuries are to those under age 15; older adults account for 24 percent of pedestrian deaths and 10 percent of pedestrian injuries.
- Pedestrian deaths are three-fold greater than combined air, marine and rail transport deaths.

For further information, contact AAA, 1000 AAA Drive, Heathrow, FL 32746-5063. Tel: (407) 444-8000.

Premarital Screening for Antibodies to Human Immunodeficiency Virus Type 1 in the United States

LYLE R. PETERSEN, MD, CAROL R. WHITE, MT, AND THE PREMARITAL SCREENING STUDY GROUP*

Abstract: To evaluate premarital human immunodeficiency virus (HIV) screening as an approach to AIDS prevention in the United States, we determined the HIV antibody seroprevalence in marriage license applicants in eight areas by blinded testing of blood specimens routinely collected for syphilis serology. The seroprevalences were 0.0–0.4 percent in women and 0.0–1.1 percent in men. We also examined the impact of mandatory premarital HIV screening on marriage rates in Louisiana and Illinois. In 1988, after screening began, 9 percent and 16 percent fewer marriage licenses than in the previous two years were issued in Louisiana and Illinois, respectively. We estimated that mandatory premarital screening, if adopted nationally, would cost $167,230,000. We conclude that compared with other HIV prevention programs mandatory premarital screening would be expensive and would probably have a minor impact on the HIV epidemic. (*Am J Public Health* 1990; 80:1087–1090.)

Introduction

Mandatory premarital screening for human immunodeficiency virus type 1 (HIV-1) antibodies, a measure proposed to reduce heterosexual and perinatal transmission of HIV infection, would require testing of 2.4 million couples annually if adopted in every state.[1] Arguments for and against mandatory screening focus on public health, scientific, ethical, political, and financial considerations.[2–4] A recent analysis arguing that premarital screening would not be cost effective was based on HIV seroprevalence rates among blood donors because rates in marriage license applicants were unknown.[2] However, HIV seroprevalence may be lower in blood donors than in marriage license applicants because these two groups are demographically dissimilar[5,6] and persons at risk for HIV infection are actively discouraged from donating blood.[7]

To evaluate the use of premarital HIV screening as an approach to HIV prevention, we determined the HIV antibody seroprevalence in marriage license applicants in eight areas. In addition, we examined the impact of mandatory premarital screening on the number of marriages in Louisiana and Illinois.

Methods

Premarital syphilis and/or rubella serologic testing is required in 20 states. From November 1985 to December 1988, we conducted blinded (test results unlinked to personal identifiers) HIV seroprevalence studies using blood specimens submitted to local or state health department laboratories for mandatory premarital syphilis screening in Alabama, California (Alameda County and City of Long Beach only), Connecticut, Georgia, Mississippi, New Mexico (Bernalillo County only), and Oklahoma. Areas for the blinded surveys were chosen on the basis of availability of specimens and desire by local or state health officials for a blinded survey. In each blinded survey, excess sera were tested for HIV antibody after the routine syphilis serologic testing was performed and personal identifiers were removed. In some locations, limited demographic data were available. No sera were collected specifically for HIV antibody testing. In Alameda County, specimens were submitted by seven public health clinics; in the other locations, by private physicians, public health clinics, the Indian Health Service, and hospital laboratories. In California, marriage license applicants who had been living together for ≥6 months could choose an option (23 percent of applicants in 1987) not requiring syphilis testing.

For each blinded survey location, comparison HIV seroprevalence data were obtained from the US Department of Defense military applicant screening program[8,9] and adjusted to the national age and gender distribution of marriage license recipients reported to the National Center for Health Statistics (NCHS).[5] The cumulative incidences of acquired immunodeficiency syndrome (AIDS) in adults (≥13 years of age) from 1978 to 1988 were computed from surveillance data from the Centers for Disease Control (CDC) and population denominators from the 1980 census.

Louisiana and Illinois began mandatory premarital HIV screening on January 1, 1988. Because only positive results were reported to the Illinois Department of Public Health, statewide seroprevalence rates in applicants could only be estimated by comparing the number of reported seropositive applicants to the total number of marriage licenses issued in Illinois during 1988. A description of the Illinois program has recently been published.[10] HIV antibody test results were not reported in Louisiana. Although statewide seroprevalence in applicants could not be directly measured in either state, seroprevalence information was obtained from six blood centers within these states that offered HIV antibody testing as a service to marriage license applicants.

The impact of the Illinois and Louisiana mandatory screening laws on marriage rates was evaluated using final data on the annual number of marriages from 1980 to 1985[11] and provisional NCHS data from 1986 through 1988 for 49 states and the District of Columbia.[12]

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TABLE 1—Prevalence of Antibody to the Human Immunodeficiency Virus Type 1 in Marriage License and Military Recruit Applicants and Cumulative Incidence of AIDS, by State, County, or City

State	Date	Marriage License Applicants % Seropositive (No. Tested)				Military Recruits % Seropositive*			Adult AIDS Incidence†		
		Females	Males	Total	95 % CI	Female	Male	Total	Female	Male	Total
Alabama	11/87-1/88	NA	NA	0.21 (4694)	0.11, 0.40	0.09	0.16	0.12	29	256	136
California‡	11/85-8/87	0.25 (786)	1.09 (736)	0.66 (1522)	0.34, 1.25	0.23	0.74	0.48	50	1648	819
California§	3/87-8/87	0.00 (137)	0.00 (140)	0.00 (277)	0.00, 1.08	0.00	0.47	0.23	52	3128	1544
Connecticut	1/88-4/88	0.39 (761)	0.27 (733)	0.33 (1494)	0.12, 0.82	0.11	0.45	0.28	138	650	381
Georgia	10/87-10/88	0.17 (2893)	0.29 (2722)	0.23 (5663)	0.13, 0.40	0.06	0.58	0.32	38	662	342
Mississipi	2/88-12/88	0.00 (1167)	0.00 (1059)	0.00 (2226)	0.00, 0.13	0.01	0.15	0.08	20	208	109
New Mexico¶	12/87-6/88	0.00 (222)	0.32 (312)	0.18 (541)	0.01, 1.18	0.00	0.11	0.05	23	441	225
Oklahoma	3/87-7/88	0.07 (4161)	0.05 (3920)	0.06 (8144)	0.02, 0.15	0.00	0.17	0.09	14	251	128

*Age- and sex-adjusted to marriage license applicants, 10/85-6/88
†Cumulative incidence per 1,000,000 adult (≥13 years of age) population 1978 to 1988
‡Alameda County, 11/85–9/86 and 1/87–8/87
§City of Long Beach
¶Bernalillo County (Albuquerque), New Mexico

The potential cost of premarital HIV screening, if adopted nationally, was based on the estimated 2,389,000 couples who married in 1988.[12] Based on the fact that observed premarital applicant HIV prevalence rates were more similar to those of military applicants than to those of other national population groups tested for HIV (e.g., blood donors), we assumed that the national HIV seroprevalence rates in marriage license applicants were equal to national rates for military applicants age-adjusted to marriage license recipients (0.31 percent men, 0.10 percent women). We assumed the average cost of HIV testing and counseling would be $35. This cost was similar to overall cost estimates used by others[2,10] and slightly lower than the average cost of HIV counseling and testing in public sites funded by the CDC ($42 for seronegative and $92 for seropositive persons). The sensitivity and specificity of sequential EIA and Western blot were assumed to be 99.0 percent and 99.999 percent, respectively. The specificity of 99.999 percent was used because the false-positive rate of the sequential enzyme immunosorbent assays (EIA) and Western blot has been shown to be less than 1 in 100,000 under optimal quality assurance conditions.[13,14]

Specimens from the blinded surveys and the HIV screening performed at blood centers in Louisiana and Illinois were tested for HIV antibody by licensed EIA. Repeatedly reactive specimens from the Alameda County and Long Beach Health Departments were confirmed by immunofluorescence assay (IFA), and repeatedly reactive specimens obtained elsewhere were confirmed by Western blot. Specimens confirmed by Western blot were considered positive at the Tulane Blood Center and at the American Red Cross Blood Services Heart of Illinois Region (Peoria, IL) if p24, p31 and either gp41 or gp160 bands were present. At the other centers, at least two of the following three bands were required: p24, gp41, and gp 120/160. These two criteria have been shown to yield similar results in asymptomatic persons.[15]

Results

Blinded Premarital HIV Seroprevalence Studies

Seroprevalences were from 0 to 0.7 percent (Table 1). Gender-specific marriage license applicant HIV antibody prevalences were 0–0.4 percent for women and 0–1.1 percent for men. Data from New Mexico, Oklahoma, Connecticut, and Georgia indicated HIV seroprevalence may be highest in persons 20 to 39 years of age (Table 2). The observed HIV antibody prevalence in Black marriage license applicants was higher than that of non-Hispanic White applicants in Georgia (Table 3). Race-specific data were not gathered in other states except Oklahoma where the number of non-Whites was small.

States with Mandatory Premarital Testing

In Illinois, 16 percent fewer marriage licenses were issued in 1988 than the average in 1986 and 1987, the two years before mandatory premarital screening was instituted (Table 4). Correspondingly, the number of marriages increased by 7 percent in states surrounding Illinois (Table 4). In Louisiana, there were 9 percent fewer marriages in 1988 than in 1986 and 1987—similar to the 7 percent decrease in its surrounding states (Table 4). The decrease in Louisiana only occurred during the period of screening, however. Compared to similar periods in the two preceding years, the number of marriage licenses issued in Louisiana decreased by 19 percent from January to June when the law was in effect, but from July to December the number remained at levels similar to those of the two preceding years (+0.5 percent). In contrast, the number of marriages decreased during the first (−18 percent) and second (−14 percent) halves of 1988 in Illinois where the law was in effect for the entire year. The decrease in marriages in states bordering Louisiana was due entirely to a decrease in marriages in Texas, a state with a population nearly four times that of Louisiana.

In Illinois, a total of 80,490 marriage licenses were issued in 1988; 26 persons (0.02 percent of 160,980 persons) with

TABLE 2—Prevalence of Antibody to Human Immunodeficiency Virus Type 1 in Marriage License Applicants, by Age and Location, 1988

Age* (years)	% Seropositive (No. tested)			
	Oklahoma Mar 87–Jul 88	New Mexico† Dec 87–Jun 88	Connecticut Oct 87–Apr 88	Georgia Oct 87–Oct 88
12–19	0.00 (1644)	0.00 (47)	0.00 (55)	0.00 (727)
20–29	0.07 (4110)	0.00 (227)	0.41 (731)	0.27 (3003)
30–39	0.08 (1288)	0.64 (156)	0.53 (378)	0.26 (1159)
≥40	0.00 (874)	0.00 (82)	0.00 (250)	0.30 (665)

*1/228 persons with unstated age in Oklahoma was seropositive, 0/29 persons in New Mexico, 0/80 persons in Connecticut, and 0/109 persons in Georgia with unknown age were seropositive.
†Bernalillo County (Albuquerque).

TABLE 3—Prevalence of Antibody to Human Immunodeficiency Virus Type 1 in Marriage License Applicants, by Race/Ethnicity and Gender, Georgia, 1987–88

Race/Ethnicity	% Seropositive (No. Tested) Women	Men	Total	95% CI
White, non-Hispanic	0.10 (1939)	0.11 (1845)	0.11 (3784)	0.04–0.30
Black	0.22 (915)	0.72 (829)	0.46 (1744)	0.21–0.94
Other	2.94 (34)	0.00 (43)	1.30 (77)	0.07–8.01

None of 58 persons with unstated race were seropositive.

TABLE 4—Number of Marriages in Illinois, States Bordering Illinois, Louisiana, States Bordering Louisiana, and All Other States,* 1985–88†

States	Average 1986–87	1988	Difference (%)
Illinois	95834	80490	−15344 (−16.0)
Border Illinois‡	159986	171052	+11066 (+6.9)
Louisiana	36714	33527	−3187 (−8.7)
Border Louisiana§	251988	234194	−17794 (−7.1)
Other States	1757026	1744556	−12470 (−0.7)

*Excluding Nevada and including District of Columbia.
†Based on provisional data from the National Center for Health Statistics. Final data may vary from provisional data.
‡Indiana (+11 percent), Wisconsin (+9 percent), Iowa (+7 percent), Missouri (+1 percent), Kentucky (+5 percent).
§Mississippi (+5 percent), Arkansas (+11 percent), Texas (−11 percent).

positive HIV antibody tests were reported. It is unknown how many of these seropositive persons were pairs.

Seroprevalences from six blood centers with HIV antibody screening services for premarital applicants in Louisiana and Illinois were 0.0 percent to 0.9 percent (Table 5). Nine of the 10 seropositive marriage license applicants identified from the six blood centers had partners who were also tested at the blood centers; all were seronegative.

National Premarital Screening Projections

A total of 9,795 persons (7,406 males and 2,389 females) of the 2,389,000 couples marrying would have HIV antibody if the seroprevalence in that population were the same as that for military applicants. At $35 per person screened, the cost would be $167,230,000. If the overall sensitivity and specificity of HIV testing were 99.0 percent and 99.999 percent, 9,697 persons (7,332 men and 2,365 women) would be correctly identified as seropositive at a cost of $17,246 per seropositive person detected. The overall predictive value positive of the sequential EIA and Western blot would be 99.5 percent under optimal quality assurance conditions. There would be 48 persons with false-positive test results.

Discussion

Observed marriage license applicant HIV seroprevalences varied considerably—from less than 0.1 percent in two states to 0.66 percent in Alameda County, California—and areas with high HIV antibody prevalences generally had the highest cumulative AIDS incidences. These data suggest that the national HIV seroprevalence in marriage license applicants is of the same order of magnitude of that in military applicants (0.21 percent [adjusted to the age and gender distribution of marriage license recipients]) than to that in blood donors (0.012 percent).[16] Our data should be interpreted with caution, however, as there were few marriage license applicants in some study locations, these locations were not randomly chosen, and the effect of selection biases could not be fully determined.

Our data from the blinded seroprevalence studies of specimens collected for routine premarital syphilis screening suggest that the seroprevalence in marriage license applicants was much higher than that indicated by our data and those of Turnock and Kelly[10] gathered from reporting of seropositive persons identified by mandatory screening in Illinois. The calculated seroprevalence (<0.02 percent) based on reporting in Illinois was half that found in the blinded study in Oklahoma, a state with a lower cumulative incidence of AIDS in adults (128 vs 203 per million adult population) and a lower HIV prevalence in military applicants (0.09 percent vs 0.24 percent). In addition, although the numbers of premarital applicants tested in the two Illinois blood banks reported here were relatively few, the observed seroprevalences were higher than the calculated statewide seroprevalence despite the fact that these two blood banks were located in relatively low AIDS incidence areas. These discrepancies could have been due to underreporting of seropositive persons to the Illinois Department of Public Health; moreover, persons who knew they were infected or were at increased risk for HIV infection might have deferred marriage or traveled to adjacent states to marry.

Marriage rates decreased in Louisiana and Illinois after the mandatory screening laws took effect. Although we could not determine what proportion of this decrease was due to HIV screening, in Louisiana, the decrease in marriages occurred only in the first half of 1988 when the screening requirements were in effect. In Illinois, the number of marriages increased in adjacent states, and others have shown that some of this increase was due to border crossing

TABLE 5—Prevalence of Antibody to Human Immunodeficiency Virus Type 1 in Marriage License Applicants Tested in Blood Centers, Illinois and Louisiana, 1988

Blood Center	Area Served	Charge per Test	Dates	% Seropositive (No. Tested) Total	95% CI
Aurora Area Blood Bank	Aurora, IL/8 counties	$40	Jan–Dec	0.06 (1553)	0.00, 0.42
Am. Red Cross, Peoria	Peoria, IL	$20†	Jan–Dec	0.05 (1951)	0.00, 0.33
Louisiana Blood Center	Shreveport/11 parishes	$20‡	Jan–Jul	0.00 (539)	0.00, 0.55
Blood Center SE Louisiana	New Orleans/13 parishes	$15‡	Jan–Jul	0.06 (3371)	0.01. 0.24
Tulane Blood Center	New Orleans/2 parishes	None§	Jan–Jul	0.10 (1914)	0.40, 0.50
Charity Hospital	New Orleans	None§	Jan–Jul	0.90 (444)	0.29, 2.45

†Red Cross provided testing services for the Peoria County Health Department.
‡If applicant donated blood for transfusion; $5 at Louisiana Blood Center and $10 at Blood Center for SE Louisiana for EIA and WB.
§One partner required to donate blood for transfusion.

by Illinois residents to avoid screening.[10,17] These data suggest that the efficacy of mandatory screening in identifying seropositive persons may be less than expected, especially if alternatives exist, such as marrying in adjacent states.

Premarital HIV screening would undoubtedly have little overall effect on heterosexual and perinatal transmission because most unmarried adults are sexually active[18] (only 63 percent of first births in the United States are conceived within marriage)[19] a negative premarital antibody test does not guarantee that a person will remain seronegative throughout the duration of the marriage; some persons counseled after a positive antibody test may not change their sexual or childbearing behavior to negate the risk of future HIV transmission; and groups with high seroprevalence may be less likely to marry before heterosexual or perinatal transmission occurs. For example, in New York State, the HIV seroprevalence in Black childbearing women is over 10 times that of White women,[20] and national figures indicate that 74 percent of first births to Black women are nonmaritally conceived compared to 31 percent in White women.[19] Data suggest that 75 percent of children with perinatally acquired AIDS in New York City are born to unmarried women.[2] Therefore, premarital HIV screening cannot be considered a substitute for other public health strategies to reduce perinatal and heterosexual HIV transmission.

The cost of mandatory HIV screening, if adopted nationally, is likely to be high relative to other HIV prevention programs. We estimated the cost to be $167,230,000, nearly 80 percent as high as the $211,889,000 Congressional appropriation to CDC for all public health HIV control measures for fiscal year 1988. The average cost of identifying a seropositive person was estimated to be $17,246, compared to approximately $925 in public sites funded by CDC (unpublished data). The actual cost of identifying a seropositive person may be much higher than we estimated because persons at high risk for HIV infection may decline to marry or may travel to adjacent states to avoid the screening requirements. In Illinois, the actual cost was estimated to be $312,000 per seropositive person during the first six months of screening.[10] Although the cost of mandatory screening could be borne by marriage license applicants, the poor—likely to be at higher risk for HIV infection—may avoid screening because of the cost.[10] The benefit of mandatory premarital HIV screening on HIV prevention is unknown.

Alternative strategies for preventing heterosexual and perinatal HIV transmission include increasing the availability of drug treatment for intravenous drug users (IVDUs) and HIV counseling and testing of clients in drug treatment centers and of all pregnant women at increased risk for HIV infection.[21,22] In addition, several states have adopted voluntary HIV screening for marriage license applicants.[23] Marriage license applicants are required to receive information about HIV infection, risk behavior, measures to avoid transmission of HIV, and availability of HIV testing and counseling. The efficacy of this approach is unknown.

We conclude that the high cost of mandatory premarital screening relative to other public health programs, its uncertain efficacy in identifying seropositive persons and preventing heterosexual or perinatal HIV infection, and the fact that persons will travel to adjacent states to avoid screening do not appear to justify its institution.

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Hepatitis Transmission among the Sioux Indians of South Dakota

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Abstract: Hepatitis A continues to occur in cyclical community-wide epidemics on the Indian reservations of South Dakota. In June 1985 a population-based serosurvey for viral hepatitis involving 120 households was conducted at the Pine Ridge and Rosebud Sioux Indian reservations in South Dakota. The serosurvey was performed shortly after a large hepatitis A epidemic on the Pine Ridge reservation in 1983–84, and immediately before a large hepatitis A epidemic on the Rosebud reservation in 1985–86. The overall seroprevalence for antibodies to hepatitis A virus (anti-HAV) was 76.2 percent (Pine Ridge reservation 80.5 percent, Rosebud reservation 72.0 percent, relative risk = 1.12, 95 percent confidence interval = 1.01, 1.24). For age groups 0 to 4 years, 54.2 percent and 36.1 percent of children were seropositive at Pine Ridge and Rosebud, respectively. Seropositivity rose rapidly with age; by age 40, more than 90 percent of persons at both Pine Ridge and Rosebud were anti-HAV positive. Only 1.1 percent of persons tested were positive for hepatitis B markers. Anti-HAV seroprevalence rates in both communities are similar to rates observed in developing countries. The surprisingly high anti-HAV seroprevalence among young children at Rosebud, where clinical hepatitis A had been virtually absent in the previous seven years, indicates that high-grade silent transmission was taking place during the interepidemic period. (*Am J Public Health* 1990; 80:1091–1094.)

Introduction

Hepatitis A is endemic among most Native American tribes in North America. Rates of hepatitis A in Native Americans in the United States are four to five times higher than national rates in recent years (Centers for Disease Control, unpublished data). Outbreaks of hepatitis A have been a problem on reservations of South Dakota for many years. In the past several decades, they have occurred regularly, usually in cyclical patterns. Typically, outbreaks appear every 7 to 10 years, each one lasting 6 to 24 months. Frequently, the outbreak cycle on one reservation does not coincide with the cycle on nearby reservations. Sometimes a large outbreak occurs at one reservation while a nearby reservation is quiescent.

Such a situation existed in 1985 at two adjacent Sioux reservations in South Dakota, Pine Ridge and Rosebud. At Pine Ridge, an outbreak of hepatitis A began in early 1983 and continued into 1984. It became quite large, eventually involving several hundred people. During the same time, the Rosebud reservation was in an interepidemic period. In late 1985, a large outbreak began at Rosebud and continued into 1986. A cross-sectional seroprevalence survey for viral hepatitis was conducted at the two reservations in June 1985, after the Pine Ridge outbreak but before the Rosebud outbreak. The results allow comparison of the hepatitis serologic profiles of a reservation in a postepidemic period with a reservation in an interepidemic period.

Methods

The Pine Ridge and Rosebud reservations lie in the rolling prairie hills of South Dakota, at the state's southern border with Nebraska. The 1985 population of Pine Ridge was 16,020; the population of Rosebud reservation was 8,473. The two reservations are divided into small villages, nine at Pine Ridge and 20 at Rosebud. The counties that make up the reservations are among the poorest in the United States as determined by per capita income. There is moderate interaction between the two communities, with occasional shared social events. Children from each reservation attend different schools. Due to the long time presence of Indian Health Service Hospitals, public health nurses, and local community health representatives, the level of hepatitis case reporting has been relatively complete at both reservations.

Epidemic Data

The data sources for hepatitis A incidence rates over two decades and for the epidemic curves of the Pine Ridge and Rosebud outbreaks were from notifiable disease reports collected by the South Dakota State Health Department. Only cases from the three largest counties in each reservation (Pine Ridge—Shannon, Jackson, Bennett counties; Rosebud—Todd, Mellette, Tripp counties) were included. Incidence rates and age-specific attack rates were calculated using 1980 US census data and intercensal population estimates.

Hepatitis Seroprevalence Survey

The geographical study area was defined as the largest villages on each reservation that together represented 75 percent of the population of each reservation: at Pine Ridge, four villages (Kyle, Oglala, Pine Ridge, and Wanblee), at Rosebud, 12 villages (Antelope, Black Pipe, Corn Creek, He Dog, Horse Creek, Parmalee, Ring Thunder, Rosebud, St. Francis, Soldier Creek, Swift Bear, and Upper Cut Meat). Using maps of each village divided into block areas, housing units were selected for study by simple random geographic sampling, 70 at each reservation. Empty or unoccupied houses were replaced with the next sequential house. Three visits to each house were made before it was classified as a refusal; refusals were not replaced. Each household selected was visited by a study team consisting of a primary investigator and a local nurse or community health representative. After obtaining consent, a questionnaire was administered to the household head asking about the age, sex, race, and clinical history of hepatitis of each household member. In addition, household characteristics were ascertained, including the number of rooms and the presence of indoor or outdoor water supply and toilet facilities. Blood specimens

From the Hepatitis Branch, Division of Viral and Rickettsial Diseases, Center for Infectious Diseases, Centers for Disease Control, Atlanta, GA (Drs. Shaw, Shapiro, Reddington, and Hadler); the Aberdeen Area Indian Health Service, Public Health Service Indian Hospital, Rapid City, SD (Dr. Welty); and the Aberdeen Area Indian Health Service, Aberdeen, SD (Dr. Dill). Address reprint requests to Craig N. Shapiro, MD, Hepatitis Branch, Division of Viral and Rickettsial Diseases, Center for Infectious Diseases, CDC, Building 6, Room 154, Atlanta, GA 30333.

were obtained from all household members one year of age or older.

Laboratory Assays

Tests for immunoglobulin M antibody to hepatitis A virus (IgM anti-HAV) and total antibody to hepatitis A virus (anti-HAV) were performed by using commercially available enzyme immunoassays; tests for hepatitis B surface antigen (HBsAg), antibody to hepatitis B core antigen (anti-HBc), and antibody to hepatitis B surface antigen (anti-HBs) were performed by using commercially available radioimmunoassays (Abbott Laboratories, North Chicago, Illinois).

Statistical Analyses

Confidence limits for relative risk estimates were calculated using the formulas of Greenland and Robins.[1]

Results

Data from Recent Outbreaks

Hepatitis A has occurred cyclically for many years on Pine Ridge and Rosebud reservations (Figure 1). The epidemic curves of hepatitis A outbreaks on Pine Ridge and Rosebud reservations in 1983–86 are shown in Figure 2. Transmission was predominantly person-to-person. There was no evidence for a common source. The age-specific attack rates for the two outbreaks show that the age distribution of patients at Pine Ridge and Rosebud were very similar and that the highest rates of disease were for younger groups aged 5 to 9 years (Table 1).

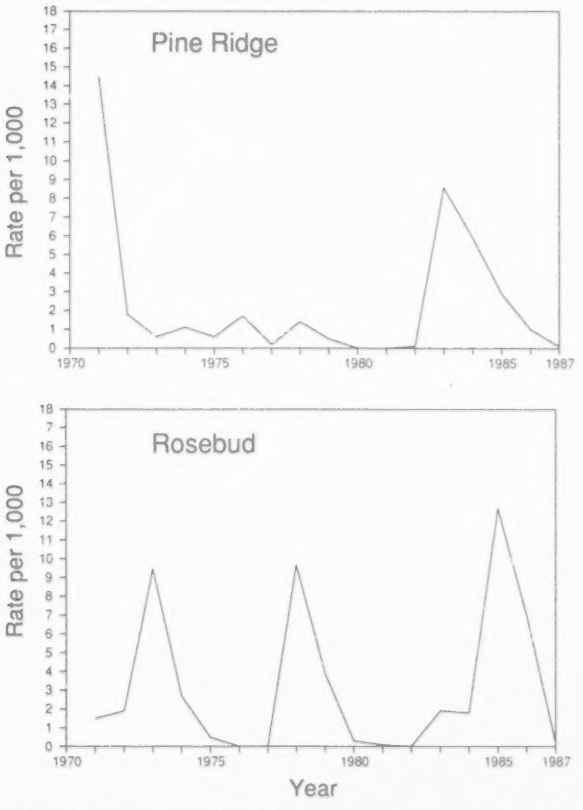

FIGURE 1—Hepatitis A Rates (per 1,000 population), Pine Ridge and Rosebud Reservations, South Dakota, 1971–87

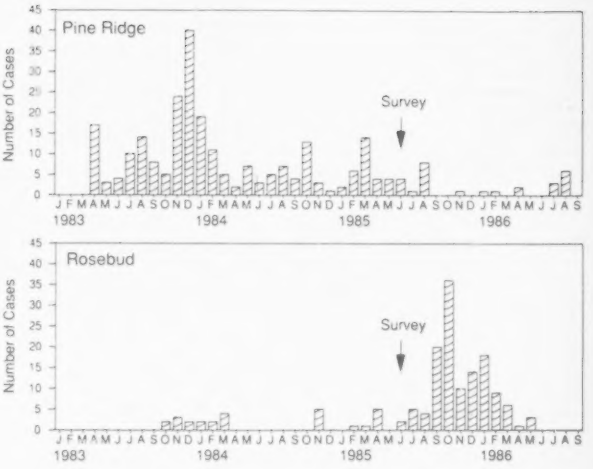

FIGURE 2—Hepatitis A Cases, by Report Date, Pine Ridge and Rosebud Reservations, South Dakota, January 1983 to September 1986

Hepatitis Seroprevalence Survey

Demographic Variables—The enrolled households numbered 120: 60 at Pine Ridge Reservation and 60 at Rosebud Reservation. A total of 605 persons lived in these households (Pine Ridge, 297; Rosebud, 308), for a mean of five persons per household (range, 1 to 16). The percentage of persons younger than 20 years of age who were in the study group did not differ substantially from the percentage in the 1980 census for the reservation areas (56.4 percent vs 53.1 percent, respectively), nor did the percentage of residents who were males (47.1 percent vs 49.7 percent, respectively).

Of the 605 persons, 590 were one year of age or older. Blood specimens from 467 persons were obtained and tested for hepatitis serologic markers. Demographic characteristics of the persons interviewed and tested are shown in Table 2. Characteristics of persons tested did not differ significantly from all household members identified.

Of the households visited, 93 percent had running water and 89 percent had flush toilets. The mean number of rooms per house was five.

TABLE 1—Age-specific Hepatitis A Attack Rates, Pine Ridge (1983–84) and Rosebud (1985–86) Reservations, South Dakota

	Pine Ridge 1983–84			Rosebud 1985–86		
Age Group (years)	Cases*	Population	AR**	Cases*	Population	AR**
0–4	64	2,064	15.5	49	1,274	19.2
5–9	87	1,972	22.1	70	1,142	30.6
10–14	34	1,895	9	13	1,041	6.2
15–19	8	1,817	2.2	11	902	6.1
20–24	8	1,294	3.1	7	744	4.7
25–29	5	1,046	2.4	5	653	3.8
30–34	3	900	1.7	2	512	2
35–49	1	1,648	0.3	5	969	2.6
50+	1	1,884	0.3	1	1,060	0.5
Total	211	14,520	7.3	163	8,297	9.8

*Native American cases reported to the South Dakota Department of Health
**Attack rate per 1000 population per year among Native Americans

TABLE 2—Demographic Characteristics and Anti-HAV Prevalence, Pine Ridge and Rosebud Reservations, South Dakota, June 1985.

Demographics	Interviewed N	(%)	Tested N	(%)	Anti-HAV Prevalence	RR (95% CI)*
Age (years)						
<20	336	(56.4)	243	(52.0)	63.4	1.42 (1.29, 1.58)
20≧	260	(43.6)	224	(48.0)	90.2	
Sex						
Male	281	(47.1)	214	(45.8)	82.2	1.04 (0.94, 1.16)
Female	316	(52.9)	253	(54.2)	79.0	
Race						
Indian	586	(98.2)	456	(97.6)	76.5	1.97 (1.36, 2.84)
White	11	(1.6)	11	(2.4)	63.6	
Born on Reservation						
Yes	461	(79.1)	351	(76.0)	82.3	1.22 (1.07, 1.40)
No	122	(20.9)	111	(24.0)	69.4	
History of Hepatitis						
Yes	32	(5.4)	28	(6.4)	100	1.37 (1.13, 1.66)
No	564	(94.6)	411	(93.6)	73.2	

*Relative risk, 95 percent confidence interval, within each category

Serologic Results—At both reservations combined, 76.2 percent (356/467) of persons tested were positive for anti-HAV. The prevalence rate at Pine Ridge was slightly higher than at Rosebud (80.5 percent vs 72.0 percent, relative risk [RR] = 1.12, 95 percent CI = 1.01, 1.24). Seropositivity rates of individual villages within each reservation were roughly comparable (Table 3). Rates in males were not significantly different from rates in females at either Pine Ridge (82.2 percent males vs 79.0 percent females, RR = 1.04, 95 percent CI = 0.92, 1.18) or Rosebud (73.8 percent males vs 70.5 percent females, RR = 1.05, 95 percent CI = 0.89, 1.23). Of the 11 Whites tested, seven (63.6 percent) were positive, compared with 349 (76.5 percent) of 456 Native Americans. Of the 356 persons positive for anti-HAV, 10 (2.8 percent) were also positive for IgM anti-HAV, indicating recent infection. Of these persons, seven were from Pine Ridge, and three were from Rosebud; the range of ages was two to 41 years (median 7.5 years).

Anti-HAV seropositivity was strongly associated with age (Figure 3). At Pine Ridge and Rosebud reservations, respectively, 54.2 percent and 36.1 percent of children aged 0–4 years were anti-HAV positive (RR = 1.50, 95 percent CI = 0.84, 2.68). Prevalence rates at both reservations rose rapidly with increasing age, although more rapidly at Pine Ridge. By age 15, over 90 percent of persons at Pine Ridge were anti-HAV positive; rates did not reach 90 percent until age 30 years at Rosebud. In young adults ages 15–29, anti-HAV seropositivity was slightly higher at Pine Ridge than at Rosebud (91.1 percent vs 71.1 percent, RR = 1.28, 95 percent CI = 1.06, 1.54). Rates between reservations were not significantly different in adults ages 30 or more years (93.8 percent vs 91.6 percent, respectively, RR = 1.02, 95 percent CI = 0.94, 1.12).

TABLE 3—Anti-HAV Prevalence by Community, Pine Ridge and Rosebud Reservations, South Dakota, June 1985

Villages	Percent Antibody Positive (N Pos/N Tested)
Pine Ridge	
Pine Ridge Village	84.7 (61/72)
Oglala	83.1 (64/77)
Kyle	72.3 (34/47)
Wanblee	77.1 (27/35)
Total	80.5 (186/231)
Rosebud	
Parmalee	75.5 (40/53)
St. Francis	83.6 (46/55)
Rosebud	67.3 (35/52)
Swift Bear	59.6 (28/47)
Antelope	72.4 (21/29)
Total	72.0 (170/236)

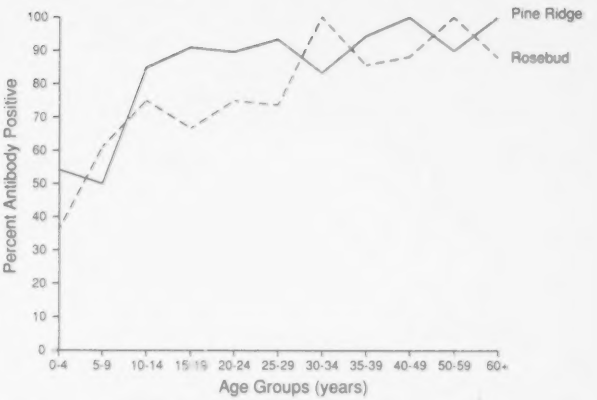

FIGURE 3—Anti-HAV Prevalence by Age Group, Pine Ridge and Rosebud Reservations, South Dakota, June 1985

Other personal characteristics which were examined included having been born on Pine Ridge or Rosebud reservation or having a history of hepatitis (Table 2). Persons born on one of the reservations were more likely to be anti-HAV-positive (82.3 percent vs 69.4 percent). Persons with a history of clinical hepatitis were also more likely to be anti-HAV-positive; this was true for persons at both Pine Ridge (RR = 1.30, 95 percent CI = 1.01, 1.68) and Rosebud (RR = 1.42, 95 percent CI = 1.07, 1.90).

Only five persons were positive for hepatitis B markers: four were immune (anti-HBc and anti-HBs positive) and one was HBsAg positive. Two persons were members of the same family—a 13-year-old boy who was HBsAg-positive, and his mother who was anti-HBs positive. The mother had acute hepatitis 13 years previously, while living off the reservation, during the third trimester of her pregnancy.

Discussion

The seroprevalence results presented here show that hepatitis A is widespread in the Pine Ridge and Rosebud reservation populations. In age groups over 30 years, 80 to 100 percent of persons have anti-HAV antibodies, indicating previous infection. These seroprevalence rates are much higher than those observed in the general U.S. population and in blood donors, in whom rates among adults are generally below 50 percent.[2] High rates of hepatitis A infection among Native Americans are due in part to poor environmental conditions and conditions related to poverty on many reservations, including crowded living quarters, inadequate housing, poor sanitation, and lack of running water.

A striking finding of this study was the high anti-HAV seroprevalence rate in young children at the Rosebud reservation (36 percent of children ages 0 to 4 years) even though Rosebud was in an interepidemic period and the last epidemic

had occurred in 1978. These rates are very much higher than observed among children in other US settings,[3,4] and are comparable to rates in children in underdeveloped countries, where hepatitis A is endemic rather than epidemic.[5,6] Similar high hepatitis A seroprevalence rates among children in interepidemic periods have been observed among the Navajo in New Mexico.[7] In contrast, other studies in settings with cyclical epidemics suggest that transmission in interepidemic periods essentially ceases.[8,9] It appears that on these Indian reservations, at least, a significant amount of disease transmission occurs even in the absence of recognized epidemics, and the transmission is primarily among young children who have the lowest clinical/subclinical disease ratio.[10] Factors that cause this interepidemic transmission to lead to epidemic transmission are still poorly defined. A possibility is that transmission occurs constantly and silently, primarily among younger age groups, who serve as a reservoir for HAV infection; epidemic transmission may supervene periodically when a cohort of susceptible persons enlarges to a trigger level. An indication of this may be the somewhat lower anti-HAV antibody prevalence observed among children and young adults at Rosebud reservation, where an outbreak was about to occur, compared with Pine Ridge, where an outbreak had just occurred.

Another striking finding is the almost total lack of hepatitis B among Sioux Indians on Pine Ridge and Rosebud reservations. The only HBsAg-positive person, a 13-year-old boy, likely acquired the infection perinatally while his mother was living off the reservation. This lack of hepatitis B is consistent with low hepatitis B prevalence found among the Navajo and Hopi in northern Arizona.[11] In contrast, hepatitis B is endemic among Native Americans and Eskimos in Alaska.[12] It may be that behaviors or other factors that promote HBV transmission among Alaskan Eskimos are absent among Native Americans of the lower 48 states; alternatively, it may be that the hepatitis B virus (HBV) has never been introduced into the latter population.

Transmission of hepatitis A on reservations in the absence of outbreaks highlights the potential limitations of immune globulin (IG) as a control measure for hepatitis A in these settings. Furthermore, when community-wide outbreaks do occur, IG may not be effective in limiting the outbreaks unless it is given to the entire community simultaneously; this is rarely done because of impractical costs and logistics. Because IG does not confer immunity longer than about three months, in populations where hepatitis A occurs in large cyclical epidemics, mass use of IG might only delay the inevitable propagation to the whole susceptible community. Such a delay might result in a greater clinical disease burden; the inevitable infections might be shifted from young children, in whom infection is usually asymptomatic, to adults, in whom clinical illness is more likely.[13]

On Indian reservations, as well as in other parts of the United States, hepatitis A has significant human and economic impact, both in terms of morbidity and occasional mortality and in diverting of public health resources from other priorities when outbreaks occur. For the immediate future, outbreaks will probably continue to occur on the South Dakota and other Indian reservations. A safe, effective hepatitis A vaccine may be the only practical solution to control hepatitis A among Native Americans, and they are therefore a likely target population both for both vaccine testing and implementation. Several prototype vaccines have been developed, and are undergoing immunogenicity trials, with efficacy trials planned in the near future.[14–17] Until such a vaccine is available, conventional measures such as the promotion of handwashing, use of IG for household contacts of acute cases, and the improvement of general sanitation will remain important priorities.

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Teaching Status and Resource Use for Patients with Acute Myocardial Infarction: A new look at the indirect costs of graduate medical education

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Abstract: To investigate whether the process of graduate medical education increases costs in teaching hospitals by causing longer lengths of stay and greater resource use, we compared lengths of stay, hospital charges, and the use of cardiovascular procedures for patients with acute myocardial infarction admitted to the teaching and nonteaching services of a university-affiliated community hospital.

After adjusting for severity of illness and demographic characteristics, patients on the teaching services had a mean length of stay that was shorter by 0.6 days ($p = 0.04$) and mean charges that were $2,060 lower ($p = 0.15$) than for patients on the nonteaching service. Patients on the teaching service also had 15 percent (95% CI: −26, −4) fewer cardiac catheterizations and 9 percent (−18, 0) fewer procedures for myocardial revascularization (angioplasty or cardiac bypass surgery).

These findings suggest that graduate medical education per se may not directly increase the use of health care resources and that the cost differences between teaching and nonteaching hospitals may be largely a consequence of other factors. These factors may include epiphenomena of teaching such as a specialized organizational structure, specialized patient care services, and continuing medical education for the nursing and medical staffs. They may also include factors not related to teaching such as differences in patients' severity of illness and sociodemographic characteristics. (*Am J Public Health* 1990; 80:1095–1100.)

Introduction

Medicare is currently providing teaching hospitals with supplementary payments to compensate them for the indirect costs of graduate medical education. However, as the government focuses on controlling costs these payments have become controversial.[1] Other third party payers also are concerned about the contribution of graduate medical education to rising health care costs.[2] There is little question that teaching hospital costs are greater than those of nonteaching hospitals. It is commonly thought that a substantial portion of this difference in costs is attributable to the educational process engendering greater resource use, both in terms of longer lengths of stay and increased use of diagnostic and therapeutic services.[2–10] Implicit in the scrutiny of the indirect costs of graduate medical education is an assumption that they may represent inefficiency or waste in health care.

Despite the prevailing belief that graduate medical education increases the use of medical services, this view is not universally accepted.[11] The findings of previous studies of this issue may be questioned for at least two reasons:

- Most of them evaluated the costs of graduate medical education for patients cared for prior to the focus on cost containment which became prominent after the introduction of prospective payment for Medicare.[2–10] The traditional academic concern to "be complete" has probably been modified by growing interest in cost-effectiveness and efficiency during the 1980s.
- Second, some investigators argue that the indirect costs of graduate medical education are merely a consequence of differences in severity of illness between patients cared for in teaching and nonteaching hospitals.[12–15] Even authors who argue that severity of illness does not account for the cost differential between teaching and nonteaching hospitals acknowledge that adjusting for severity of illness and/or case-mix substantially decreases the magnitude of the cost differential.[3–5]

The present study re-examines the issue of indirect costs of graduate medical education and focuses specifically on whether a graduate medical education program fosters greater use of medical services and longer lengths of stay by comparing the teaching and nonteaching services of a single institution.

Methods

We compared lengths of stay, total hospital charges, and the use of cardiovascular procedures for patients treated for acute myocardial infarction in 1985 on the teaching and nonteaching services of a single hospital. We chose myocardial infarction as the "tracer" because it is a common disease, clinical indicators of disease severity are easily identifiable through chart review, and there are costly, disease-specific procedures for the diagnosis and treatment of coronary artery disease.

Setting

Our data derive from Abbott Northwestern Hospital, a private, secondary and tertiary care hospital with 780 beds staffed for adult patients. The hospital is affiliated with the University of Minnesota Medical School but has its own accredited internal medicine residency training program with 12 interns or residents in each year of a three-year program. Abbott Northwestern is not a member of the Council of Teaching Hospitals, and is considered a minor teaching hospital.

Description of the Training Program

The medical service was organized with a teaching and a nonteaching service, although there was no physical separation of the two services and all patients used the same nursing stations, ancillary services, etc. Attending physicians

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admitted their patients to either the teaching or the nonteaching service, based on their own discretion, although beds on the teaching service were occasionally not available since the teaching service had a limited capacity. The attending physicians received the same reimbursement regardless of whether a patient was on the teaching or nonteaching service, so that there was no financial incentive involved in choice of service. Patients admitted to the nonteaching service were cared for solely by the private attending physicians, except in the rare case of medical emergencies, such as cardiac arrests, when residents were the only available physicians. Patients admitted to the teaching service were still the responsibility of the private attending physicians, although much of the decision-making was delegated to the teaching service ward teams. These teams were each comprised of an intern, a second- or third-year resident, a third-year medical student, and one of four internists who comprised the full-time teaching faculty. In their supervisory roles, the private attending physicians maintained routine contact with the teaching service ward teams through progress notes and informal meetings. The full-time teaching faculty conducted formal, bedside rounds after each admitting day (three hours per week per ward team), and each ward team had an additional two hours of teaching rounds per week with a separate "teaching-only" attending. These "teaching-only" faculty included internists and medical subspecialists who were not part of the full-time teaching faculty but who were part of the clinical teaching faculty of the University of Minnesota Medical School. Patient care on the teaching service was also supervised on a daily basis by three chief residents (in their 4th postgraduate year) assigned to the eight ward teams.

Facilities for Cardiac Care

Abbott Northwestern hospital had 73 critical care beds, 95 telemetry beds, a cardiac catheterization suite, and operating rooms for open heart surgery. Approximately 1,300 open heart surgeries and 900 percutaneous coronary angioplasties were performed each year at this hospital. The medical director of the critical care units was an internist who was not a cardiologist, and there were no standard orders or operating procedures that confined or limited the type of care that cardiac patients received. Patients could be cared for in the critical care units without a cardiologist attending and without a consultation from a cardiologist. Likewise, cardiac catheterization was performed under the direction of each individual cardiologist and not a catheterization laboratory director.

Patient Selection

We identified all patients discharged with a diagnosis of acute myocardial infarction (AMI) in 1985 from computerized discharge abstracts and reviewed the charts of these individuals. Of 325 patients with a discharge diagnosis of AMI we excluded 99 patients who had been transferred from another acute care facility because information concerning severity of illness could not be obtained consistently. We excluded 41 of the remaining 226 patients because chart review did not confirm a diagnosis of AMI (an elevated serum creatine kinase with a positive MB fraction, or sudden death within 24 hours of admission). Five patients with confirmed AMIs were excluded because complete information was not available for determining severity of illness. The final study cohort of 180 patients therefore included all patients directly admitted to the hospital in 1985 with a confirmed diagnosis of AMI for whom accurate severity of illness information was available. In analyses of hospital charges we included only 177 patients because charge data were not available for three patients.

Data Collection

Data were collected from chart review, computerized discharge abstracts, and the accounting office. All charts were reviewed by one of us (ISU) to collect information on age, race, sex, whether the attending physician was a cardiologist, and, if the attending was not a cardiologist, whether the patient was seen by a cardiologist in consultation. To assess the general severity of acute medical illnesses, each patient was classified by a modified APACHE II score.[16] Because age was a separate variable in our analyses we excluded points for age from the APACHE II score. We recorded the number of diagnoses listed on the discharge summary as an approximation of comorbid disease. To assess the severity of cardiac illness we noted whether each patient had a history of a previous AMI, whether there was evidence of congestive heart failure on admission, and whether during the hospitalization there was recurrent chest pain, shock, cardiac arrest, sustained ventricular dysrhythmia, or severe conduction system disturbance (2nd or 3rd degree atrioventricular block or severe bradycardia).

From the discharge abstracts and the accounting office, we obtained data on primary and secondary payor, mortality, length of stay, total hospital charges, and the use of the following cardiovascular procedures: percutaneous coronary angioplasty, coronary artery bypass surgery, cardiac catheterization, Swan-Ganz catheterization, temporary pacemaker placement, and echocardiograms. We used hospital charges as an estimate of costs. Unit charges were identical on the teaching and nonteaching services and therefore total hospital charges should be a good proxy for relative costs even if they are not equivalent to absolute costs. Because angioplasty and bypass surgery may be viewed as different methods of myocardial revascularization these two outcomes were also combined to form a composite variable. Three patients had coronary angioplasty fail and therefore had bypass surgery as well. These patients were counted as having only one episode of myocardial revascularization.

Data Analysis

Our primary goal was to compare procedure use, length of stay, and total charges between the teaching and nonteaching services. To adjust for potential confounding due to differences in severity of illness and demographic characteristics, we performed a two-stage analysis using propensity scores as described by Rosenbaum and Rubin.[17,18]

A "propensity score," defined as the probability of being on the teaching service as a function of the confounding variables, was calculated for each patient. Patients were then stratified by propensity score and outcomes were compared using the techniques for stratified data described below.

We used a logistic regression model to calculate the propensity scores as a function of 11 potential confounders: age, sex, type of insurance (health maintenance organization or other), history of previous myocardial infarction, APACHE II score, recurrent chest pain, congestive heart failure, atrioventricular block or bradycardia, shock, cardiac arrest or sustained ventricular dysrhythmia, and the number of comorbid diagnoses. There was a linear relationship between age and the probability of being on the teaching service, so age was used as a continuous variable in the model. Interaction terms added little to the predictive ability of the model and were excluded. The patients were then grouped into five strata on the basis of the probabilities

derived from the logistic model. For example, as a function of their severity of illness and other characteristics, patients in the lowest stratum had on average a 23 percent chance of being on the teaching service while patients in the highest stratum had a 78 percent chance of being on the teaching service.

Summary estimates of differences in hospital charges, lengths of stay, and procedure use between the teaching and nonteaching service were calculated using weighted averages across the five strata based on propensity scores. These summary estimates are therefore average differences that are adjusted for differences in patients' severity of illness and demographic characteristics.

Average differences in mean length of stay and charges were estimated by weighting each stratum specific difference by the inverse of the variance of the difference.[19] Average differences in median length of stay and charges were estimated by weighting each stratum specific difference in median length of stay or charges by the number of observations in that stratum, thereby producing a standardized median difference.[19] A stratified Wilcoxon rank sum test was used to test significance since length of stay and charges did not have normal distributions.[20]

Adjusted differences in procedure and mortality rates were calculated as standardized rate differences, again using the number of observations in each stratum as the weight, because several strata had marginal totals of zero.[19] Ninety-five percent confidence intervals (CI) for these standardized rate differences were based on the chi-square test statistic.[19]

To determine whether differences in resource use between the teaching and nonteaching service could be due to differences in the number of patients on each service who had a cardiologist as their attending physician, or to differences in the frequency of consultation by a cardiologist on each service, we controlled for these factors in a separate analysis. In this analysis we added dichotomous variables representing the specialty of the attending physician (cardiologist or not) and whether a patient had a cardiology consultation (yes or no) to the logistic regression model defined above, producing a second propensity score model based on 13 potential confounders. Patients were again divided into five strata on the basis of propensity scores and the above noted techniques for stratified analysis were employed to calculate weighted averages for procedure rates, length of stay and charges.

To assess the importance of outliers, we repeated our analyses excluding four patients (all on the teaching service) who had a length of stay greater than the 1985 Medicare outlier limit, which was 35 days.

The 95 percent CI for all univariate comparisons were based on the chi-square statistic[21] for categorical variables and Student's t-distribution[21] for continuous variables.

Results

Patient Characteristics

The number of patients admitted to the teaching and nonteaching services, their clinical characteristics, and the percentages enrolled in health maintenance organizations, cared for by a specialist in cardiology or seen by a cardiologist in consultation, are indicated in Table 1.

Although age and sex distributions were similar for the two groups, patients on the teaching service were generally more ill. Specifically, they had more comorbid diagnoses and more frequently had congestive heart failure on admission, recurrent chest pain, and cardiac arrest or sustained ventricular dysrhythmia.

Severity of Illness and Resource Use

The univariate associations of severity of illness with procedure use, length of stay, total charges, and mortality are presented in Table 2. Generally, the severity of illness indicators were well correlated with resource use and therefore seem to be reasonable covariates to use when controlling for severity of illness in our analyses. The patterns of procedure use, length of stay, charges and mortality conform to conventional clinical wisdom.

Resource Use and Teaching Status

Procedure rates, length of stay, hospital charges, and mortality on the teaching and nonteaching services are presented in Table 3. Patients on the teaching service had significantly fewer cardiac catheterizations and underwent myocardial revascularization less frequently. However, they had more frequent use of echocardiography. These differences persisted after adjustment for severity of illness and demographic characteristics. There was no difference in the

TABLE 1—Patient Characteristics

Characteristics	Teaching (N = 95)	Nonteaching (N = 85)	% Difference* (95% CI)
	mean ± SD		
Age (years)	73 ± 11	71 ± 14	2 (−1.7, 5.7)
APACHE II Score**	6.0 ± 4.4	5.8 ± 4.6	0.2 (−1.1, 1.5)
Number of comorbid diagnoses	7.4 ± 6.2	5.8 ± 4.5	1.6 (0.0, 3.2)
		% (N)	
Male	57 (54)	59 (50)	−2 (−16, 12)
History of myocardial infarction	28 (27)	28 (24)	0 (−13, 13)
Recurrent chest pain	51 (46)	41 (35)	10 (−5, 25)
Atrioventricular block or bradycardia	7 (7)	7 (6)	0 (−7, 7)
Cardiac arrest or sustained ventricular dysrhythima	25 (24)	16 (14)	9 (−3, 21)
Congestive heart failure on admission	75 (71)	52 (44)	23 (9, 37)
Shock	5 (5)	8 (7)	−3 (−10, 4)
Health maintenance organization members	37 (35)	40 (34)	−3 (−17, 11)
Cardiology attending	11 (10)	26 (22)	−15 (−26, −4)
Cardiology consult	55 (52)	45 (38)	10 (−5, 25)
Mortality	28 (27)	20 (17)	8 (−4, 29)

*differences are teaching minus nonteaching.
**the possible range is from 0 to 64, the actual range was from 0 to 23.

TABLE 2—Resource Use Related to Severity of Illness

Resource Use	Previous AMI		Recurrent Chest Pain		Congestive Failure		AV Block or Bradycardia		Cardiac arrest or Sustained Ventricular Dysrhythmia		Shock	
	present	absent	present	absent	present	absent	present	absent	present	absent	present	absent
Number	51	129	83	97	115	65	13	167	38	142	12	168
% Angioplasty	2	7	7	4	2	12	0	6	3	6	0	6
% Coronary bypass surgery	12	5	11	3	6	8	8	7	8	6	0	7
% Angioplasty or bypass surgery	12	10	16	6	7	17	8	11	8	11	0	11
% Cardiac catheterization	16	19	24	13	10	32	15	19	8	21	0	20
% Swan-Ganz catheterization	12	12	12	11	17	3	23	11	26	8	50	9
% Temporary pacemaker	6	11	11	8	12	5	31	8	26	5	25	8
% Echocardiogram	37	33	35	33	36	31	31	34	26	36	42	33
Mean length of stay (days)	10.1	10.5	12.5	8.6	10.7	9.8	6.9	10.6	7.4	11.2	6.2	10.7
($) Mean hospital charges	$11,038	10,065	12,943	8,143	10,942	9,277	11,673	10,234	10,807	10,221	10,658	10,317
% Mortality	35	20	27	23	34	8	62	22	79	10	92	20

use of Swan-Ganz catheterization or temporary pacemakers between the two services.

Before adjusting for severity of illness and demographic characteristics there were equivocal differences in length of stay between the two services; the teaching service had a higher mean length of stay but a lower median length of stay. After adjustment, the teaching service clearly had shorter lengths of stay. For hospital charges, patients on the teaching service tended to have higher charges before adjusting for severity and demographic characteristics, but this pattern reversed after adjustment for these factors.

The differences in length of stay and hospital charges persisted when outliers were excluded from the analysis. The adjusted mean length of stay for patients on the teaching service was 1.1 days shorter and the adjusted median length of stay was 0.5 days shorter ($p = 0.009$). Adjusted mean charges were $2,257 less and adjusted median charges were $1,653 less ($p = 0.07$).

The differences in resource use between the teaching and nonteaching services also persisted when we controlled for the specialty of the attending physician (cardiology or other) and whether a patient was seen in consultation by a cardiologist. The teaching service still had a lower use of cardiac catheterization, i.e., −11 percent (95% CI: −22, 0), and tended to have less myocardial revascularization, i.e., angioplasty −3 percent (95% CI: −8, 2); coronary bypass surgery −3 percent (95% CI: −11, 5); either −5 percent (95% CI: −13, 3). Controlling for these same variables minimized differences in lengths of stay and hospital charges. The teaching service had an adjusted mean length of stay that was 0.1 days shorter and an adjusted median length of stay that was 0.5 days shorter ($p = 0.09$). Adjusted mean charges were $16 higher on the teaching service, while adjusted median charges were $508 lower on the teaching service ($p = 0.46$).

Despite differences in resource use, in-hospital mortality on the two services was comparable. After adjusting for demographic characteristics and severity of illness, patients on the teaching services had a mortality rate that was 4 percent (95% CI: −10, 18) higher than for patients on the nonteaching service.

TABLE 3—Resource Use Related to Teaching Status

	Teaching (N = 95)	Nonteaching (N = 85)	crude difference*	adjusted** difference (95% CI)
	% (N)	% (N)	%	
Angioplasty	2 (2)	9 (8)	−7	−6 (−12, 0%)
Coronary bypass surgery	5 (5)	8 (7)	−3	4 (−11, 3)
Angioplasty or bypass surgery	6 (6)	15 (13)	−9	−9 (−18, 0)
Cardiac catheterization	11 (10)	27 (23)	−16	−15 (−26, −4)
Swan-Ganz catheterization	13 (12)	11 (9)	2	0 (0, 0)
Temporary pacemaker	9 (9)	9 (8)	0	−2 (−31, 27)
Echocardiogram	41 (39)	26 (22)	15	14 (0, 28)
Length of stay (days)				
mean ± SD	10.6 ± 7.8	10.2 ± 5.4	0.4	−0.6
median	9	10	−1.0	−1.2
p***			0.27	0.04
Hospital charges ($)				
mean ± SD	$10499 ± 9501	10164 ± 7611	335	−2060
median	$7544	7459	85	−1469
p***			0.73	0.15

*all differences are teaching minus nonteaching

**adjusted for: age, gender, insurer, previous MI, recurrent chest pain, congestive heart failure, AV block or bradycardia, cardiac arrest or sustained ventricular dysrhythmia, shock, APACHE II score, and number of comorbid diagnoses.

***p-values are for differences between teaching and nonteaching services, based on the Wilcoxon rank sum test.

Discussion

Our data do not support the view that care on teaching services necessarily entails greater use of resources than care on nonteaching services, when adjusted for severity of illness. On the contrary, these data show that once adjustments were made for differences in severity of illness, patients on a teaching service had shorter lengths of stay and received a conservative type of care that used less cardiac catheterization and myocardial revascularization.

There may be several reasons why our results differ from those of previous investigators. First, our data were collected in 1985 and almost all previous studies have analyzed data collected prior to 1981.[2-10] We believe that emphasis on efficiency and "cost effectiveness" in academic medical centers is more common now than it was during the late 1970s and early 1980s. This may be especially true for the hospital we studied, since the Twin Cities has a very competitive health care market.

A second explanation may be that patients in teaching hospitals are more severely ill and much of this difference in severity may not have been controlled for by previous investigators using methods of DRG (diagnosis related group) case-mix adjustment. We used clinically relevant indicators of severity that were specific to one illness—acute myocardial infarction. The changes in patterns for length of stay and charges that we found before and after adjustment for severity of illness support the conclusion that there is significant variation in disease severity between teaching and nonteaching patients that may not be adequately controlled for by simple case mix adjustment.

A third explanation may be that the setting of our study allowed us to better isolate the effect of a teaching program on resource use. In our single hospital setting the primary difference between the teaching and nonteaching services was the presence of the teaching program and not other institutional or organizational characteristics. Results from previous studies which compared separate teaching and nonteaching hospitals may have been confounded by systematic differences between the two types of hospitals in terms of organization and other characteristics not directly related to teaching.[3,5,8,10] Even those previous studies which compared teaching and nonteaching services within a single institution may have been confounded by organizational differences since most studies evaluated physically separate patient care units.[2,4,6,7,9]

Certain characteristics of the decision-making process on the teaching services may also have influenced the pattern of procedure use we found. Before a patient on the teaching service underwent a procedure, the housestaff, teaching attending, private attending, and the cardiologist came to a working agreement on a treatment plan. It is possible that this interaction decreased utilization in a way analogous to mandatory second opinion programs.[23,24]

Nevertheless, there are several limitations to this study. First, patients were not randomly assigned to the teaching and nonteaching services and therefore differed in a variety of characteristics. It is possible that patients admitted to the teaching service were in other ways less "appropriate" candidates for procedures than patients on the nonteaching services. We did not have sufficient data to ascertain the appropriateness of, or indications for, individual procedures, but we did attempt to collect data on and control for those patient characteristics that correlated with and likely influenced patient management. Therefore, we feel that differences in procedure use between the two services are unlikely to be related to the severity of acute cardiac disease, and are more likely to be due to differences in practice style on the teaching and nonteaching services that are independent of disease severity. However, we cannot exclude the possibility that differences in unmeasured, clinically relevant characteristics between patients on the teaching and nonteaching services accounted for our results.

Second, it is possible that variations in the practice style of a few attending physicians accounted for our results. However, 71 attending physicians had patients in our study and the mean number of patients per attending physician was 2.5 (range 1–11).

A third potential problem with our findings is that the records on the teaching and nonteaching services may have contained systematically different amounts and detail of information regarding severity of illness. The physician-reviewer who abstracted all the charts did not note such a discrepancy, especially for the parameters of severity and utilization that we collected. Therefore we do not feel that our findings are an artifact of chart completeness.

Finally, we decided to study only one hospital and one disease in an effort to control for as many confounders as possible. This limits our ability to generalize our results to other settings and other diseases. An analysis of the management of other illness which have less stereotyped patterns of care or which have fewer options for very specialized procedures may show different results. While it is also possible that the teaching service we studied may be atypical and not representative of teaching services at other types of institutions, we feel that the nature of the internal medicine training program we studied was very similar to that of most university-based programs. It is also likely to be representative of those found in other nonCOTH (Council of Teaching Hospitals) minor teaching hospitals which in total comprise approximately 72 percent of all teaching hospitals in the country and have 19 percent of all residents.[25] These nonCOTH minor teaching hospitals would therefore receive approximately 20 percent of Medicare's payments for indirect medical education.[26]

Overall, this study suggests that graduate medical education does not universally lead to longer lengths of stay and greater use of medical resources. It may therefore be unwarranted to equate the cost differences between teaching and nonteaching hospitals with nonessential clinical expenditures generated by graduate medical education. This does not imply that teaching hospitals have the same costs as nonteaching hospitals. Graduate medical education may lead to "indirect" costs that are associated with teaching such as floor space devoted to conference rooms, larger medical libraries, and support for teaching faculty. The presence of graduate medical education may encourage teaching hospitals to have more active programs for nursing education and continuing education for staff physicians. Teaching hospitals may also require a more sophisticated organizational structure and higher level of technical support because of the academic mission which leads to caring for patients who require extraordinarily specialized and highly technical care. Previous studies have shown that hospitals which accept a large number of transfer patients have higher average costs per case than hospitals receiving few transfer patients.[27] Teaching hospitals may constitute a large percentage of such hospitals because of specialized services that attract referrals. Other reasons for higher costs in teaching hospitals may not be directly related to graduate medical education at all.

For example, there may be important differences in the socioeconomic characteristics of patients in teaching hospitals that increase resource use and bad debt.[28]

Medicare's supplementary payment to teaching hospitals to cover the indirect costs of graduate medical education is already controversial and has been reduced by 30 percent since 1983. Further reduction is now under serious consideration. Other payers are also questioning the high costs of care in teaching hospitals. The results of this study suggest that these costs may not be a function of unnecessarily longer lengths of stay and greater use of health care resources. We postulate that much of the higher costs noted in teaching hospitals may now come from the overhead expense of providing and developing specialized services or from differences in the patient populations of teaching and nonteaching hospitals that are not accounted for in DRG case-mix adjustments. In order to institute proper reform of reimbursement for graduate medical education, further work is needed to define more accurately the components of graduate medical education that currently influence health care costs.

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Call for Presentations at 1991 Annual Conference of AHA Section for Psychiatric and Substance Abuse Services

The American Hospital Association Section for Psychiatric and Substance Abuse Services invites proposals for presentations at their annual conference, "Changes and Challenges, Management of Psychiatric and Substance Abuse Services." The conference, which will be held Jun 5–7, 1991 in Boston, attracts hundreds of multidisciplinary mental health professionals from hospital-based psychiatric and substance abuse programs.

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The Effects of Workplace Health Promotion on Absenteeism and Employment Costs in a Large Industrial Population

ROBERT L. BERTERA, DRPH

Abstract: We evaluated the impact of a comprehensive workplace health promotion program on absences among full-time employees in a large, multi-location, diversified industrial company. A pretest-posttest control group design was used to study 41 intervention sites and 19 control sites with 29,315 and 14,573 hourly employees, respectively. Blue-collar employees at intervention sites experienced an 14.0 percent decline in disability days over two years versus a 5.8 percent decline at control sites. This resulted in a net difference of 11,726 fewer disability days over two years at program sites compared with non-program sites. Savings due to lower disability costs at intervention sites offset program costs in the first year, and provided a return of $2.05 for every dollar invested in the program by the end of the second year. These results suggest that comprehensive workplace health promotion programs can reduce disability days among blue collar employees and provide a good return on investment. (*Am J Public Health* 1990; 80:1101–1105.)

Introduction

Health education has significant potential for reducing risk factors.[1–13] The effectiveness of workplace health promotion programs in reducing health risks has been demonstrated in the areas of high blood pressure control[14–16] and smoking cessation.[17–19] The evidence is more tentative in other areas of lifestyle change.[20–35]

Health education in the workplace is growing rapidly, driven by the promise of providing several non-economic,[33,36–38] as well as economic benefits.[18,38–44] Some authors emphasize the need for cost benefit and cost effectiveness analyses in order to examine the cost-effectiveness of workplace health promotion.[18,37,40,41,45] Others point out the limitations to the existing literature.[38,39,41]

The purpose of this study was to evaluate the impact of a comprehensive workplace health promotion program on illness absences not related to occupational causes.

Methods

Program Description

The program includes five core elements: training for site coordinators; a Health Promotion Activity Committee (HealthPAC); Orientation and Publicity; Health Risk Appraisal; and a variety of self-directed and group health education opportunities. These were first pilot tested in 1981 and 1982 at two manufacturing locations, refined, and then offered at the 41 intervention sites beginning in late 1983 and early 1984.

The program is introduced and administered by a network of site and departmental coordinators. Approximately 60 percent of these individuals are non-medical personnel selected for their communication skills, interest in health enhancement, and organizational abilities; in most cases, they serve part time in conjunction with their regular jobs. The remaining 40 percent are site medical personnel including nurses, physicians, and physician assistants.

At each location, a Health Promotion Activity Committee (HealthPAC) consisting of five to 15 employees from all levels of the organization assists in planning, publicity, and implementation.

The corporate health promotion staff offers a variety of three-day workshops designed to provide program guidelines, materials, and support for coordinators and HealthPACs. A quarterly newsletter keeps coordinators, committee members and management informed about site activities, new programs, and progress toward program objectives.

All program participants complete a voluntary, computer-scored health risk survey (available on request to author) containing 36 items on health status, personal habits, and lifestyle. Assistance is provided for interpreting appraisal results in groups using a videotape explanation, and individually through consultation with site medical personnel for all employees who request it. A copy of the appraisal is filed with the medical record for review during periodic medical evaluations which are offered to all employees at one, two, or three year intervals, based on age.

Health promotion activities include a variety of group and self-directed opportunities. Four- to 10-week class cycles provide opportunities at lunchtime or after shift to raise awareness or learn new skills. Topics for group and self-directed programs include smoking cessation, fitness, weight control, lipid control, stress management, and healthy back.

All employees receive a bi-monthly health and fitness magazine that helps keep employees and family members informed about current health and wellness developments. Challenges and incentive programs attempt to involve large numbers of employees in fitness, weight control, and smoking cessation activities. Cafeterias and vending machines offer heart healthy foods. Employees can check and record their own blood pressure and weight at machines and scales located in high traffic locations.

Certain program components were offered on company time including: program orientation activities, Health Risk Appraisal meetings, individual counseling, and health promotion activities presented to employees during safety meetings. The time allowed for these activities varied from 30 minutes to three hours, depending on employee interest and site commitment to employee health improvement. All employees must attend regular safety meetings on company time. Some of these meetings introduce and reinforce topics important to employee wellness such as: stress management, dental health, weight control, fitness, healthy back, high blood pressure control, nutrition guidelines, and smoking cessation. Safety meetings usually occur at least monthly and last from 15 to 60 minutes.

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Program Costs and Benefits

Actual program costs, disability wage costs, and disability wage savings were analyzed for the baseline year and two program years. Health promotion program costs include all expenses in 1986 dollars for: staff and committee time; instructors and group leaders; educational materials; publicity and promotion; health risk appraisals; and recognition awards and incentives. Program costs did not include: office space and utilities; fitness equipment (only a few sites had any at the time of the study); and the value of employee time when attending activities during working hours.

Disability wage costs included actual wages and benefits paid to hourly employees in 1986 dollars for days of absence due to illness. Savings were calculated as the difference between disability wages paid at the end of the first and second program years subtracted from disability wages paid in the baseline year, prior to the program's introduction. Disability wage costs and savings exclude other costs that may be related to absenteeism such as health care claims and replacement worker expenses.

Study Population and Design

The study population consisted of hourly employees who were part of the US workforce in a large, diversified manufacturing company. White collar employees were excluded from the study because the major outcome measure—disability days due to non-occupational illness—was recorded only for hourly employees. The hourly workforce had the following characteristics: more than three-fourths (82.7 percent) male; 16.1 percent Black; and more than one-third (41.0 percent) were 40 or more years of age.

We used a pretest-posttest equivalent control group design.[46] Out of 100 locations that were providing comprehensive health promotion services, 60 were included in this study because they had at least 100 hourly employees on site. This excluded smaller sites from the study such as unit plants, warehouses, and transfer depots, as well as predominantly white collar locations such as sales offices, service units, and research centers. All study sites followed consistent policies on disability leave and reporting throughout the study. Although site-specific data on turnover rates were not available, the company has a long tradition of very low employee turnover.

The intervention sites consisted of 41 locations which elected to initiate the health promotion program by June 1985 and met the above inclusion criteria. All sites had to obtain prior approval to initiate the health promotion program by submitting a written implementation plan to the corporate health promotion manager. This review process made it possible to identify the study's control sites. The control sites consisted of 19 locations that had not adopted the program by June of 1985.

The two study groups were compared for equivalence on a number of demographic and social characteristics at baseline. The 41 program sites did not differ significantly from the 19 non-program sites in terms of the average number per site of: employees; males; employees 40 years of age or older; or the total number of hourly or salaried employees (Table 1).

The analysis is based on absenteeism data from 43,888 hourly employees from January 1984 through December 1986. The 41 program sites employed 29,315 hourly employees while the 19 non-program sites employed 14, 573 hourly employees.

TABLE 1—Demographic Profile Of Hourly Employees at Health Promotion Program Sites vs Non-program Sites

Mean Number per Site[a]	Study Groups[b] Program Sites (N = 41)	Non-Program Sites (N = 19)
Total Hourly Employees	715	767
Males	582	625
Age 40 or Older	325	324
Total Number of Hourly Employees	29,315	14,573
Total Number of Employees (Hourly and Salary)	42,435	22,116

[a]Based on 1984 averages per site for hourly employees except where noted as totals for the study group.
[b]No significant differences by t tests, 2 tail, separate variance estimate.

Unit of Analysis and Measurement of Outcomes

The primary outcome variable, using the sites as units of analysis, was the annual mean number of disability days per hourly employee. Days lost due to disability is defined as a measure of absenteeism that includes all illnesses not related to occupational causes. Disability days are recorded on time cards signed by supervisors and submitted to site time keepers for payroll purposes. Site disability days are reported quarterly to the corporate Compensation and Benefits Division which aggregates site data into annual reports.

Another outcome variable was the cost of wages, compensation, and benefits paid in 1986 dollars for days absent due to illness. For several years before this study began, all 60 study locations reported actual annual costs of wages, compensation, and benefits per hourly employee as part of an ongoing corporate accounting system separate from the health promotion program.

The health promotion program costs were obtained from two sources: 1) many items such as educational materials, supplies, health risk appraisals, and incentive awards were obtained from the corporate program, which provided expense records when sites were charged back for these goods and services; 2) sites were asked to submit annual budgets including information on staff and committee time. These sources were used to calculate the average annual program expenditures per hourly employee at each of the 41 intervention sites. The urban Consumer Price Index was used to convert health promotion program costs and disability wage costs into 1986 dollars and thus control for the effects of inflation from 1984 through 1986.

Results

The health promotion program began at the 41 intervention sites between November 1983 and June 1985. Most sites began the program in the first quarter of 1984; however, due to start-up delays, the program was actually available to employees an average of 20 months per site during the two-year study period.

Disability Days

As shown in Table 2, employees at program sites experienced a somewhat higher average number of days lost due to disability in the baseline year (1984). In the first year after the program was introduced (1985), the average number of disability days dropped 10.5 percent at program sites while it increased 1.9 percent at non-program sites.

TABLE 2—Mean Disability Days Lost by Hourly Employees by Program Year for Program and Non-program Sites

Year	Disability Days Lost by Study Group	
	Program Sites (N = 41)	Non-Program Sites (N = 19)
1984 (Baseline Pre-Program)	5.7	5.2
1985 (Year 1)	5.1	5.3
1986 (Year 2)	4.9	4.9
1984–1986 (decline)*	0.7	0.3

*Differences is 0.4 disability days (95 percent confidence interval 0.3, 0.5).

TABLE 3—Mean Disability Days by Hourly Employees Baseline Year (1984) vs Program Years One and Two for Sites with High vs Low Participation

Year	Disability Days Lost by Site Participation Level	
	High (N = 20)	Low (N = 21)
1984 (Baseline Pre-Program)	5.7	5.5
1985 (Year 1)	4.9	5.2
1986 (Year 2)	5.0	4.9
1984–1986 (decline)*	0.7	0.6

*Difference is 0.1 disability days (95 percent confidence interval 0.03, 0.23).

By the end of the second year, disability days dropped 14.0 percent at program sites and 5.8 percent at non-program sites. This represents 0.7 fewer disability days at program sites and 0.3 fewer days at non-program sites. The difference is 0.4 disability days per hourly employee (95 percent confidence interval 0.3, 0.5 days), which resulted in a savings of 11,726 fewer disability days at program sites compared with non-program sites over two years. This savings is the equivalent of 49 person years of effort that can be spent to produce goods and services.

The data suggest that the health promotion program had some influence on lowering disability days among hourly employees. It should be noted that other potential influences on disability statistics, such as early retirement programs and changes in medical insurance plans, were implemented simultaneously at both program and non-program locations during this period, thus providing some control for these external influences.

Disability Days by Participation Level

Although all 41 program sites met company health promotion guidelines (see Program Description, above), sites probably varied in the resources that they allocated to the program. Due to the number of sites, only limited anecdotal information was obtained in the course of the study to suggest resource variation as a confounding factor; one indirect indicator of how employees perceived the program was participation rates. Participation was measured by the number of employees who completed the Health Risk Survey form. Employees who completed the 36-item risk survey received a computer-scored Health Risk Appraisal within about 30 days.

Program sites where 50 percent or more of employees completed the Health Risk Survey had an average survey completion rate of 70.6 percent (CI = 69, 72), and are referred to as "High Participation" sites (N = 20). Program sites where less than 50 percent of employees completed the Health Risk Survey averaged only a 30 percent completion rate (CI = 26, 34), and are classified as "Low Participation" sites (N = 21).

One indicator that demonstrates differences in the intensity of program activity at high versus low participation sites was the variety of on-site classes offered. Sites that obtained a "high" response to the Health Risk Appraisal offered 6.2 topics, compared with 4.2 topics at low participation sites (difference 2.0, 95 percent CI = 1.56, 2.44).

As shown in Table 3, mean disability days declined in the first year by 14.0 percent at high participation sites and by 5.4 percent at low participation sites. By the end of the second year, disability days had declined 12.3 percent at program sites and 10.9 percent at non-program sites. This reflects a mean difference of 0.1 disability days (95 percent CI = 0.03, 0.23 days) between the two participation levels.

These data suggest that while higher participation was associated with greater declines in disability days in the first year, the association was not maintained in the second year. Table 4 indicates that there were about 50 percent more hourly employees at low participation than at high participation sites. It is possible that participation level was influenced by site size in a way that enabled smaller sites to gain higher participation and lower disability in the first year due to simplified start up logistics. By the second year, however, larger sites could have offered the program to more of their employees, thus reducing the first year differences that appeared to favor high participation sites. Another fact that supports this view is that indicators of participation were heavily dependent on first program year activity, again making it more likely that smaller sites would be classified as high participation sites.

Health promotion program costs were higher than in the first program year ($1.4 million) than the second year ($.7 million). This reflects higher initial investments of staff time, coordinator and committee time, training, publicity, and educational materials and incentives, some of which were not repeated in the second year. Savings due to lower disability costs were enough to offset program costs in the first year, where every dollar invested in health promotion yielded $1.11 in lower disability costs; in the second program year, every $1.00 spent on health promotion yielded disability savings of $2.05 (Table 5). Total return on investment over two years averaged $1.42 in lower disability wage costs for every $1.00 invested in health promotion.

TABLE 4—Demographic Indicators for Hourly Employees at Program Sites with Low vs High Participation

Mean Number per Site[a]	Site Participation Level[b]	
	High (N = 20)	Low (N = 19)
Total Hourly Employees	568	905
Males	446	759
Age 40 or Older	242	439
Total Number of Hourly Employees	11,927	18,110
Total Number of Employees (Hourly and Salary)	19,257	25,252

[a]Based on 1984 averages per site for hourly employees except where noted as totals for the study group.
[b]No significant differences by t tests, 2 tail, separate variance estimate.

TABLE 5—Program Costs and Disability Savings[a] by Program Participation Level and Year

Program Analysis	All Program Sites (N = 41 Sites)	Site Participation Level: Low (N = 21 Sites)	Site Participation Level: High (N = 20 Sites)
Year 1 (1985)			
Costs	$1,436,965	$ 736,031	$ 700,933
Savings	1,596,877	708,069	888,808
Return on Investment[b]	$1.11	$ .96	$1.27
Year 2 (1986)			
Costs	$ 714,312	$ 366,075	$ 348,237
Savings	1,463,524	641,632	821,891
Return on Investment[b]	$2.05	$1.75	$2.36
Combined (1985 & 1986)			
Costs	$2,151,277	$1,102,107	$1,049,170
Savings	3,060,401	1,349,701	1,710,699
Return on Investment[b]	$1.42	$1.22	$1.63

[a]All Costs and Savings figures are in 1986 dollars.
[b]Based on disability savings divided by program costs.

Total return on investment over two years varied by program participation level, with high participation sites saving $1.63 in lower disability costs for every dollar invested over two years, versus $1.22 for low participation sites (Table 5).

These return-on-investment data are conservative estimates because they exclude other potential savings that may be associated with lower absenteeism including lower costs for: company health care claims; replacement workers; and employee out-of-pocket health care expenses.

Discussion

Intervention sites self-selected themselves by initiating comprehensive employee health promotion activities prior to June 1985. This decision may have been linked to factors that could have predisposed self-selected sites to reduce disability days through means other than health promotion.

If present, this bias could lead to over-estimating the potential impact of the program. Future studies could control this potential bias by randomly assigning sites to intervention and control conditions.

The control sites consisted of 19 locations that had not adopted the comprehensive program by June 1985. However, a few of the 19 control locations may have initiated portions of the program after June 1985 and thus received partial interventions from July 1985 through December 1986. This potentially dilutes the intervention effect which may have been reflected in the second-year absenteeism declines for control sites, and probably produces an underestimate of the program's potential impact. No systematic information was collected on the nature or extent of potential control site contamination. The extensive communication networks and decentralized decision-making in a large, diversified company makes it difficult to measure and control such effects.

Disability days were tracked for three years including the baseline year. Longer follow-up periods would be desirable to measure maintenance effects. Unfortunately, the tracking system for disability days was discarded in 1987 as part of a restructuring of human services resources within the company.

Site employee participation levels were estimated by the number who completed the health risk appraisal. This is an indirect but convenient measure of perceived interest and quality. While some evidence exists that the number of activities offered on the site is related to the risk appraisal completion, future studies should seek additional information on program participation, activities, and expenditures. The measurement and attribution of potential program impacts could also be improved by obtaining information on geographic or intergroup variations of employee turnover, job satisfaction, and other measures of attitudes or beliefs that could affect absenteeism patterns.

Recent published reports, while not using absenteeism as an outcome, have reported savings associated with comprehensive health promotion programs in the workplace. In a study of Johnson & Johnson's comprehensive approach, inpatient hospital insurance costs compared to controls[47] were nearly $1 million lower over five years. These savings were attributed to lower rates of increase in the number of hospital days and in the number of admissions among employees at program sites.

In a study of a comprehensive health promotion and wellness education at Blue Cross-Blue Shield subscriber companies, employees with the program claimed 24 percent fewer health insurance dollars over five years compared to similar employees who were not receiving the program.[48] The savings in lower health care insurance costs produced a return on investments of $1.45 for each dollar spent on the program.

These studies and our results suggest that comprehensive workplace health promotion programs can return $1.45 in lower hospital insurance costs and another $1.42 in lower disability wage costs for every dollar invested in health promotion. If these findings can be confirmed in other studies, it appears that health promotion programs are good business because they provide a favorable return on investment while helping to improve indicators of health among blue collar employees.

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USEPA to Sponsor International Symposium on Radon

The United States Environmental Protection Agency has announced the dates for the International Symposium on Radon and Radon Reduction Technology will be April 2–5, 1991, in Philadelphia, Pennsylvania, and has issued a call for papers with the theme being 'A New Decade of Progress.'

The symposium will provide a forum for the exchange of technical information on radon and radon reduction technology in the indoor environment. The major topics considered for the symposium will be: experience in the development and application of radon reduction and radon-resistant construction techniques, the measurement of radon and radon progeny, and the assessment of radon-derived health impacts.

Topics of greatest interest are: Radon Control, Measurement Methods (air and soil), Health Issues, Foreign and Domestic Radon Programs and Policy Issues, Radon Surveys, Public Information and Education, Radon in the Natural Environment.

Abstracts of 150 words or less should be submitted by September 30 to: Timothy M. Dyess, US-EPA, AEERL/Radon Mitigation Branch, MD-54, Research Triangle Park, NC 27711. Papers should provide study results; however, theoretical discussions of concepts and mechanisms will also be considered. Abstracts will be screened by the sponsors and papers will be solicited to cover areas of special concern. Notification of acceptance of papers will be done by November 15, 1990, after which the author will be required to submit an original and five copies of the paper by January 31, 1991.

Caffeine-Containing Beverages, Total Fluid Consumption, and Premenstrual Syndrome

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Abstract: The main objective of this study was to evaluate whether daily consumption of caffeine-containing beverages is related to the prevalence and severity of premenstrual syndrome apart from any effects of daily total fluid consumption. A secondary objective was to determine whether daily total fluid consumption itself is related to premenstrual syndrome. The study is based on 841 responses to a questionnaire probing menstrual and premenstrual health, and daily fluid consumption, which was mailed to female university students in Oregon. Analysis of the data revealed that consumption of caffeine-containing beverages was strongly related to the prevalence of premenstrual syndrome. Among women with more severe symptoms, the relation between consumption of caffeine-containing beverages and premenstrual syndrome was dose-dependent, with prevalence odds ratios equal to 1.3 for consumers of one cup of a caffeine-containing beverage per day and increasing steadily to 7.0 for consumers of eight to 10 cups per day. The effects were apparent among both caffeine-containing tea/coffee consumers and caffeine-containing soda consumers. The observed effects were only slightly reduced when daily total fluid consumption was controlled. Daily total fluid consumption also was related to the prevalence of premenstrual symptoms although the effects were large only for consumers of 13–19 cups of fluid per day (the largest amount studied). (*Am J Public Health* 1990; 80:1106–1110.)

Introduction

Previous investigations have indicated that the consumption of caffeine-containing beverages is strongly related to the prevalence and severity of premenstrual syndrome (PMS), and that such effects exist for a wide variety of premenstrual symptoms.[1–4] Further work demonstrated that the association between premenstrual symptoms and consumption of caffeine-containing beverages occurs even among women who lack other putative causes of the syndrome.[5] Such putative causes included alcohol use, cigarette use, lack of exercise, oral contraceptive use, and a diet high in beef and refined sugar products.[6–18]

One possible explanation for the observed association between consumption of caffeine-containing beverages and premenstrual symptoms is that consumption of these beverages is related to daily total fluid consumption, and that it is daily total fluid consumption which is causally related to premenstrual symptoms. Such an explanation might be consistent with the retention of fluid experienced by many women who report premenstrual symptoms.

This study was undertaken to assess the relation between premenstrual syndrome and caffeine-containing beverage intake, and between premenstrual syndrome and total fluid intake, controlling for any confounding effects attributable to the other variable. In addition, we wanted to describe the dose-response relation between premenstrual syndrome and consumption of caffeine-containing beverages and of total fluid, and to discover which types of caffeine-containing beverages might exert an effect.

Methods

Study Population and Data Collection

The study was conducted at a public university in Oregon. Eligible subjects were the 1,419 currently enrolled, female students who resided in any of the university's residential dormitories.

In October 1988, each eligible student was sent a questionnaire, together with a cover letter explaining the general nature of the study (a study of women's health) and requesting her participation. The questionnaire solicited information about the presence and severity of premenstrual symptoms and of menstrual symptoms, demographic characteristics, use of over-the-counter drugs containing caffeine, and typical daily consumption of beverages including caffeine-containing beverages. The questions pertaining to premenstrual symptoms asked each respondent to indicate whether she experienced premenstrual symptoms and, if she did, to score the severity (mild, moderate, or severe) of each in a list of 10 premenstrual symptoms plus an "other symptom" category. To reduce the likelihood that the study participants would suspect the hypotheses under investigation, we included questions about menstrual health similar in number and detail to those probing premenstrual health. A definition of the syndrome, which emphasized the timing of symptoms with respect to the onset of menses, was included in the questionnaire.

The questions pertaining to daily beverage intake asked for the number of cups, defined on the questionnaire as "eight ounces, or one measuring cupful," of each in a list of 11 beverages plus an "other beverage" category the respondent consumed during a typical day. One of the 11 beverages was water.

Eight-hundred-and-sixty-nine women (61 percent) returned the questionnaire after two mailings. (The second mailing was conducted in January 1989.) The data for 28 women subsequently were excluded from the data analysis: two women reported having endometriosis, four women did not menstruate on a regular basis, and 10 women did not provide information on the presence of premenstrual symptoms or on the daily consumption of beverages. An additional 12 respondents were excluded because their daily consumption of caffeine-containing beverages was between 11 and 30 cups, making the cup-specific prevalence odds estimates unstable (as described in the section on data analysis). These exclusions left data on 841 women for analysis. Ninety-six per cent of these women were between the ages of 18 and 22 years.

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Data Analysis

We estimated the premenstrual syndrome prevalence odds ratios, the odds of having premenstrual syndrome among "exposed" women divided by the odds among "unexposed" women, for five levels of daily consumption of caffeine-containing beverages (one cup, two to four cups, five cups, six or seven cups, and eight to 10 cups) and for four levels of daily total fluid intake (six or seven cups, eight to 10 cups, 11 to 12 cups, and 13 to 19 cups). These intervals were determined empirically on the basis of the uniformity of the premenstrual syndrome prevalence odds ratio point estimates for each level of consumption within an interval. For the three subjects who used caffeine-containing drugs on a daily basis, the estimates of the number of cups of a caffeine-containing beverage consumed each day were increased. The amount of increase equaled the equivalent number of cups of a caffeine-containing beverage for the caffeine content of the drug. The equivalency was determined by the information provided on each drug's packaging material. There were too few subjects who reported consuming more than 10 cups of a caffeine-containing beverage each day, or more than 19 cups of total fluid each day, to provide information for higher levels of consumption. Daily total fluid consumption was calculated by summing the numbers of cups of beverages consumed each day for each of the 11 listed beverages plus the "other" beverage category.

Confidence intervals for the prevalence odds ratios were based on Miettinen's test-based procedure, using the programs of Rothman and Boice.[19]

The overall trend in the data relating premenstrual symptoms and caffeine-containing beverages intake, and premenstrual symptoms and total fluid intake, was tested using the method described by Mantel.[20] The method of maximum likelihood was used to estimate the slope, and standard error for the slope, for the logistic curve relating premenstrual syndrome and daily consumption of caffeine-containing beverages or daily total fluid intake.[21]

We assessed the magnitude of confounding of the premenstrual syndrome prevalence odds ratio by either daily consumption of caffeine-containing beverages or by daily total fluid consumption (depending upon which of these two variables was the variable of interest) by stratifying the data into categories of the potential confounding factor and obtaining an estimate for a uniform prevalence odds ratio by the method of maximum likelihood.[22] The categories used were identical to the categories formed by empirically determining the intervals over which the premenstrual syndrome prevalence odds ratio was uniform.

The level of severity of premenstrual syndrome was determined by each participant in response to a question asking her to assess the overall severity of her symptoms. In addition, a "PMS score" was calculated for each woman. This score was equal to the sum of each respondent's rating of severity for each in the list of 10 symptoms plus the "other" symptoms category, as contained in the questionnaire. A score of one was assigned to each symptom rated mild, a score of two was assigned to each moderate symptom, and a score of three was assigned to each severe symptom. The range of possible PMS scores, therefore, was 0 (no premenstrual symptoms) to 33 (11 severe symptoms).

Results

Prevalence of Premenstrual Symptoms and Types of Beverages Consumed

Six-hundred-and-sixteen (73 percent) of the 841 respondents reported experiencing at least one premenstrual symptom, and 603 (72 percent) reported consuming at least one cup of a caffeine-containing beverage each day. For all types of beverages consumed, a total of 7,048 cups of fluid were reported to be consumed each day: 39 percent water, 20 percent caffeine-containing soda, 12 percent fruit juice, 11 percent milk, 6 percent caffeine-containing coffee, 6 percent caffeine-free soda, 3 percent hot chocolate, 2 percent each caffeine-containing tea and herbal tea, and less than 1 percent each decaffeinated coffee and grain beverages: other beverages accounted for less than 1 percent of the total cups consumed. Seventy-one percent of the total volume of caffeine-containing beverage consumed was soda, 21 percent was coffee, and 8 percent was tea.

Consumption of Caffeine-containing Beverages

The overall prevalence odds ratio for daily consumption of one to 10 caffeine-containing beverages among women who reported experiencing at least one premenstrual symptom compared with women who reported no premenstrual symptoms was 1.5 (90% confidence interval = 1.1, 2.0). This effect was restricted primarily to women whose PMS scores equaled 16 through 31 (the highest score observed).

For the 399 women with PMS scores between 1 and 15 (median score = 9), the prevalence odds ratio for daily consumption of any caffeine-containing beverages (one to ten cups per day) was 1.3 (90% CI = 0.9, 1.7). This point estimate remained unchanged when controlled for daily total fluid consumption. The estimates for one to five cups per day, and for six to 10 cups per day, were similar (1.2 and 1.1, respectively) when daily total fluid consumption was controlled (Table 1).

Among women with PMS scores from 1 to 15, 61 percent rated the overall severity of their PMS symptoms "mild" while only 2 percent rated their symptoms "severe."

Table 2 shows the prevalence odds ratios of premenstrual syndrome for consumption of caffeine-containing beverages among the 217 women whose PMS scores equaled 16 through 31 (median score = 19). For these women, the overall prevalence odds ratio (one to 10 cups of caffeine-containing beverages per day relative to no cups) was 2.0 (90% CI = 1.4, 2.9). A steadily increasing prevalence odds ratio was observed for increasing amounts of daily consumption of caffeine-containing beverages, ranging from 1.3 for one cup per day to 7.0 for eight to 10 cups per day. All of the estimated prevalence odds ratios, except the estimate for one cup per day, were slightly reduced when daily total fluid consumption was controlled (Table 2).

The p-value of the test for trend between premenstrual syndrome and daily cups of caffeine-containing beverages

TABLE 1—Prevalence Odds Ratios of Premenstrual Syndrome According to Daily Consumption of Caffeine-containing Beverage Consumption among the 399 Women Whose PMS Scores Equaled 1 through 15

	Daily Consumption of Caffeine-containing Beverages (Cups)		
Control for Total Fluid	0 POR*	1–5 POR*	6–10 POR*
No	1.0	1.2 (0.9, 1.7)	1.6 (0.8, 3.1)
Yes	1.0	1.2 (0.9, 1.7)	1.1 (0.6, 2.4)
Number of Exposed Women**	194	396	34

*Prevalence Odds Ratio, Relative to 0 Cups per Day (90% Confidence Interval).
**Includes Cases and Controls.

TABLE 2—Prevalence Odds Ratios of Premenstrual Syndrome According to Daily Consumption of Caffeine-containing Beverages with and without Controlling for Daily Consumption of Total Fluid Intake among the 217 Women Whose PMS Scores Equaled 16 through 31

Control for Total Fluid	Daily Consumption of Caffeine-containing Beverages (Cups)					
	0 POR*	1 POR*	2–4 POR*	5 POR*	6–7 POR*	8–10 POR*
No	1.0	1.3	2.0	2.5	5.0	7.0
Yes	1.0	1.3	1.8	2.1	3.3	6.5
Number of Exposed Women**	121	85	172	22	27	15

*Prevalence Odds Ratio, Relative to 0 Cups per Day.
**Includes Cases and Controls.

consumed was <0.001. The slope of the logistic curve relating premenstrual syndrome and daily consumption of caffeine-containing beverages (without control for total fluid intake) was 0.22 (standard error = 0.047). The slope of the logistic curve relating premenstrual syndrome and caffeine-containing beverage consumption, controlling for daily total fluid intake, was reduced to 0.18 (standard error = 0.046); thus, each additional daily cup of caffeine-containing beverage was associated with a 20 percent increase in the prevalence of premenstrual syndrome ($e \cdot 18 = 1.2$).

Among women with PMS scores from 16 to 31, 12 percent rated the overall severity of her symptoms "mild" while 18 percent rated her symptoms "severe."

Types of Caffeine-containing Beverages

Table 3 shows the prevalence odds ratios of premenstrual syndrome among women with PMS scores equal to 16 through 31 who consumed either caffeine-containing soda or caffeine-containing tea or coffee on a daily basis. The data show that both caffeine-containing soda and caffeine-containing tea/coffee were associated with an increase in the prevalence of premenstrual symptoms, and that the effect was higher, per amount of exposure, for consumers of tea/coffee than for soda consumers over the range of consumption studied (0 to 8 cups of soda, and 0 to 3 cups of tea/coffee). There were too few data on which to base effect estimates for larger amounts of consumption.

TABLE 3—Prevalence Odds Ratios of Premenstrual Syndrome According to Daily Consumption of Caffeine-containing Soda or Tea/Coffee among Women Whose PMS Scores Equaled 16 through 31

Daily Consumption of Caffeine-containing Soda (Cups)**		
0 POR*	1–5 POR*	6–8 POR*
1.0	1.3 (0.9, 2.0)	7.0 (2.1, 22.9)
Daily Consumption of Caffeine-containing Tea/Coffee (Cups)***		
0 POR*	1 POR*	2–3 POR*
1.0	1.4 (0.6, 3.3)	5.3 (2.3, 12.2)

*Prevalence Odds Ratio, Relative to 0 Cups per Day (90% Confidence Interval).
**Based on the 176 cases who consumed caffeine-containing soda on a daily basis but who did not consume caffeine-containing tea or coffee, and on 121 cases who consumed no caffeine-containing beverages.
***Based on the 41 cases who consumed caffeine-containing tea or coffee on a daily basis but who did not consume caffeine-containing soda, and on 121 cases who consumed no caffeine-containing beverages.

Total Fluid Intake

The overall prevalence odds ratio for daily consumption of six to 19 cups of fluid, relative to daily consumption of two to five cups, among women who reported experiencing at least one premenstrual symptom compared with women who reported no symptoms was 1.7 (90% CI = 1.0, 3.0). The prevalence odds ratios for women with PMS scores between 1 and 15 (less severe symptoms) were 1.2 (0.6, 2.4), 1.7 (0.9, 3.3), and 2.1 (1.0, 4.5) for 6–7 cups per day, 8 to 10 cups per day, and 11–19 cups per day, respectively.

Table 4 shows the prevalence odds ratios of premenstrual syndrome for total fluid intake, with and without control for daily consumption of caffeine-containing beverages among the 217 women with PMS scores between 16 and 31 (the same subset of women referred to in Table 2). For these women, the overall prevalence odds ratio (six to 19 cups of fluid consumed per day relative to two to five cups consumed per day) was 1.7 (90% CI = 1.1, 2.7). A steadily increasing prevalence odds ratio was observed for increasing amounts of daily total fluid intake, although the size of the increase was small from six or seven to 11–12 cups of fluid per day. The prevalence odds ratio estimates were reduced somewhat when daily consumption of caffeine-containing beverages was controlled. In both sets of estimates, the only large effects observed were among women consuming between 13 and 19 cups of fluid each day.

The p-values associated with the overall trend between premenstrual syndrome and daily total fluid intake were <0.001 and 0.01 without and with control for daily consumption of caffeine-containing beverages, respectively. The corresponding slopes of the logistic curves were 0.091 (standard error = 0.027) and 0.065 (standard error = 0.026).

Discussion

We found a strong association between consumption of caffeine-containing beverages and the prevalence of premenstrual symptoms which was not explained by daily total fluid consumption. The effects for women with PMS scores between 16 and 31 (women with more severe symptoms) were highly dose-dependent, and were present both among women whose source of caffeine was soda and to a stronger extent (on a per cup basis) among women whose source of caffeine was tea and/or coffee. The relation between daily total fluid consumption and premenstrual symptoms, both with and without control for daily consumption of caffeine-

TABLE 4—Prevalence Odds Ratios of Premenstrual Syndrome According to Daily Total Fluid Consumption with and without Controlling for Daily Consumption of Caffeine-containing Beverages among the 217 Women Whose PMS Scores Equaled 16 through 31

Control for Caffeine-containing Beverages	Daily Total Fluid Consumption (Cups)				
	2–5 POR*	6–7 POR*	8–10 POR*	11–12 POR*	13–19 POR*
No	1.0	1.3	1.5	1.7	3.4
Yes	1.0	1.2	1.3	1.5	2.3
Number of Exposed Women**	59	102	144	63	66

*Prevalence Odds Ratio, Relative to 2–5 Cups per Day.
**Includes Cases and Controls.

containing beverages, was more moderate (over the ranges of values studied) than was the relation for daily consumption of caffeine-containing beverages. The relation between premenstrual symptoms and daily total fluid intake appeared to be dose-dependent, at least when the highest level of intake (13–19 cups per day) was taken into account. The fact that the estimated effects for daily total fluid intake were similar for women with PMS scores between one and 15 (mild symptoms), and for women with PMS scores between 16 and 31 (more severe symptoms), however, seems difficult to reconcile with a causal interpretation of the observed effects.

One possible source of error in this study warrants discussion. We used a self-administered questionnaire to collect information about the presence and severity of premenstrual symptoms. Although a definition of premenstrual syndrome was included in the questionnaire, it may be that some women confused premenstrual with menstrual symptoms or otherwise misinterpreted the meaning of "premenstrual symptoms," and responded accordingly. To evaluate this possibility, 51 study participants volunteered to keep a three-month daily record of symptoms, and to record the first and last days of their menstrual periods. (These women also maintained a daily record of certain dietary factors, exercise history, and stressful life events which may have affected daily symptoms such as anxiety or depression.) These women were told that the purpose of the daily diary was to obtain more detailed information about symptoms throughout the menstrual cycle than was possible using a one-time mail questionnaire. Forty-three (84 percent) of the volunteers completed at least two months of the daily diary. Of these women, 32 (74 percent) completed all three months. One of us (AMR), blinded with respect to the volunteers' responses on the mail questionnaire, rated the severity of premenstrual symptoms (no symptoms, mild, or moderate/severe) for each of the 43 subjects who completed at least two months of the diary, and then compared these ratings to the PMS scores as determined from each woman's mail questionnaire. Overall, 60 percent (26/43) of the ratings and PMS scores matched; that is, the blind assessment of severity, which was based on the actual symptoms recorded in the daily diaries, corresponded to the assessment of PMS severity as summarized in the PMS score (0 = no PMS, 1–15 = mild, 16 or higher = moderate/severe). The majority of the misclassification (11 of 17 misclassified cases) occurred among women who were rated as not having premenstrual syndrome but whose PMS scores equaled one to 15 (mild). Considerably less misclassification occurred between other categories of PMS severity.

The effects of such misclassification, if generalizable to the other study participants, would be to reduce somewhat the size of the observed associations between consumption of caffeine-containing beverages or total fluid intake and PMS among women with PMS scores of 16 or higher, and to substantially reduce the size of the observed associations between consumption of caffeine-containing beverages or total fluid intake and PMS among women with scores of one to 15. The lack of a strong, observed association between consumption of caffeine-containing beverages and PMS among women with PMS scores of one to 15 may be attributable in large part to the inclusion of women who do not regularly experience premenstrual symptoms into this PMS group.

Neither observation bias nor selection bias would seem to be a potentially large source of error in the present study because data from both women who experience premenstrual symptoms and women who do not experience these symptoms were collected in the same manner using identical mail questionnaires; the questionnaire contained an equal number of questions about menstrual health as it contained about premenstrual health; the study hypothesis was not disclosed to the study participants nor is the hypothesis generally well-known or accepted to be true among the population studied; and none of the 51 study participants who volunteered to keep daily records of symptoms (with whom there was bi-weekly personal contact) indicated that she suspected the specific nature of the study hypotheses.

The observed associations between premenstrual symptoms and daily consumption of caffeine-containing beverages theoretically could reflect some uncontrolled third factor but any such factor would have to be present among both caffeine-containing soda consumers and among caffeine-containing tea/coffee consumers, would have to vary jointly with the amounts of such beverages consumed, and would have to be prevalent among young women in both the United States and the People's Republic of China.[5] Neither alcohol use, cigarette use, exercise habits, oral contraceptive use, nor a diet high in beef or refined sugar products, all putative causes/preventives of premenstrual syndrome, satisfies such criteria. A mechanism involving the depressive action of adenosine on central neurons has been suggested by Phillis for how caffeine might cause premenstrual syndrome.[23]

Theoretically, a definitive evaluation of the effects, if any, of caffeine-containing beverages on premenstrual syndrome would involve a randomized trial. The inherent difficulties in conducting such a trial, however, would be considerable.

It seems worth considering that the etiological factors of premenstrual syndrome may operate anew during each menstrual cycle, making the latent period between exposure to caffeine-containing beverages and the onset of premenstrual syndrome short. If true, any beneficial effects which arise from the elimination of caffeine from the diet should be apparent within a relatively few menstrual cycles. For this reason, and because there are few, if any, known beneficial effects associated with caffeine-containing beverage consumption, women who wish to reduce the severity of their premenstrual symptoms may want to consider eliminating caffeine-containing beverages from their diets and then evaluating any changes in the severity of their symptoms after several months. Such a decision, determined on the basis of each woman's personal history with premenstrual symptoms, should be made with full knowledge that there may be no benefits derived from such abstinence for a particular woman. This is the case, however, for many public health recommendations whose bases reside in population-based (epidemiologic) research.

APPENDIX

Subset of the Questionnaire: Question About Beverage Intake

23. About how many *cups* of each of the following beverages do you drink during a typical *day?* (One "cup" is equal to about eight ounces, or one measuring cupful.)

Water:_____
Fruit juice_____
Milk_____
Caffeine-free soft drink_____
Caffeine-containing soft drink_____
Hot chocolate_____
Decaffeinated coffee_____
Caffeine-containing coffee_____
Herbal tea (non-caffeine-containing tea)_____
Caffeine-containing tea_____
Milk shake, ice cream soda_____
Other beverages (note that alcoholic drinks are listed in question 24)_____

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CPHA to Sponsor November 29 Symposium on Proposals for ICD–10

With the implementation of the 10th revision of the *International Classification of Diseases* (ICD–10) scheduled for the first half of this decade, health care providers, policy makers, and others will be able to classify and collect more comprehensive patient data than ever before. ICD–10 represents a complete restructuring of ICD–9–CM, the classification system currently used in the United States to code patient diagnoses in the inpatient hospital setting. Designers of ICD–10 are structuring the new taxonomy so that health care professionals will be able to capture data that will facilitate "cradle to grave" health care analyses.

To address the changes in coding technology that will result with ICD–10, the Commission on Professional and Hospital Activities (CPHA) of Ann Arbor, Michigan is sponsoring a national symposium in Chicago, November 29, 1990. Attendees at the symposium will evaluate the work that has been done thus far on chapter 21 of ICD–10, which deals in large part with ancillary factors, such as social environment or occupational hazards which influence the patient's health status and necessitate contact with health services, both as an inpatient and in the ambulatory setting. Participants from national organizations, speakers who will address specific issues, and a reactor panel all will be featured in the symposium. Health care professionals interested in learning more about the conference or in participating on the reactor panel should contact Marge Zernott, Director of Classification Development at CPHA, tel: (313) 930-7990.

Walking and Serum Cholesterol in Adults

LARRY A. TUCKER, PHD, AND GLENN M. FRIEDMAN, MD

Abstract: We measured the association between walking for exercise and the ratio of total cholesterol/HDL cholesterol in 3,621 adults. After controlling for age, gender, income, body fat, alcohol use, exercise other than walking, and cigarette smoking, adults in the high, moderate, and low duration walking categories were compared to those in the no walking-no exercise category. The relative risk for total/HDL ratios of 5.0 or more were .46 (95% CI = .27, .80), .48 (95% CI = .30, .76), and 1.11 (95% CI = .81, 1.53) respectively. (*Am J Public Health* 1990; 80:1111–1113.)

Introduction

Although hypercholesterolemia is a key risk factor of cardiovascular disease,[1] its determinants, including physiologic, demographic and lifestyle factors, are less well established. Mounting evidence indicates that physical activity fosters favorable levels of serum cholesterol,[2–6] but findings have been inconsistent,[7–13] and many of the investigations have failed to control for the effects of alcohol use, body fat, tobacco use and other factors.

Despite the present-day enthusiasm for walking as a means of promoting health and fitness,[14,15] only two investigations have been conducted to determine the relation between walking and serum cholesterol,[10,16] and they have shown mixed results. Hence, the present study was conducted to assess the relation between walking for exercise and serum cholesterol, particularly the total cholesterol/high density lipoprotein cholesterol (HDL) ratio in 3,621 adult men and women. A secondary purpose was to ascertain the extent to which physiologic, demographic, and lifestyle factors confound the association.

Methods

Subjects

Subjects were employees of over 25 different companies that chose to participate in an ongoing health promotion/disease prevention screening program. Approximately 62 percent of the subjects were female, 85 percent were White, 69 percent were married, and 66 percent reported some college education. Mean age of the sample was 40.0 years (SD = 11.1) and the median gross family income was between $25,000–$30,000.

Approximately 14 percent of the employees elected not to participate in all of the health evaluations leaving a sample of 3,621 adults for the present investigation.

Instrumentation and Procedures

Registered nurses employed by Health Advancement Services, Inc., collected all of the data. Each subject was examined individually and privately for 60 minutes after participating in an orientation and completing an informed consent form. A structured questionnaire was used to collect demographic and lifestyle information. Weekly duration and frequency of walking for exercise was assessed as recommended by Stephens, *et al*,[17] but walking intensity was not measured. Total time spent exercising each week was also measured. A step test, the Kasch 3-minute Pulse Recovery Test,[18] assessed cardiovascular fitness. In the present study, as reported walking duration increased, physical fitness increased showing strong linear (Mantel-Haenszel: $\chi^2 = 48.0$, df = 1, $p = 0.0001$) and general associations ($\chi^2 = 87.5$, df = 18, $p = 0.0001$). Indirect validation of the total exercise time variable was demonstrated previously.[19]

Approximately 10 cc of blood was drawn from each subject and analyzed using the enzymatic method to determine serum cholesterol levels.[20] A Harpenden skinfold caliper was employed to assess subcutaneous fat at three body sites on each subject: for males, thigh, chest, abdomen; for females, iliac crest, triceps, thigh. The sum of the skinfold measurement along with age and gender were used to calculate the total body fat percentage of each subject.[21]

Data Analysis

Subjects were grouped into four categories according to their self-reported, weekly duration of walking for exercise and total exercise time:

- *None*: subjects reporting no weekly walking for exercise nor any regular exercise;
- *Low*: ½–2 hours of weekly walking for exercise;
- *Moderate*: 2½–4 hours of weekly exercise walking;
- *High*: 4½ hrs per week or more of exercise walking.

The total serum cholesterol/HDL ratio was calculated because the total/HDL ratio has greater heart disease predictive utility than the others used individually.[1] A total/HDL ratio of 5.0 or greater was used to signify high risk according to current recommendations.[22]

The relation between weekly walking duration and elevated total cholesterol/HDL ratios was measured by the odds ratio.[23] Subjects who reported no weekly walking for exercise nor any regular physical exercise were used as the reference group. Mantel-Haenszel summary risk estimates[24,25] were employed to control for the potential confounders: age, gender, income, body fat, alcohol use, cigarette smoking, and exercise other than walking. Analysis of covariance (ANCOVA) was used to determine mean total cholesterol, HDL-C and total/HDL cholesterol differences among the walking groups with the potential confounders controlled.

Results

Table 1 shows the relations between the control variables, walking duration, and the total cholesterol/HDL ratio. High walking duration was more common in those who were male, leaner, poorer, nonsmokers or light smokers, not heavy drinkers, those who performed little exercise other than walking, and those in their 20s or above 50 years of age. Those with elevated total/HDL ratios tended to be male,

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TABLE 1—Walking Duration and the Total/HDL-C Ratio According to the Control Variables

Control Variables	col%	Hours of Weekly Exercise Walking (row %)				Total/HDL ratio ≥5
		none[a]	1/2–2	2 1/2–4	4 1/2+	
Total Group	100	54.4	26.8	11.2	7.5	27.5
Age (years)						
19–29	19.4	49.8	29.8	11.9	8.5	17.0
30–39	32.2	53.7	30.0	8.8	7.6	25.4
40–49	26.8	60.0	22.4	12.2	5.4	30.2
50+	21.6	51.7	25.6	13.4	9.3	36.6
Gender						
male	38.1	53.4	25.3	11.0	10.3	44.8
female	61.9	55.1	27.8	11.4	5.8	14.2
Income (k)						
not reported	7.6	56.7	24.0	10.6	8.7	32.7
<15	9.7	50.4	22.6	14.6	12.3	23.7
16–24	17.5	58.2	24.5	10.3	7.1	26.1
25–44	36.8	55.7	26.2	11.1	7.1	28.0
45+	28.4	51.4	31.4	10.9	6.3	26.4
Body Fat						
lean	8.9	42.5	32.8	12.6	12.2	13.6
moderate	57.2	50.0	30.1	12.3	7.7	24.6
obese	33.9	59.5	23.4	9.8	7.3	39.0
Alcohol Use						
none/light	70.0	52.8	28.8	11.4	7.1	19.8
moderate	26.3	56.8	23.0	10.9	9.3	45.1
heavy	3.7	73.0	15.1	6.4	5.6	49.2
Exercise*						
none	83.8	80.2	8.4	6.8	4.6	27.4
1/2–2 hrs.	10.3	0.0	67.8	15.2	17.0	21.4
2–3 1/2 hrs.	3.6	0.0	70.6	29.4	0.0	25.2
> 3 1/2 hrs.	2.3	0.0	85.1	14.9	0.0	20.8
Smoking						
non/ex	80.2	52.8	28.8	11.4	7.0	25.0
1–20 daily	14.9	56.6	21.6	11.6	10.2	35.4
21 + daily	5.0	69.3	17.1	8.0	5.7	46.3

*"Exercise" included all exercise reported except walking.
[a]"None" (reference group), under the "Hours of Weekly Exercise Walking" heading included adults who did not walk for exercise or perform any other type of regular exercise.

older, fatter, smokers, drinkers, non-exercisers, and not poor.

Table 2 displays the prevalence of elevated total cholesterol/HDL ratios among the adults in the reference and walking categories, and estimated risk for each of the groups before and after adjustment for the potential confounders.

Table 3 shows the mean cholesterol differences among the four walking categories with the potential confounders controlled statistically (ANCOVA).

Discussion

The present findings indicate that adults who walk for exercise 2½–4 hours or more each week tend to have less than one-half the prevalence of elevated total cholesterol/HDL ratios (total/HDL ratio ≥ 5.0) as those who do not walk or exercise regularly.

There are a number of explanations as to why walking duration and the total/HDL ratios appear to be strongly related. High total cholesterol/HDL ratios may reflect problems that lead to decreased exercise-walking. Because subjects with elevated total/HDL ratios tend to be more obese and smoke more than their counterparts[1,26] and because obese adults and those who smoke tend to walk less than non-obese and non-smokers (findings of present study), adults with elevated ratios may choose not to walk as much as others. Elevated total cholesterol/HDL ratios and exercise-walking each may be caused by other common factors.

TABLE 2—Estimated Risk of Elevated Total/HDL Ratio by Weekly Walking Duration

Weekly Walking Duration	Variable Controlled	Elevated Total/HDL Ratio[a]			
		N	%	RR_{mh}	95% CI
None (n = 1,970)	none	583	29.6	1.00	—
1/2–2 hrs (n = 972)	none	233	24.0	0.75	0.63,0.89
	age, gender, ses, fat, alcohol, smoking, other exercise[b]			1.11	0.81,1.53
2 1/2–4 hrs (n = 406)	none	90	22.2	0.68	0.53,0.87
	age, gender, ses, fat, alcohol, smoking, other exercise[b]			0.48	0.30,0.76
4 1/2 hrs + (n = 273)	none	73	26.7	0.87	0.65,1.15
	age, gender, ses, fat, alcohol, smoking, other exercise[b]			0.46	0.27,0.80

NOTE: Subjects who reported no regular exercise walking or other exercise were used as the reference group.
[a]Elevated Total/HDL ratios were defined as those ratios ≥ 5.0.
[b]Other exercise included all exercise reported other than walking.

TABLE 3—Adjusted Mean Differences in Cholesterol among the Walking Groups

	Weekly Walking Duration							
	None		Low		Moderate		High	
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Total/HDL Ratio	4.81	1.6	4.70	1.4	4.49	1.3	4.53	1.6
Total-C (mmol/L)	5.34	1.1	5.28	1.0	5.19	1.1	5.19	1.1
HDL-C (mmol/L)	1.18	0.3	1.20	0.3	1.22	0.3	1.22	0.3

Means were adjusted for differences in age, gender, income, body fat, alcohol use, exercise other than walking, and cigarette smoking.

With the moderate and high duration walking groups combined and the covariates controlled, the following mean differences resulted (mmol/L):

Total/HDL ratio: None (M = 4.86) Moderate + High (M = 4.53), difference (.33); 95% confidence limits of the difference (.18, .46), $p < .05$

Total-C: None (M = 5.33) Moderate + High (M = 5.14), difference (0.19); 95% confidence limits of the difference (0.08, 0.29), $p < .05$

HDL-C: None (M = 1.17) Moderate + High (M = 1.21), difference (0.04); 95% confidence limits of the difference (0.01, 0.06), $p < .05$

Other potential confounders, particularly diet, could account for the inverse association between walking duration and elevated total/HDL ratios since research suggests that exercisers may consume different diets than non-exercisers,[27] and diet may be related to serum cholesterol levels.[1]

Finally, desirable total/HDL ratios may, in fact, result from moderate to high levels of exercise walking. Numerous studies confirm a connection between other types of aerobic exercise, besides walking, and favorable levels of serum cholesterol.[2–6]

Because subjects in the present study had relatively high socioeconomic status and education levels, generalization to poorer, less educated groups will necessitate further research.

Given the correlational design of this study, cause-and-effect conclusions are not warranted. However, if a causal relation is assumed, then at least 2½ to 4 hours of exercise walking per week should be practiced for protection against elevated total/HDL ratios. In the present study, ½ to 2 hours of weekly walking for exercise was not associated with decreased risk of high total/HDL ratios.

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Public Health Briefs

Abstinence from Smoking and Expired-Air Carbon Monoxide Levels: Lactose Intolerance as a Possible Source of Error

Ann D. McNeill, PhD, Lesley A. Owen, PhD, Michael Belcher, RMN, Gay Sutherland, MPhil, and Simon Fleming, MSc

Abstract: We investigated a possible source of contamination of expired-air carbon monoxide (CO) readings on CO monitors from raised expired-air hydrogen (H_2) levels, namely lactose intolerance, the norm in certain ethnic groups. A significant correlation between expired-air CO and H_2 readings was observed in four non-smoking lactose maldigesters. (*Am J Public Health* 1990; 80:1114–1115.)

Introduction

Expired-air carbon monoxide (CO) measurement is a simple and cheap method of validating non-smoking status in ex-smokers.[1] While falling short of the specificity and sensitivity of cotinine measures, it is the preferred method in a wide range of field settings.[2] Its application takes on particular value in the light of the increasing use of nicotine replacement strategies in smoking cessation. It is important that this test be valid if the success rates of smoking cessation interventions are to be correctly estimated and individuals spared needless embarrassment should they be suspected of not telling the truth about their smoking status.

We investigated a possible source of false positives in expired-air CO readings.

Method and Results

Bedfont EC50 and EC50 micro smokerlyzer portable CO monitors are the most popular means of measuring expired-air CO in smoking clinics in the United Kingdom[3] and are also used in the United States. The non-smoking/smoking cut-off CO value most commonly used is 10 ppm.[2] A non-smoking member of our staff was surprised to find a level of 36 ppm expired-air CO after routinely blowing into such a monitor as a demonstration of its use. This raised level was obtained with other Bedfont CO monitors and was not attributable to raised ambient CO.

Expired-air CO levels after 20 seconds breath-holding are in approximate equilibrium with the concentration of carboxyhaemoglobin in the blood.[1] However, in this case, carboxyhaemoglobin levels were normal (1.0 percent) indicating that expired-air CO levels were not raised and that the CO monitor must have been picking up another gas.

The instruction manual for these monitors indicated that they are specially constructed to be insensitive to interferences from hydrogen (H_2), alcohol, and other organic species.[4] As alcohol was eliminated as a possible source of interference, H_2 levels were investigated. The technical specification for the CO monitors indicates that 20 ppm H_2 would give less than a 4 ppm reading.[4]

Raised H_2 levels in humans can arise from several causes, such as lactose intolerance, excessive bacteria in the small intestine and variations in transit time through the small intestine.[5] Recent studies also indicate that breath H_2 excretion increases between three and seven hours after normal subjects have ingested flours derived from whole or refined wheat, oats, corn, potatoes or baked beans.[6]

Monitoring the subject's CO levels throughout the day revealed a strong diurnal pattern which was possibly related to food intake (breath hydrogen tends to be higher after waking).[6] Further investigation revealed that this subject was lactose intolerant. A Bedfont EC60 H_2 monitor which measures the H_2 content of expired air, confirmed that H_2 levels were raised and mirrored the apparent CO levels throughout a typical working day (see Figure 1). This suggested that the CO reading was being contaminated by raised H_2 levels.

The relation between expired-air CO and H_2 levels was further investigated using lactose tolerance tests.[7] A significant correlation between the expired-air CO and H_2 readings was observed in this subject and in an additional three

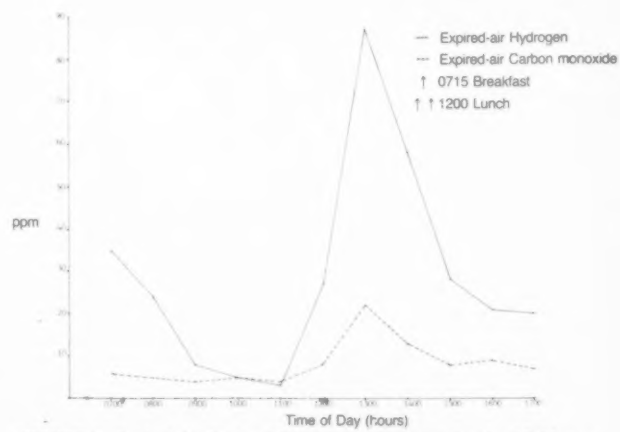

FIGURE 1—Expired-Air Carbon Monoxide and Hydrogen Levels of Subject as a Function of Time of Day

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non-smokers who were known to be lactose maldigesters ($r = 0.99$, $r = 0.95$, $r = 0.97$, and $r = 0.75$; $n = 6$ for each subject; range for CO values = 3 to 34 ppm; range for H_2 levels = 2 to 183 ppm). A regression analysis was carried out using the CO and H_2 values to estimate the level of hydrogen at which a CO reading of 10 ppm would be obtained. A linear trend was fitted across time and yielded a non-significant time-subject interaction and between-subject effect. An expired-air H_2 level of 38.91 ppm would be sufficient to register 10 ppm on a CO monitor (2.5 percent of individuals with levels of 42.07 would register less than 10 ppm, 2.5 percent of individuals with levels of 35.75 would register above 10 ppm). In lactose maldigesters a CO level of 10 ppm would therefore be achieved by consuming, for example, 350 ml or approximately two small glasses of milk.

Discussion

Despite the methodological limitations imposed by the small sample size, the strong patterns observed indicate that there may be cases where individuals classified as smokers on the basis of CO validation have a condition which results in raised expired-air H_2 levels. The proportion of the population who would fall into this category is unknown. As stated earlier, one of the causes of raised H_2 levels is lactose intolerance, which is thought to have a strong hereditary component: a recent estimate of primary lactose intolerance in the UK is given as 6 percent.[8] However, it can also be acquired following diseases which damage the small intestinal villi, for example, giardiasis, coeliac disease, tropical sprue, chronic alcoholism, rotavirus infections in children, and cholera.[7,8] Furthermore, not all lactose intolerant individuals are aware of their condition[9] and some can tolerate moderate quantities of milk, possibly as a result of acquired tolerance to the symptoms.[7]

The likelihood of middle-class Caucasians having raised H_2 levels due to lactose intolerance which could contaminate CO levels may be minimal. However, in certain ethnic groups the prevalence of lactose intolerance is much higher, for example, Jews,[9] American Blacks (among whom the figure is 75 percent), and certain African and Oriental races where it approaches 100 percent.[8] It would be counter-productive if a greater proportion of minority groups, when attempting smoking cessation, were to be wrongly labeled. Among such groups, CO validation may need to be supplemented by cotinine, or if nicotine replacement is being used, by carboxyhaemoglobin analysis. It would not be sufficient when performing the expired-air CO test to ask when the subject last consumed a dairy product as there are considerable inter and intraindividual differences in rates of absorption.[6]

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Abstracts Sought for Nursing Research Conference

Abstracts are invited for the 10th annual research conference, entitled "Building Knowledge for Nursing: The Impact of the Human Immunodeficiency Virus," co-sponsored by the University of South Florida College of Nursing, and Delta Beta Chapter of Sigma Theta Tau, and scheduled to be held February 21–22, 1991 in Tampa, Florida. The deadline for submission of abstracts is September 30.

For further information, contact: Loretto Ramella Lopez, RN, EdD, Chair, Research Committee, University of South Florida, College of Nursing, Tampa, FL 33612-4799. Telephone: (813) 974-2191.

A Population-Based Descriptive Study of Housefire Deaths in North Carolina

MICHAEL J. PATETTA, MA, AND THOMAS B. COLE, MD, MPH

Abstract: We report a population-based study of housefire deaths in North Carolina in 1985 using data obtained from fire investigators and the North Carolina medical examiner system. The crude death rate was 3.2 per 100,000 population; age-specific death rates were highest for ages 75–84 years. Death rates for Whites were one-third as high as death rates for other races. Of those decedents tested for alcohol, 56 percent had blood alcohol levels ≥ 22 mmol/L. Most fatal fires were caused by heating units or cigarettes. (*Am J Public Health* 1990; 80:1116–1117.)

Introduction

Three-fourths of the 5,000 fire and burn deaths that occur annually in the United States are caused by housefires.[1,2] In 1984, the US housefire death rate[1] was 1.9 per 100,000 population. However, in the South the rate[1] was 2.5 per 100,000, and in the Southeast in recent years rates have been in the range of 3.05–5.14 per 100,000.[2]

Studies of housefire mortality for the Southeast have found that most deaths occur in heating-related fires, whereas smoking-related fires are responsible for most of the deaths in other parts of the country.[3] There is some evidence that the higher death rate for the Southeast may be attributable to a lower prevalence of smoke detectors and a greater use of portable heating equipment compared with other regions.[4,5]

To describe the epidemiology of housefire deaths in a Southeastern state, we conducted a statewide, population-based study of housefire deaths in North Carolina.

Methods

Records of all housefire deaths in North Carolina in 1985 were obtained from the Office of the Chief Medical Examiner. State law mandates reporting of all fatal injuries to a medical examiner. County medical examiners complete a report (which includes toxicologic information, reports of death scene visits and interviews with witnesses, autopsy records, and other relevant data) for each investigated death for review by a forensic pathologist.

Information about the causes of the housefires was requested from the local fire marshalls or county sheriffs, and responses were received for 70 percent of housefire deaths. The causes of the housefires were documented in Medical Examiner Reports or State Bureau of Investigation Reports for an additional 22 percent of deaths.

Rates and proportions were calculated from pooled data. Housefire death rates are expressed per 100,000 population, but these rates are approximate because some decedents were not North Carolina residents and information was not obtained for North Carolina residents who died out of state.

Results

In 1985, 159 housefires resulted in 200 deaths, accounting for 75 percent of all North Carolina deaths from fire and burns. All but three decedents were North Carolina residents. The crude death rate from housefires was 3.2 per 100,000 population. Death rates were highest (8.8/100,000) for persons ages 75–84 years (Table 1) and for persons other than White (7.1) (Table 2).

Of 157 persons who died at the scene of the fire and were tested for carboxyhemoglobin, 131 (83 percent) were reported by a medical examiner to have died of carbon monoxide poisoning and 26 from burns. Of the 26 not tested, seven (27 percent) were reported to have died of carbon monoxide poisoning and 19 from burns.

Of 160 decedents ≥ 15 years of age, 56 percent of the 130 tested for alcohol had blood alcohol levels ≥ 22 mmol/L (legally impaired under North Carolina driving laws). Of those tested, 85 percent who died in cooking-related housefires, 60 percent who died in smoking-related housefires, and 39 percent who died in heating-related housefires had blood alcohol levels ≥ 22 mmol/L.

Most of the victims (59 percent) died when single-family houses burned, although 76 percent of the North Carolina population lived in single-family houses according to the 1980 census. Although only 9 percent of the North Carolina population lived in mobile homes in 1980, 19 percent died in mobile home fires. Smoke detector information was provided for 72 fatal housefires. Of the 109 deaths in these housefires, 94 percent occurred in dwellings without smoke detectors.

More deaths resulted from housefires caused by heating units (33 percent) than from any other cause (Table 3). Of the 60 heating-related housefire deaths, 20 percent were associated with the use of flammable liquids and 25 percent with the ignition of furniture or furnishings. Woodstoves caused the fires that resulted in 37 percent of heating-related deaths, and kerosene heaters were responsible for an additional 20 percent.

TABLE 1—Age-specific Housefire Death Rates,* North Carolina, 1985

Age Group (years)	Number of Deaths	Death Rate
0–4	23	5.5
5–14	17	1.9
15–24	20	1.8
25–34	12	1.1
35–44	20	2.4
45–54	22	3.5
55–64	27	4.6
65–74	35	8.1
75–84	19	8.8
≥85	5	8.1
Total	200	3.2

*Death rates per 100,000 popuiation are approximate because all instate deaths were counted regardless of the state of residence.

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TABLE 2—Race/Sex-Specific Housefire Death Rates,* North Carolina, 1985

Race/Sex Group	Number of Deaths	Death Rates
White Males	65	2.8
White Females	34	1.4
Other** Males	54	7.1
Other Females	47	5.7
Total	200	3.2

*Death rates per 100,000 population are approximate because all instate deaths were counted regardless of the state of residence.

**With the exception of one American Indian male, all deceased persons of other races were Black. Census data for 1985 do not provide population estimates for Blacks and American Indians.

TABLE 3—Housefire Deaths by Cause* of Fire, North Carolina, 1985

Cause of Fire	Number (%) of Deaths
Heating	60 (33)
Smoking	47 (26)
Cooking	16 (9)
Electrical	15 (8)
Arson	10 (6)
Open Flame	9 (5)
Children Playing	9 (5)
Natural	1 (1)
Undetermined	16 (8)
Total**	183 (101)

*Causes are assigned according to the hierarchy used by the National Fire Data Center of the Federal Emergency Management Agency, so that the number of deaths in each sucessive cause is exclusive of all previously listed causes. For 17 deaths, cause of fire information was not provided by fire investigators.

**Total is > 100% due to rounding.

The second most common cause of housefire deaths was smoking (26 percent). Of the 47 smoking-related housefire deaths, 89 percent occurred in fires caused by the ignition of furniture.

Discussion

Only 16 persons who died in 1985 as result of housefires in North Carolina left the scene of the fire alive. Therefore efforts to improve the emergency treatment and transport of persons injured in housefires could prevent at most 8 percent of housefire deaths. To prevent housefire deaths, fires must be prevented or escaped.

Assuming that the proportion of the North Carolina population living in mobile homes did not change from 1980 to 1985, mobile home dwellers are 2.7 times as likely to die in housefires as persons who live in single-family detached homes. A similar risk was found in New Mexico.[6]

We estimate that the lethal[7–10] combination of alcohol and cigarettes resulted in at least one-fourth of the deaths reported in this study. The proportion of fatally injured persons tested who were impaired with alcohol was greater for housefires (56 percent) than that for any other cause of unintentional injury in North Carolina. The use of fire-safe cigarettes[11] may be the most effective way to prevent deaths associated with drinking and smoking.[2,6]

Escape from housefires is facilitated by early warning, which can be provided by smoke detectors.[8,12] The risk of death from housefire is estimated to be 2.5 times greater for persons who live in homes without smoke detectors.[13] Unfortunately, persons who are very young, very old, or very drunk may not be able to respond to an alarm.[11,13–15] From our data we estimate that these groups accounted for over half of the deaths.

Our study indicates that the risk of housefire death is likely to be multifactorial, with ignition source, type of dwelling, and behavior each playing important roles.

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We would like to thank the Fire and Rescue Service Division of the North Carolina Department of Insurance; the North Carolina State Bureau of Investigation; and the Division of Post-Mortem Medicolegal Examination of the North Carolina Department of Environment, Health, and Natural Resources for their guidance, their data, and their enthusiastic support.

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Estimating the Full Cost of Workplace Injuries

MICHAEL T. FRENCH, PHD

Abstract: While many studies estimate medical costs and lost wages from workplace injuries, few have estimated pain and suffering costs. This study develops a wage-risk model for railroad workers and estimates the total cost of on-the-job injuries. On average, the total cost of an on-the-job injury to a railroad worker in 1980 was between $19,500 and $22,500. These estimates are substantially higher than the sum of medical costs plus lost wages. (*Am J Public Health* 1990; 80:1118–1119.)

Introduction

Workplace injuries impose a variety of costs on injured workers. The most frequently estimated costs are expenditures for medical services and wages lost from missed work. But injured workers also bear other costs such as pain and suffering.

Economists use several methods to estimate the cost of workplace injuries. The conventional approach is the cost-of-illness method.[1–5] In simple form, the cost-of-illness method estimates the cost of injury as the sum of medical-related expenditures plus lost wages. The strengths of the cost-of-illness method are that it uses accessible data on medical expenditures and personal earnings, and it is easy to apply. The main weakness of the cost-of-illness method is that it neglects important components of the total cost of workplace injury.

Recently, economists have developed methods to estimate the cost of workplace injuries based on individuals' willingness to pay to reduce injury risks. If workers perceive and understand the consequences of on-the-job injuries, the willingness-to-pay method captures the full range of costs to individuals.

The willingness-to-pay method is not without limitations, however. If workers underestimate their true exposure to workplace risks and if real or perceived barriers inhibit workers from reacting to risk changes (e.g., long-term labor contracts), then injury cost estimates will be downward biased. In addition, since willingness to pay is influenced by workers' income, *ability* to pay is inseparable from *willingness* to pay. Nevertheless, the willingness-to-pay method represents a major stride toward estimating the full cost of workplace injuries.

Several researchers have used the willingness-to-pay method to estimate the cost of premature death,[6–10] but few have studied nonfatal injuries.[11,12] This paper develops a willingness-to-pay model and estimates the full cost to workers of on-the-job injuries in the railroad industry.

Methods

Generally, workers' wages depend on demographic characteristics of the workers (e.g., age, education, marital status, and region of residence), characteristics of the job (e.g., blue collar, white collar, skilled, or unskilled), and the riskiness of the job. Job risk can be measured as the number of injuries per million worker-hours. Using multivariate regression analysis applied to data from a sample of workers, researchers can estimate the importance of each variable in determining workers' wages. In particular, the analysis shows how much workers must be paid to get them to accept additional workplace risk, other things held equal. The amount workers must be paid to accept additional risk is called the "compensating wage differential."

The compensating wage differential is at the heart of the willingness-to-pay method for estimating total cost imposed on workers by on-the-job injuries. For example, suppose the regression analysis shows that the compensating wage differential is $0.50, calculated on weekly wages. In other words, on average, workers in the sample are willing to accept a $0.50 reduction in weekly wages ($26 per year) for a one-unit decrease in the workplace injury rate. If the annual probability of workplace injury is 0.004, then four in 1,000 workers are likely to be injured on the job each year. Since on average each individual is willing to accept a $26 reduction in annual wages to reduce the probability of injury to 0.003, or three in 1,000, then the workers' total willingness to pay to avoid one injury is ($26 per worker)(1,000 exposed workers) = $26,000.

This analysis uses wage, demographic, and occupational data from the March 1980 Current Population Survey[13] plus on-the-job injury data from the Federal Railroad Administration[14] and the Association of American Railroads[15] to estimate railroad workers' willingness to pay to reduce workplace risk. The sample includes railroad workers living in the 48 continental states who reported wages, demographic characteristics, personal characteristics, and occupation by Census Bureau categories. Discarding records with missing data and records for workers who had more than one employer in 1980 left a sample of 460 railroad workers. Table 1 lists all variables used in the regression model along with sample means and standard deviations.

TABLE 1—Variable Means and Standard Deviations (N = 460)

Variables	Mean	Standard Deivation
Weekly wage	437.375	133.542
Male	0.935	0.247
Caucasian	0.928	0.258
Age	39.863	12.831
Included in company pension plan	0.876	0.330
Included in company health plan	0.961	0.194
Head of household	0.878	0.327
Residence in northeastern US	0.163	0.370
Residence in southern US	0.320	0.467
Residence in north central US	0.243	0.430
Married	0.748	0.435
Highest grade attended	12.920	2.420
Occupation		
Professional and technical	0.028	0.166
Managers and administrators	0.141	0.348
Sales and clerical	0.122	0.327
Craftsmen	0.352	0.478
Operators	0.235	0.424
Laborers	0.020	0.139
Injuries per million worker-hours	32.974	23.241

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Results

I estimated several different wage-risk regression models. Table 2 gives a complete set of parameter estimates and regression statistics from the linear and log-linear specifications. Most coefficients have plausible signs and are statistically different from zero at the 0.05 or higher level of significance.

On average, the estimated annual compensating wage differential for railroad workers is 0.0021 (the regression coefficient for the risk variable from the log-linear specification) multiplied by $437 (the average weekly wage for workers in the sample), multiplied by 49 (the average number of weeks worked per year), which yields a value of $45. To compute a total willingness-to-pay estimate, I multiply the compensating differential estimate ($45) by the average number of workers affected by the injury risk [500 workers = (1 million worker-hours/2,000 hours)], yielding an estimate of $22,500 per injury. Using the linear specification, I estimate railroad workers' total willingness to pay to reduce the risk of workplace injury at $19,500 per injury. All other estimates from different specifications of the regression model fall in the range of $19,500 to $22,500.

TABLE 2—Estimation Results for Linear and Log-Linear Wage-Risk Equations

Independent Variable	Parameter Estimates (N = 460)* Log-linear Equation (R^2 = 0.32)	Linear Equation (R^2 = 0.31)
Constant	4.8940	4.9504
	(0.1371)	(59.5639)
Married	0.0027	−4.2994
	(0.0311)	(13.5067)
Head of household	0.0739	36.7836
	(0.0416)	(18.0759)
Male	0.0653	17.9962
	(0.0603)	(26.2137)
Caucasian	0.0321	23.3190
	(0.0491)	(21.3247)
Included in company pension plan	0.1210	34.7646
	(0.0388)	(16.8389)
Included in company health plan	0.1337	18.0227
	(0.0671)	(29.1577)
Residence in northeastern US	−0.0034	3.6966
	(0.0387)	(16.8197)
Residence in north central US	0.0592	22.3930
	(0.0323)	(14.0148)
Residence in southern US	−0.0295	−12.7819
	(0.0350)	(15.2006)
Age	0.0025	1.2286
	(0.0011)	(0.4786)
Highest grade attended	0.0187	9.4581
	(0.0059)	(2.5827)
Professional and technical	0.4783	169.4618
	(0.1066)	(46.3366)
Managers and administrators	0.5523	225.6686
	(0.0675)	(29.3057)
Sales and Clerical	0.3213	94.4426
	(0.0812)	(35.2666)
Craftsmen	0.3467	118.2067
	(0.0588)	(25.5369)
Operators	0.2958	104.5947
	(0.0487)	(21.1727)
Laborers	0.1074	33.4127
	(0.0975)	(42.3456)
Injuries per million worker-hours	0.0021	0.7964
	(0.0009)	(0.3967)

*standard errors in parentheses

Discussion

In 1980, the average on-the-job injury in the railroad industry resulted in 16 days away from work. If workers received no compensation for lost wages and if injured workers spent all 16 days in the hospital—which are clearly overstatements—then the cost-of-illness method estimates the average cost of an injury to an injured worker at only $5,318 ($245 per day for hospital care and $437 per week in lost wages).

Willingness-to-pay estimates from the wage-risk model presented in this paper indicate that, on average, railroad industry employees faced an expected cost of between $19,500 and $22,500 per injury. These estimates are considerably larger than the sum of medical costs and lost wages because they include the cost of pain and suffering. Comparing cost-of-illness and willingness-to-pay estimates suggests that the *avoided* cost of pain and suffering is a substantial component of the total value of workplace safety for railroad workers and that conventional cost-of-illness estimates grossly underestimate the full cost of these injuries.

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Perforating Eye Injury in Allegheny County, Pennsylvania

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Abstract: From 1980 through 1986, acute perforating eye injury (ICD codes 871.0–871.9) was diagnosed in 345 residents of Allegheny County, Pennsylvania. The mean incidence rate was 3.49 per 100,000 person years. There was no significant change in incidence over the seven-year period. The largest number of injuries occurred among individuals working with tools, of which 47 percent were occupational. Males had a 6.5-fold risk of injury relative to females. Blacks had a risk of 2.2 times that of Whites, mainly due to an excess of assaultive injuries. Individuals who had had recent ocular surgery accounted for 4.6 percent of cases overall, and for 31.6 percent of cases in those over age 60. (*Am J Public Health* 1990; 80:1120–1122.)

Introduction

Eye injuries are a major cause of visual impairment. They are the leading cause of monocular blindness in the United States, and account for 7 percent of bilateral blindness in the 20–64 year age group.[4]

While several population-based studies of eye injuries have been published, they include predominantly minor injuries which are unlikely to lead to loss of vision.[1–6] Perforating eye injuries threaten vision and can be expected to result in hospitalization at the time of occurrence. Two previous hospital record studies of eye injuries in defined populations have included a report of the incidence of perforating eye injuries. Karlson and Klein reported an incidence of ocular lacerations of two per 100,000 in Dane County, Wisconsin in 1979.[3] Tielsh, *et al*, reported an incidence of open wounds of the eyeball of 3.81/100,000 in Maryland during the years 1979 through 1986.[6]

Only one previous study has reported both incidence rates and risk factors for perforating eye injuries. Blomdahl and Norrell[7] studied perforating eye injuries in the population of Stockholm through review of hospital records and interviews of patients identified as cases. They reported an incidence of 60.6 per million population among men and 11.8 per million among women during the period 1974–79. The highest incidence was in the 10–19 years age group. The most frequent circumstance of injury was paid work, followed by play or sport, domestic work, assault, and traffic accidents. Metal and glass fragments were the most common perforating objects, followed by nails and knives.

We describe the epidemiology of perforating eye injury in Allegheny County, Pennsylvania during the years 1980 through 1986.

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Methods

Inpatient records were reviewed at the 24 hospitals in Allegheny County and surrounding areas which serve Allegheny County residents. Cases were defined as any Allegheny County resident discharged from one of the listed hospitals with a primary or secondary diagnosis of perforating eye injury (ICD codes 871.0–871.9).

Demographic information, as well as address at the time of admission, the type of injury incurred, and surgical procedure performed, if any, were abstracted from the records. A brief description of the circumstances surrounding each injury was compiled; information was available for 97 percent of cases. E-codes from the record were not used. Cases included were only those with a first time admission for that diagnosis. Cases seen at more than one hospital for the incident injury (e.g. a community and a tertiary care hospital) were counted only once.

Two hospitals refused to allow review of their medical records. However, they provided a count of cases meeting our ICD code criteria, although no information as to whether the case was an Allegheny County resident. From this information, we estimate that we missed three cases at most from these hospitals.

Population data for Allegheny County were obtained from the Pennsylvania State Data Center. For 1980, these data were from the national census. For other years, estimates developed by the State Data Center by sampling were employed. Rates for each year were age standardized against the 1980 Allegheny County population; 95 percent confidence limits for all rates were calculated.[8] Taylor series confidence intervals were constructed for all measures of relative risk.[9]

In order to assess the effect of socioeconomic status on risk of injury, census tract of residence was determined from the Cole's directory for a random sample of 150 cases, stratified by race.[10] Mean income was determined for census tract of residence from 1980 census data.[11]

Results

Three hundred forty-five Allegheny County residents sustained perforating eye injuries during the years 1980 through 1986, for a mean incidence rate of 3.49 per 100,000

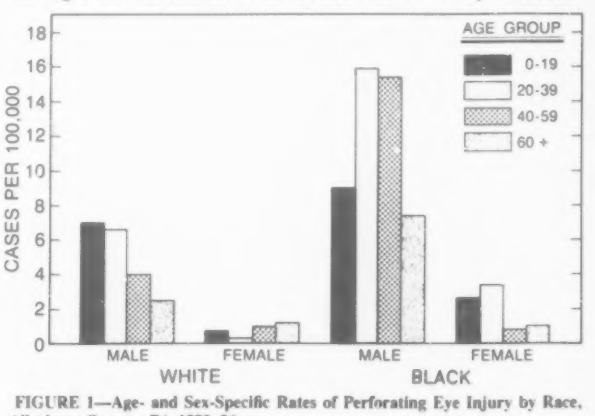

FIGURE 1—Age- and Sex-Specific Rates of Perforating Eye Injury by Race, Allegheny County, PA 1980–86

person years. Annual incidence rates were stable over the seven-year period. Mean incidence rates for the seven-year period, specific for age, race and sex, are shown in Figure 1.

There was only a slight difference in incidence between Whites and Blacks for those under age 20. For those over age 20, the incidence in Whites was reduced, while for Blacks it increased, reaching a peak among those ages 20–59, and then declined in those over age 60 (Table 1). Assaultive injuries were the major contributor to the increased incidence in Blacks (Table 2). When assaultive injuries were excluded, there was no difference in rates between the racial groups for those ages 20–39, although an excess of injuries was still seen among Blacks over age 40.

Injuries to males occurred at a rate 6.6 times that of females, with excess risk for males in all age groups (Table 1) and all categories of cause (Table 2). When occupational injuries were excluded, the relative risk for males compared to females remained elevated at 5.4.

Circumstances of injury are presented in Table 2. The most frequent circumstance of injury, accounting for 23 percent of cases, was work with tools; 47 percent of injuries in this category were occupational. Most injuries occurred when bits of metal, wood, or other material broke off and were propelled into the eye, or when a hammered nail recoiled. However, the tool itself was the cause of injury in four of 11 cases occurring during car repair. In contrast, the tool itself caused injury in only one out of 68 cases not associated with car repair. Five out of 36 occupational injuries occurred when a particle was propelled by another person's work or by machinery in the workplace.

Of recreational activity and childplay injuries, 23 percent were due to BBs.

Minor trauma (e.g. "bumped eye") to individuals with a history of opthalmologic surgery was responsible for 3.2 percent of injuries. We separately analyzed all injuries which had occurred within three months of ocular surgery, whether due to minor or more significant trauma. These injuries accounted for 4.6 percent of cases overall. Among those over age 60, 31.6 percent of all injuries had occurred within three months of surgery. In contrast only 1.3 percent of injuries in those under age 60 were associated with recent ocular surgery.

Falls were the circumstance of injury for 6.7 percent of cases. Seven of 23 fall injuries occurred in nonnormal eyes (postsurgical or underlying condition such as Terrien's marginal degeneration).

Seven and a half percent of injuries were sustained in motor vehicle accidents. The hospital record provided little information about the part of the vehicle actually causing the injury.

TABLE 1—Relative Risk of Perforating Eye Injury, Black:White and Male:Female, by Age, Allegheny County, PA 1980–86

Age	RR B/W	(95% CI)	RR M/F	(95% CI)
0–19	1.30	(.82, 2.07)	7.71	(6.68, 8.74)
20–39	2.77	(1.89, 4.07)	11.18	(10, 12.36)
40–59	2.93	(1.64, 5.23)	4.88	(3.21, 6.55)
60+	2.17	(1.09, 3.25)	2.53	(1.25, 3.81)
Total (Age-Adjusted)	2.18	(1.53, 3.09)	6.55	(5.98, 7.12)

Ten percent required enucleation. Thirteen of these 34 cases were due to assault.

Individuals living in census tracts with mean annual incomes below $10,000 had an incidence rate of 7.4 per 100,000 person years, compared to a rate of 3.2 per 100,000 person years for those living in census tracts with mean incomes above $10,000. The relative risk for low income vs high income census tract residence was 2.3, with a 95 percent confidence interval of 1.47, 3.56.

Discussion

Comparison with the Swedish study of perforating eye injuries (6.6 per 100,000 for men and 1.2 for women) shows that our sex-specific incidence rates of 5.7 and 1.5, respectively are quite similar; they also showed no consistent trend over time.

Young males experienced the highest risk of injury in both studies. We found an increased risk among Blacks. However, this excess risk may represent lower socioeconomic status of Blacks. Individuals living in low income census tracts had a higher risk of injury; mean income for Blacks in Allegheny County is substantially lower than that for Whites.[11]

Among individuals working with tools, over half the injuries occurred in nonoccupational settings. In the occupational setting, injuries can be prevented through required eye protection or separation of persons without eye protection from areas in which they might sustain injury. The many injuries which occurred during home and car maintenance and repair are preventable if people become aware of the risk of eye injury in this setting and wear protection. Efforts to increase public awareness of this hazard are indicated.

Many product-associated injuries can be prevented either by modification or elimination of the product. The continuing rate of visual loss due to BB gun injuries is an example.

TABLE 2—Relative Risk of Perforating Eye Injury by Circumstance, Black:White and Male:Female, Allegheny County, PA 1980–86

Circumstance	Cases (%)	RR B/W	(95% CI)	RR M/F	(95% CI)
Work with tools	78 (22.6)	1.03	(.45, 2.34)	38.80	(3.1, 517.2)
Recreation/Childplay	69 (20.0)	1.35	(.59, 3.08)	13.35	(2.54, 71.69)
Assault	47 (13.6)	9.41	(3.11, 28.5)	6.42	(1.35, 30.24)
Motor Vehicle Accident	26 (7.5)	1.03	(.25, 4.26)	12.84	(.90, 194.9)
Falls	23 (6.7)	1.96	(.70, 10.32)	1.39	(.29, 6.55)
Post Surgical-Minor Trauma	11 (3.2)	2.37	(.33, 16.99)	3.52	(.28, 45.55)
Other*	79 (22.9)	1.93	(.82, 4.52)	4.95	(1.72, 14.25)
Unknown	12 (3.5)	1.40	(.20, 9.74)	1.40	(.20, 9.74)

*explosions, walking into objects, recoiling objects, animal bites and scratches, accidental gunshot wounds.

A surprisingly large number of injuries occurred among those with recent ocular surgery. This should be investigated further, as such cases were responsible for over a third of injuries to those over age 60.

ACKNOWLEDGMENTS

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Fatal and Severe Injury: Scooter and Moped Crashes in California, 1985

MICHAEL SALATKA, MPH, SHAKEH ARZEMANIAN, MPH, JESS F. KRAUS, MPH, PHD, AND CRAIG L. ANDERSON, DHSC, MPH

Abstract: Fatal and severe injury crashes for scooters and mopeds in California for 1985 were compared with those for motorcycles during the same year. Scooters had more than twice the injury crash rate of mopeds but one-half the rate of motorcycles. Age of injured drivers and crash patterns for scooters, mopeds, and motorcycles varied significantly. (*Am J Public Health* 1990; 80:1122–1124.)

Introduction

In the past 15 years, the use of mopeds and scooters in the United States has increased markedly[1]; by 1987, there were an estimated 360,000 scooters and 170,000 mopeds in use.[2]

Some information is available on injuries from moped crashes[3–13] but virtually none on injuries involving scooters. Unlike motorcycles, mopeds and most scooters are not designed for traveling long distances or at high speeds and are not legal for use on freeways in California and most other states. We examine fatal and severe injury crashes for operators of mopeds and scooters in California during 1985 and compare their crash rates and other characteristics to those of motorcycles.

Methods

Definition, Criteria, and Crash Data Source

A moped is a two-wheeled, motor-powered bicycle with an automatic transmission and 12- to 17-inch wheels; almost all have an engine displacement of 49 cc or less. As single rider vehicles, they are not designed to carry passengers. Models made before 1981 may have pedals for propulsion.[14–18]

A scooter is a two-wheeled, motor-powered vehicle with an enclosed engine, automatic transmission, 10- to 12-inch wheels, and engine displacement ranging from 50 cc to 250 cc. Scooters have a floorboard, no pedals for propulsion, and may have a passenger seat.[14,18,19]

In California, the operator of a scooter must pass the same licensing tests as motorcycle operators; scooters must be registered annually. A moped may be operated with any class of driver's license. Annual registration is required for scooters and mopeds without pedals, including mopeds manufactured since 1981.[20]

This research was limited to moped and scooter crashes on public roads in California during 1985 that resulted in either a fatal or severe injury to the driver of the vehicle. General methods of crash injury data collection are contained in an earlier report.[21] Severe injuries are those resulting in a distorted body member or a laceration from which blood flows freely. Off-road injury crashes were excluded.

Fatal and severe injury crashes were identified from the computerized Statewide Integrated Traffic Records System maintained by the California Highway Patrol.

Crash Data and Exclusions

Of the 322 crashes of two- and three-wheel motor vehicles that were not street-legal motorcycles, the following were excluded: 81 off-road motorcycles, 33 all-terrain vehicles, 18 other vehicles that could not be identified.

Information on the vehicle identification number, vehicle license plate number, make, model, and model year was abstracted from the original crash report.

The 1985 California Department of Motor Vehicles registration file provided information on 156,114 two- or three-wheeled vehicles after all motorcycles were excluded. Off-road or all-terrain vehicles (16,649) and scooters or mopeds with registrations expiring before 1985 or first sold in 1986 (41,572) were also excluded. The number of scooters and mopeds at risk of crashing during 1985 was 97,893 in the State of California.

The date on which the vehicle was first sold or registered in California and the registration expiration date were used to

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TABLE 1—Fatal and Severe Injury Scooter, Moped, and Motorcycle Crash Rates per 10,000 Registered Vehicle-Years, California, 1985

Vehicle	Crashes	Registered Vehicle Years	Crash Rate	Standard Error*
Scooters	131	34,174	38	3
Mopeds	59	37,286	16	2
Total**	190	75,907	25	2
Motorcycles***	1,832	246,562	74	2

*Calculated as (crashes)$^{1/2}$/(registered vehicle-years).
**The total includes 4,447 registered vehicle-years for scooters and mopeds that could not be classified as to type.
***Motorcycle data from reference 21.

estimate potential use-months during 1985. The scooters and mopeds registered in 1985 represented 75,907 potential use-years.

The 17-character Vehicle Identification Number was used to distinguish scooters and mopeds from other two- and three-wheel motor-powered vehicles.

Data on motorcycle crashes from a previous report[21] are included for comparison. The chi-square statistic was used to test for homogeneity between results for scooters, mopeds, and motorcycles.

Results

Almost 69 percent of the 190 total crashes involved scooters and 31 percent involved mopeds. Scooters had more than twice the crash rate of mopeds, although both rates were considerably less than the rate for motorcycles (Table 1).

The percentages of fatal injury crashes were 8.5 for mopeds, 11.4 for scooters, and 19.6 for motorcycles. The odds of motorcycle fatal injuries compared to those of mopeds and scooters was 1.9 (95% confidence interval = 1.2, 2.9).

Over 59 percent of injured scooter operators and almost 41 percent of injured moped operators were under 20 years of age (Table 2), compared with less than 20 percent of injured motorcycle operators. Nearly 5 percent of the scooter crashes and about 7 percent of the moped crashes involved an operator less than 15 years of age.

In the fatal and severe scooter and moped crashes, 79 percent of the operators were male. Over 98 percent of injured motorcycle operators were male.

Broadside (either vehicle) collisions accounted for 41 percent of the scooter crashes and 51 percent of the moped crashes (Table 3). With the data available, we could not distinguish which vehicle was struck in broadside collisions.

Scooters and mopeds have considerably higher proportions of broadside and rear-end collisions than do motorcycles. Motorcycles, however, overturned* and hit objects more often than did scooters and mopeds.

Scooters and mopeds crashed most frequently in spring and fall, and the highest proportion of crashes occurred on Monday and Friday. More than 60 percent of all scooter and moped crashes occurred between noon and 8:00 pm, and fewer than 10 percent occurred between 2:00 am and 10:00 am. Motorcycles crashed most frequently in the summer and on Saturdays and Sundays. Fifty-one percent of motorcycle crashes occurred between noon and 8 pm.

*Defined by the California Highway Patrol as falling to the ground without a preceding collision.

TABLE 2—Number and Cumulative Percentage of Fatal and Severe Injury Scooter and Moped Crashes According to Age of Operator, California, 1985

	Scooters			Mopeds			Motorcycles		
Age (years)	Number	Percent	Cumulative Percent	Number	Percent	Cumulative Percent	Number	Percent	Cumulative Percent
<15	6	4.6	4.6	4	6.8	6.8	6	0.3	0.3
15–19	71	54.6	59.2	20	33.9	40.7	352	19.4	19.7
20–24	19	14.6	73.8	16	27.1	67.8	674	37.0	56.7
25–29	10	7.7	81.5	3	5.1	72.9	362	19.9	76.6
30+	24	18.5	100	16	27.1	100	425	23.4	100.0
All Ages	130*	100.0		59	100.0		1,819*	100.0	

*Age was unknown for one scooter operator and 13 motorcycle operators.

TABLE 3—Fatal and Severe Injury Scooter, Moped, and Motorcycle Crashes by Collision Type, California, 1985

	Scooters		Mopeds		Motorcycles	
Collision Type	Number	Percent	Number	Percent	Number	Percent
Impact with another vehicle	107	81.7	52	88.1	1191	65.0
Head-on	18	13.7	6	10.2	179	9.8
Broadside	54	41.2	30	50.8	610	33.3
Rear-end	24	18.3	9	15.2	215	11.7
Sideswipe	11	8.4	7	11.9	187	10.2
No other Vehicle	24	18.3	7	11.9	641	35.0
Hit object	13	9.9	2	3.4	363	19.8
Overturn	7	5.3	4	6.8	245	13.4
Other	4	3.0	1	1.7	33	1.8
All Types	131	100.0	59	100.0	1832	100.0

Over 87 percent of fatal and severe scooter and moped crashes occurred in urban areas. About 95 percent of the crashes occurred on streets and roads that are not state highways compared with 79 percent of motorcycle crashes.

Discussion

Scooters have a higher crash rate than mopeds. This could represent different usage patterns or a different crash risk with similar usage.

The higher percentage of severe injury crashes as opposed to fatal crashes, for mopeds and scooters compared with motorcycles, suggests different patterns of use and speed capabilities of these vehicles.

The higher proportion of scooter and moped crashes involving a collision with another motor vehicle may be due to their frequent use in urban areas and their decreased visibility to other motorists due to their smaller size.

Teenagers had the highest number of fatal and severe injury scooter and moped crashes, which is consistent with the observation that these vehicles are used principally by adolescents and young adults.[8,14] Because scooter and moped crashes occur mostly among teenage drivers, countermeasure efforts might be most effective if directed specifically toward this population.

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Call for Abstracts for PREVENTION 91

PREVENTION 91, the eighth annual national disease prevention and health promotion meeting sponsored by the American College of Preventive Medicine and the Association of Teachers of Preventive Medicine as well as numerous co-sponsors, will be held in Baltimore, Maryland March 16–19, 1991. The meeting theme, "Building an Economic Framework," will be the focus of four days of general sessions, concurrent sessions, poster sessions, workshops, and computer demonstrations.

Abstracts are sought for presentations at PREVENTION 91 which are directly linked to the meeting theme or to one of the topics already selected as the focus for the concurrent scientific sessions. Abstracts must be submitted on the standard abstract form, which may be obtained from: Hazel Keimowitz, PREVENTION 91, 1015 15th Street, NW, Suite 403, Washington, DC 20005. Tel: (202) 789-0006. Abstracts must be postmarked by October 1, 1990 to qualify for this year's consideration.

PREVENTION 91 will present up to six $100 awards to residents/students who submit and present the best abstracts describing their research. Selection criteria include rigorous scientific methodology, clarity, organization, originality, and relevance to the meeting theme.

City Directories as Sources for Survey Work in Low- and Middle-Income Black Communities

EDWARD J. STANEK III, PHD, HARRIS PASTIDES, PHD, WILLIAM A. DARITY, PHD, AND MARY ELKINS, MS

Abstract: Commercial directories and governmental lists of dwelling units in low income urban Black communities in four eastern cities were evaluated for completeness. With rare exceptions, less than 90 percent of dwelling units were included in any one list and no list adequately identified multiple dwelling unit structures. Since household income is likely to be lower among households in such structures, all lists tend to miss the very poor, i.e., those who may be at highest health risk. (*Am J Public Health* 1990; 80:1125–1126.)

Introduction

Community-based surveys are commonly used for needs assessment and program evaluation. The starting point for postal or personal interview surveys is a list of dwelling units (DUs) (or individuals) in the study community. There are usually several possible sources for such lists. An important factor in selecting a list is the degree to which a list covers the actual target population. The list selected is often the list with the largest number of entries.

Knowledge of coverage is important for interpretation of survey results. Certain commercial lists claim to cover, on average, over 95 percent of the DUs.[1] If coverage does not selectively underrepresent groups of individuals, such lists may be adequate. However, low coverage has been reported for DUs in multiple DU structures[1] and for areas that are hard to enumerate.[2] Socioeconomic status (SES) of such areas is likely to be low, and health risks of residents high. Lack of coverage of DUs in such areas may have an important impact on interpretation of results.

To evaluate the potential for bias due to inadequate coverage of low and middle SES communities, we evaluated coverage in lists from four communities: from Columbia, South Carolina, Durham, North Carolina, Hartford, Connecticut, and Springfield, Massachusetts. We discuss the implications of these results for frame construction and subsequent study interpretation.

Methods

Two commercial and two or three governmental lists (voter and property lists) were available in each community. A field list was also constructed by having study personnel canvass the areas on foot using an established protocol.[3] Coverage of each list was evaluated by comparison with a population frame. The population frame included DUs present in the original field list, along with DUs subsequently verified after combining lists.

Study communities were selected in each city using 1980 census data to include at least 300 DUs with a Black adult, low/middle income level, and non-adjacent to areas used in a larger study. Each community was defined on a census map, and boundaries confirmed by physical check. The communities and lists of DUs are given in the Appendix.

Verified computer lists for each source were edited for duplicates. All commercial and governmental lists were then combined to form a single list representing non-field listing sources with one record per DU. Special computer programs were written in SAS to accomplish the matching.[4] Further details concerning individual lists, and list processing are given elsewhere.[5]

Results

The communities in the four cities differed by number of residential structures, percent of structures that contained multiple DUs, and number of DUs. The number of residential structures contained in the final frames are 445, 273, 213, and 1205, in Columbia, Durham, Hartford, and Springfield, respectively. In Columbia and Springfield, a small portion of the residential structures contained more than one DU (0.9 percent and 5.4 percent), while a larger portion of residential structures were multiple DU structures in Durham (39.6 percent) and Hartford (69.5 percent). The total number of DUs identified per community ranged from 1,334 in Springfield to 396 in Durham. The percent of DUs identified as single DU structures ranged from 10.7 percent in Hartford to 84.2 percent in Columbia.

Results of the comparison of each list with the final frame in each community are summarized in Table 1. The original field list identified between 92.0–95.6 percent of the residential structures in all areas except Springfield. In each city (except Springfield) close to 90+ percent of residential structures were identified by at least one other list. However, 10 lists identified less than 80 percent of residential structures.

Coverage of DUs was lower. Two coverage rates for each list are shown in Table 1, where DUs have been grouped by single or multiple DU structure. In order for a DU to be correctly identified, the street number and apartment number/letter in a source must agree with that given in the final population frame. Field listings resulted in more than 80 percent coverage of DUs from single or multiple unit structures. Other lists covered at most 93 percent of single DUs, and 61 percent of multiple DUs.

Overall, DU coverage via the original field list was 95.2 percent (Columbia), 89.9 percent (Durham), 91.7 percent, (Hartford) and 84.7 percent (Springfield). In contrast, the only lists that identified more than 70 percent of the DUs

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TABLE 1—Percent Identification of Structures and Dwelling Units by City and Source

City	Source	Structures n	Structures (%)	DUs Identified by Structure Type: Single n	Single (%)	Multiple n	Multiple (%)
Columbia, SC	1st Field List	421	(95)	417	(95)	82	(99)
	Polk	410	(92)	408	(93)	51	(61)
	Tax List	390	(88)	387	(88)	2	(2)
	Tax Map	297	(67)	296	(67)	0	(0)
	Voter	280	(63)	277	(63)	17	(21)
	Cross Reference	248	(56)	245	(56)	2	(2)
Durham, NC	1st Field List	261	(96)	154	(93)	202	(88)
	Polk	245	(90)	144	(87)	139	(60)
	Parcel	245	(90)	149	(90)	10	(4)
	Property List	215	(79)	136	(82)	8	(4)
	Voter	200	(73)	114	(69)	42	(18)
	Cross Reference	175	(64)	108	(66)	19	(8)
Hartford, CT	1st Field List	197	(92)	55	(85)	503	(93)
	Tax Map	195	(92)	60	(92)	10	(2)
	Johnson	183	(86)	48	(74)	46	(9)
	Voter	170	(80)	38	(59)	11	(2)
	Hill-Donnelly	118	(55)	27	(42)	10	(2)
Springfield, MA	1st Field List	1009	(84)	946	(83)	184	(95)
	Voter	990	(82)	925	(81)	3	(2)
	Johnson	866	(72)	826	(73)	11	(6)
	Real Estate	826	(69)	805	(71)	3	(2)
	Hill-Donnelly	564	(47)	536	(47)	3	(2)

correctly were the Polk in Columbia (87.6 percent) and Durham (71.5 percent), and the Tax List in Columbia (74.2 percent). No single list enabled easy identification of multiple DU structures, so as to be useful for targeted list up-dating. The same set of DUs were missed by all lists.[5]

Discussion

The results of this study illustrate the limitations in coverage of existing lists of residential structures and dwelling units in low income urban communities. Low coverage has implications for estimation of community health parameters and interpretation and generalization of study results. As confirmed here, dwelling units not included in a sampling frame are not likely to be missing at random. Missed DUs are likely to be from multiple unit structures or those having no visible identifying number. Such households are likely to differ by income/education from DUs that are listed. If response parameters, including a wide variety of risk factors or exposures, differ by such characteristics, estimates of population parameters not adjusting for differential coverage will be biased. Greater efforts are needed to identify possible variables that differentiate DUs (and household members) included or excluded from sample frames.[6] A healthy skepticism is warranted for characterizations of populations that do not account for such possible biases.

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APPENDIX—Summary of Sources of Commercial and Governmental Lists of Dwelling Units from Four US Cities

City*	Source	List (last update)
Columbia, SC	R.L. Polk & Co.	Polk (87)
	City Publishing Co.	Cross-Reference (87)**
	Election Office#	Voter List (3/88)#
	Tax Office	Tax List (6/86)##
	Tax Office	Tax Map (12/87)##
Durham, NC	R.L. Polk & Co.	Polk (86)
	City Publishing Co.	Cross-Reference (87)**
	Election Office#	Voter List (5/87)#
	Real Estate Office	Parcel List (4/87)
	Real Estate Office	Property Tax Location Registry (1/88)##
Hartford, CT	Hill-Donnelly Co.**	Hill-Donnelly (87)**
	Johnson Publishing**	Johnson Publishing (4/87)**
	Election Office#	Voter List (4/88)#
	Tax Office##	Real Estate Tape (4/88)##
	Tax Office	Assessor's Maps (10/84)##
Springfield, MA	Hill-Donnelly Co.**	Hill-Donnelly (87)**
	Johnson Publishing**	Johnson Publishing (87)**
	Election Commission#	Eligible Voter List (10/87)#
	City Planning##	Real Estate Tape (12/87)##

*Columbia: Census Tract: 110 (Blks 102–107, 110–111, 115, 201–208)
Durham: Census Tract: 3.01.2 (Blks 204–211, 303–306)
Hartford: Census Tract: 5014 (Blks 102–103) & 5018 (Blks 206, 301–303)
Springfield: Census Tract: 8015.02 (Blks all)

**Excludes dwelling units without listed phone numbers

#Includes registered voters only except Springfield which mandates listing all adults age 18 and over.

##Usually does not include number of dwelling units in structure. Lists only structures.

AUTHOR NOTE: Under "Sources," the terms "Tax Office" and "Real Estate Office" refer to those government offices which do assessments for real estate tax purposes.

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Predictors of Vaccination Behavior among Persons Ages 65 Years and Older

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Abstract: We estimated influenza vaccination coverage of 32 percent among persons 65 years of age and older from the 1987 Behavioral Risk Factor Surveillance System survey. Race other than White, obesity, lack of seatbelt use, and current smoking were associated with decreased likelihood of having been vaccinated. Controlling for these factors, the best predictor of having received influenza vaccination was having had a medical checkup within the last year (Odds Ratio = 2.40, 95% confidence interval = 1.84, 3.14). (*Am J Public Health* 1990; 80:1127–1129.)

Introduction

Influenza annually accounts for substantial morbidity and mortality in the United States, especially in older persons. Influenza resulted in an average of approximately 20,000 excess deaths per year during six influenza epidemics from 1972 to 1981, primarily among persons ages 65 years and over.[1] Influenza vaccine has been shown to be approximately 75 percent effective in reducing deaths from influenza and its complications among high-risk older persons living in institutions.[2] Annual vaccination against influenza is recommended for all persons ≥ 65 years old.[3–5] Yet, despite the continuing morbidity and mortality caused by influenza among older adults, most do not receive annual immunization. The purpose of this study was to identify sociodemographic and health-related attributes that may be associated with influenza vaccine coverage among persons ages 65 years and older.

Methods

Data were analyzed from the 1987 Behavioral Risk Factor Surveillance System (BRFSS), a state-based system which monitors self-reported risk behaviors through monthly telephone surveys of adults.[6] Thirty-one states and the District of Columbia participated in the 1987 BRFSS. A sample of households was selected using a multi-stage cluster sampling design. Individual households within eligible clusters were selected for contact by random-digit dialing.[7] After contacting a household, a respondent was selected randomly from all adults residing in the household.[8] In addition to questions on demographic and lifestyle/behavioral characteristics, one question was asked regarding whether the respondent had received an influenza vaccination within the previous 12 months.

We assessed risk factors (i.e., demographic factors, health "risk-taking" behaviors, and preventive health practices) for differential influenza vaccination coverage levels using weighted data (to compensate for sources of variation in selection probabilities).[9] We calculated estimates of the relative likelihood of having received influenza vaccination among groups with specified characteristics using methods which take into account the complex sampling design.[10] In order to simultaneously control for all demographic and behavioral variables, we also used mutivariate logistic regression to discern whether each of the various demographic and behavioral factors were significantly associated with the likelihood of having received influenza vaccination.

Results

Interviews were completed for 9,851 respondents who were 65 years of age or older; 9,799 (over 99 percent) reported knowing whether or not they had received an influenza vaccination within the previous 12 months. Of these, 32 percent reported having been vaccinated against influenza.[11]

There were no important differences in vaccination coverage among persons 65 years of age or older based on gender, education level, or household income (Table 1). Within employment subgroups, retired persons had the highest vaccination coverage (33 percent vaccinated), followed by persons currently working outside their homes (30 percent), homemakers (27 percent), and those currently unemployed (19 percent); in general, persons employed outside their home were equally as likely to have received the vaccination as those without outside employment. Of the demographic factors, race was most strongly associated with having received influenza vaccination. Whites reported higher vaccination coverage levels (34 percent vaccinated) than did Blacks (22 percent) and Hispanics (16 percent); overall, Whites were significantly more likely than other races to have had influenza vaccination (Relative Risk (RR) = 1.63, 95% CI = 1.38, 1.93). This observation held within all household income strata.

When health "risk-taking" behaviors were examined, persons who were obese (RR = 0.86, 95% CI = 0.76, 0.97), persons who did not regularly use seatbelts (RR = 0.89, 95% CI = 0.80, 0.99), and current smokers (RR = 0.80, 95% CI = 0.68, 0.93) were less likely to have received the influenza vaccine (Table 1). There were no differences in vaccination coverage among older persons who were users of smokeless tobacco products, at risk for acute or chronic alcohol consumption problems (including self-reported drinking and driving), or who had a sedentary lifestyle.

The general characteristics that were most strongly associated with whether persons over 64 years old received an influenza vaccination during the preceding year were those related to knowledge and utilization of other health care services (Table 1). Persons who had had a medical examination within the preceding year were twice as likely to have received influenza vaccination than persons who had not had a checkup within the preceding year. Persons who had ever

Address reprint requests to Technical Information Services, Center for Prevention Services, Centers For Disease Control, 1600 Clifton Road (MS-E06), Atlanta, GA 30333. Drs. Stehr-Green, Sprauer, and Williams are with the Division of Immunization, Center for Prevention Services at CDC; Mr. Sullivan is with the Division of Nutrition, Center for Chronic Disease Prevention and Health Promotion, CDC. This paper, submitted to the Journal October 30, 1989, was revised and accepted for publication January 18, 1990.

TABLE 1—Relative Risks of Receipt of Influenza Vaccine by Demographic and Behavioral Risk Factors among Persons 65 Years of Age and Older[a]—Behavioral Risk Factor Surveillance System, 1987

Risk Factors	% with Risk Factor	% Vaccinated in Risk Group[b]	Relative Risk[c]	95% CI[d]
Demographic Characteristics				
Male sex	33.6	34.1	1.09	(0.98, 1.22)
White race	89.2	33.8	1.63	(1.38, 1.93)
High school graduate	56.5	32.1	0.98	(0.88, 1.09)
College graduate	12.6	36.3	1.14	(0.98, 1.33)
Low income (< $10,000/yr)	37.5	31.9	0.98	(0.87, 1.10)
Employed outside home	9.1	29.8	0.91	(0.75, 1.11)
Health Risk-taking Behaviors				
Obesity	28.8	29.4	0.86	(0.76, 0.97)
Lack of seatbelt use	36.8	29.6	0.89	(0.80, 0.99)
Current smoking	13.3	26.5	0.80	(0.68, 0.93)
Use of smokeless tobacco	3.6	31.2	0.96	(0.72, 1.28)
Acute drinking problem	2.1	39.3	1.22	(0.83, 1.78)
Chronic drinking problem	2.7	25.4	0.78	(0.56, 1.08)
Drinking and driving	0.2	17.5	0.54	(0.25, 1.15)
Sedentary lifestyle	67.5	31.9	0.96	(0.86, 1.08)
Utilization of Health Care				
Medical exam in last year	80.0	35.9	2.00	(1.73, 2.31)
Ever told had hypertension	45.6	30.0	1.18	(1.06, 1.31)
(Women) Know of mammography	85.6	32.1	1.26	(1.04, 1.53)
Ever had mammogram	44.7	36.7	1.31	(1.14, 1.50)
Mammogram in last year	60.2	40.3	1.27	(1.03, 1.55)

[a]Total sample size was 9,799 persons; however, due to incomplete responses for some questions, the effective sample size for individual analysis varied.
[b]Data for each respondent were weighted by a factor that was inversely related to that respondent's probability of selection in order to compensate for sources of variation in selection probabilities.
[c]Ratio of the proportion of persons with the specified demographic or behavioral characteristic who received an influenza vaccination within the last year to the proportion of persons without the specified characteristic who received the vaccination.
[d]95% confidence intervals taking into account the complex sample design.

been told by a health professional that they had high blood pressure were somewhat more likely to have received influenza vaccination; however, among these persons there was no increased likelihood of vaccination in those currently taking antihypertensive medications nor among those who reportedly were still hypertensive. Among women, knowledge and utilization of mammography for breast cancer screening was associated with a greater likelihood of receiving influenza vaccination.

When influences of all demographic and behavioral factors were examined simultaneously, those that were previously shown not to be associated were again not correlated with receipt of influenza vaccination. Estimates of relative risk for vaccination associated with race, obesity, lack of seatbelt use, current smoking, or having been diagnosed with hypertension were essentially unchanged. Having had a medical checkup within the last year remained most strongly associated with having received influenza vaccination (Odds Ratio = 2.40, 95% CI = 1.84, 3.15).

Discussion

Our ability to generalize from these estimates is somewhat limited due to non-random selection of states, possible bias due to differences in immunization levels among persons not included in the survey, inability to verify self-reports of immunization status, and the likelihood that there are other factors affecting receipt of influenza vaccination that were not included in these data. However, these results are consistent with previous studies[12, 13] that show utilization of health care services as a strong predictor of whether or not an individual receives influenza vaccination.

Because a recommendation by a health care provider appears to be one of the most important factors influencing a person's decision to be vaccinated,[13] greater attention among providers to implementing recommended immunization practices could help improve influenza vaccine coverage. Annual influenza immunization should be incorporated into their practices; all other vaccines appropriate for adults (pneumococcal, hepatitis B, measles, mumps, and rubella vaccines, and diphtheria and tetanus toxoids) should be offered at every opportunity.[3–5] Barriers (whether sociocultural, physical, or financial) to health care for older persons should be minimized to provide optimal access to preventive, as well as curative, services. In addition, health education efforts to promote healthier lifestyles (e.g., stopping smoking, routinely using seatbelts, and following appropriate exercise programs) should be expanded to include adult immunization as part of overall health maintenance for persons over 64 years of age.

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HIV Seroprevalence and the Acceptance of Voluntary HIV Testing among Newly Incarcerated Male Prison Inmates in Wisconsin

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Abstract: In 1986–88, voluntary and blinded HIV testing was conducted among Wisconsin male prison entrants. The HIV seroprevalence was 0.30 percent in 1986, 0.53 percent in 1987, and 0.56 percent in 1988. The seroprevalence rates among entrants tested voluntarily did not differ from those tested blindly. Voluntary HIV testing was accepted by 71 percent of male prison entrants in 1988; among entrants reporting intravenous drug use 83 percent consented to voluntary HIV testing. Voluntary HIV testing of entrants appears to be an effective screening strategy in Wisconsin prisons. (*Am J Public Health* 1990; 80:1129–1131.)

Introduction

Because intravenous drug use (IVDU) is associated with both criminal activity and human immunodeficiency virus (HIV) infection, many persons sentenced to correctional facilities are at high risk of HIV infection. While most strategies aimed at minimizing HIV transmission in prisons include provisions for HIV testing, there is a divergence of opinion as to whether testing should be voluntary or mandatory. The World Health Organization has recommended against mandatory testing of prisoners.[1] The American Medical Association, however, has stated that mandatory testing is necessary to protect inmates.[2] Currently, 14 states and the Federal Prison System have mandatory testing policies.[3] In the Wisconsin Correctional System HIV testing is voluntary. We conducted HIV seroprevalence surveys among newly incarcerated male inmates in Wisconsin in 1986, 1987, and 1988 to provide HIV seroprevalence estimates and to evaluate the acceptance of voluntary HIV testing in Wisconsin prisons.

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Methods

In Wisconsin, male inmates are admitted to a central intake facility where they receive medical examinations and provide blood specimens for hepatitis B serologic testing; residual sera are retained in a serum bank. While at the intake facility, all inmates view a HIV-related educational videotape, are assessed individually by a nurse for HIV-related risk factors, and are given the opportunity to provide written consent to voluntary HIV testing with post-test counseling.

In 1986, a random sample of banked serum specimens obtained from inmates incarcerated between January 1 and September 29 of that year was selected, all identifying information was removed from these specimens (specimen blinding), and they were then tested for antibody to HIV. In 1987 and 1988, blinded seroprevalence surveys were conducted among inmates incarcerated between January 1 and August 31 of each year who declined voluntary tests. Results of blinded testing were combined with results of voluntary testing performed during the same periods.

Beginning in March 1988, newly admitted inmates were asked to complete voluntary self-administered HIV risk assessment questionnaires as part of the routine intake procedure. Data on inmate age, race, and place of sentencing were obtained from prison records. The blinded study design was approved by the Human Subjects Committee of the University of Wisconsin-Madison.

Specimens were tested at the Wisconsin State Laboratory of Hygiene for the presence of antibody to HIV by enzyme linked immunosorbent assay (Abbott). Repeatedly reactive specimens were tested by Western blot assay. Specimens with at least two viral protein bands (from among p24, gp41, or gp120/gp160) demonstrated by Western blot assay were considered HIV seropositive.

Data were analyzed using *Epi Info* software (Centers for Disease Control, Atlanta, Georgia). The Yates Corrected Chi Square Text (χ^2) was used. Confidence intervals (CI) for odds ratios (OR) were calculated by either the exact or Cornfield method. Mantel-Haenszel weighted odds ratios were used in the age-adjusted analysis.[4]

Results

In 1986, a 48 percent random sample of sera obtained from inmates admitted during the survey period was blindly tested for antibodies to HIV (Table 1). In 1987, 40 percent of

TABLE 1—Results of Voluntary and Blinded HIV Seroprevalence Surveys Conducted among Newly Incarcerated Male Prison Inmates in Wisconsin, 1986–1988

	Year Surveyed		
	1986*	1987	1988
Inmates**	2,087	1,783	1,675
Total sample			
Tested	997	1,689	1,621
HIV+	3	9	9
% HIV+ (95% CI)	0.30 (0, 0.64)	0.53 (0.18, 0.88)	0.56 (0.20, 0.92)
Voluntary sample			
Tested	—	708	1,190
HIV+	—	6	7
% HIV+ (95% CI)	—	0.85 (0.17, 1.53)	0.59 (0.15, 1.03)
Blinded sample			
Tested	997	981	431
HIV+	3	3	2
% HIV+ (95% CI)	0.30 (0, 0.64)	0.31 (0, 0.66)	0.46 (0, 1.10)

*The 1986 survey did not include a voluntary component.
**Adult male inmates newly incarcerated during the study periods.

inmates admitted during the survey period were tested voluntarily; banked specimens with sufficient quantity of sera were available for blinded testing of 92 percent of those declining voluntary HIV testing. In 1988, comparable figures were 71 and 89 percent.

The 1986 HIV seroprevalence does not differ from that observed in 1987 (OR = 1.8, 95% CI = 0.4, 10.2) or in 1988 (OR = 1.8, 95% CI = 0.5, 10.4) (Table 1). The HIV seroprevalence among those tested voluntarily exceeded but did not differ from the HIV seroprevalence among nonvolunteers in 1987 (OR = 2.8, 95% CI = 0.6, 17.3) and 1988 (OR = 1.3, 95% CI = 0.2, 12.6). Combining 1987 and 1988 data, 72 percent of HIV seropositive inmates were detected by voluntary testing.

The rate of voluntary HIV testing has exceeded 60 percent since the third quarter of 1987, reaching 71 percent in 1988. The consent rate for voluntary testing decreased with increasing age ranging from 78 percent of those less than 20 years old to 56 percent among inmates 40 or more years of age (χ^2_{trend}, $p < 0.001$). Rates of voluntary consent were not significantly associated either with race or place of sentencing.

In 1988, 989 (78 percent) of those offered the voluntary risk assessment responded; among respondents 246 (25 percent) reported IVDU. Among respondents reporting IVDU, six (2.4 percent) of 245 tested for antibody to HIV were seropositive compared to none (zero percent) among 741 not reporting IVDU. Among all respondents, 76 percent consented to voluntary HIV testing. Among respondents who reported IVDU, 83 percent (203 of 246) consented to voluntary HIV testing compared to 74 percent (550 of 743) not reporting this behavior (age adjusted OR = 1.8, 95% CI = 1.2, 2.6).

Discussion

Nationally, HIV seroprevalence among prison inmates has ranged from 0–17.4 percent.[5–7] In Wisconsin the rate is relatively low and has remained stable over a three-year period. In our sample, HIV infection was strongly associated with IVDU and the comparably low HIV seroprevalence we observed is a consequence of the relatively low HIV seroprevalence among intravenous drug users sentenced to Wisconsin prisons. Our estimate (2.4 percent) of the HIV seroprevalence among male prison entrants reporting IVDU closely concurs with a 1987 estimate (2.3 percent) of the HIV seroprevalence among intravenous drug users enrolled in drug treatment in Milwaukee.[8]

Voluntary HIV testing in prisons would be ineffective if HIV-infected inmates or those at risk of infection, especially intravenous drug users, systematically self-deferred from voluntary testing. This has occurred in non-prison settings.[9] However, in our study, the rate of voluntary HIV testing among inmates reporting IVDU exceeded that of inmates denying this behavior. Moreover, the results of the 1987 and 1988 surveys indicate that HIV seropositive inmates did not systematically decline voluntary testing. Similar results have been reported among prison inmates in Oregon.[10]

Because of the risk of HIV transmission in prison populations, prevention strategies are needed. We believe that voluntary behavior change, facilitated by effective risk reduction counseling, is the most desirable method of reducing HIV transmission. If voluntary behavior change is a goal, well-accepted voluntary testing may be advantageous to mandatory testing because it enables inmates and counselors to establish a relationship based upon consent, thus promoting the acceptance of risk reduction counseling.

In Wisconsin prisons, voluntary HIV testing and counseling is an effective method of screening inmates for HIV infection. Because the results of this study may not be generalizable to areas of high HIV seroprevalence, each correctional jurisdiction that offers voluntary HIV counseling and testing should evaluate their programs to assure that these services are reaching high-risk inmates.

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Sixth International Conference on Hypertension in Blacks to Meet in Brazil

The Sixth International Conference on Hypertension in Blacks will be held in Salvador, Bahia, Brazil, August 1–5, 1991, and will feature new research papers and state-of-the-art lectures from a pool of 33 countries throughout the world. Conference highlights include:

- Current concepts of ethnicity.
- Blood pressure distributions in racially mixed populations.
- The physiology of stress and hypertension.
- Advances in pharmaceutical agents acting on the renin system.
- Advances in molecular genetics of hypertension.
- Socioeconomic status, ethnicity and hypertension.
- Culture, diet and cardiovascular disease.
- Hypertension: The interplay of genetics and environment.
- The ongoing process of miscegenation.
- Primary health care and hypertension.

The conference will also feature lectures on Brazilian history and culture, since Brazil has the largest population of persons of African ancestry outside of Africa. Topics include:

- Religious practices in Bahia.
- The African roots of Brazilian art.
- Jorge Amado and modern Brazilian literature.
- Slave rebellions in Brazil.
- History and politics of Brazil today.
- Women in Brazilian society.
- The future of Brazilian society: Challenges and perspectives.

Abstracts must be postmarked by February 1, 1991, and submitted on the standard abstract form, available from: International Society on Hypertension in Blacks, 69 Butler Street, SE, Atlanta, GA 30303. Tel: (404) 589-3810.

New from nchs

Use of Contraception and Family Planning Services in the United States, 1988

WILLIAM D. MOSHER, PHD, AND WILLIAM F. PRATT, PHD

Use of Contraception

The leading contraceptive method overall in both 1982 and 1988 was the pill, but female sterilization was approaching the pill in popularity by 1988: 31 percent of contracepting women were using the pill and 28 percent were using sterilization in 1988.[1] If male and female sterilization are considered together as one method, however, then sterilization was the leading method, used by 39 percent of contracepting couples. Among currently married and formerly married women, female sterilization alone was the leading method. Among never married women, the pill was the leading method by far (Table 1). These findings are based on the National Survey of Family Growth (NSFG), a national sample of women in the United States 15–44 years of age. Personal interviews were conducted in the homes of 8,450 women in 1988, 7,969 women in 1982, and 9,797 women in 1973.

The oral contraceptive pill is the leading nonsurgical method of contraception, and the most effective (except for sterilization) in preventing pregnancy.[2] But it does not prevent the spread of sexually transmitted diseases. The condom is less effective at preventing pregnancy than the pill because it is often not used correctly or consistently,[2] but it is useful for preventing the spread of sexually transmitted diseases. Complete estimates of condom use should therefore include women who rely on their partners to use condoms to prevent sexually transmitted diseases,even if they do not use them to prevent pregnancy. This is a more complex methodological problem which is under study, but not covered in the new reports.

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Condom Use

Among never married women using contraception, the proportion who relied on their partner's use of a condom increased from 12 to 20 percent between 1982 and 1988 (Table 1). The condom, however, is sometimes used in combination with other methods—often, the pill. Women who use the pill, sterilization, or the IUD (intrauterine device) along with the condom are not classified in the statistics cited above, and in Table 1, as using the condom, but as using the other method, because the other is more effective in *preventing pregnancy*.[2] If we count all women who reported that their partners use condoms for birth control in 1988, even if they are using some other birth control method (such as the pill), we get an estimate of about 5.8 million women ages 15–44 whose partners were currently using the condom[1]—about 10 percent of the 58 million women of reproductive age.

The IUD and Female Sterilization

In 1985 and 1986, the IUD was withdrawn from the US market by two of its major manufacturers.[3] As a result, use of the IUD dropped by two-thirds between 1982 and 1988, from 2.2 million women in 1982 to 0.7 million in 1988.

TABLE 1—Number of Women 15–44 Years of Age, Percent and Number Using Contraception, and Percent Distribution by Method Used, According to Marital Status: US, 1982 and 1988

	Total		Never married		Currently married		Formerly married	
	1982	1988	1982	1988	1982	1988	1982	1988
Number of women (thousands)	54,099	57,900	19,164	21,058	28,231	29,147	6,704	7,695
Percent using contraception	55.7	60.3	35.3	41.9	69.7	74.3	55.5	57.6
Number using contraception (thousands)	30,133	34,914	6,765	8,823	19,677	21,656	3,721	4,432
All contraceptors	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Female sterilization	23.2	27.5	3.7	6.4	26.9	31.4	39.2	50.7
Male sterilization	10.9	11.7	1.8	1.8	15.5	17.3	3.4	3.6
Pill	28.0	30.7	53.0	59.0	19.3	20.4	28.4	25.3
IUD	7.1	2.0	5.4	1.3	6.9	2.0	11.5	3.6
Diaphragm	8.1	5.7	13.4	4.9	6.5	6.2	6.7	5.3
Condom	12.0	14.6	11.6	19.6	14.1	14.3	1.5	5.9
Other	10.7	7.7	11.1	7.0	10.8	8.4	9.2	5.7

SOURCE: Reference 1.

Groups that had relatively high proportions using the IUD in 1982 tended to increase their use of female sterilization by 1988. For example, the percent of formerly married contraceptive users who were using the IUD fell from 12 percent in 1982 to 4 percent in 1988, and the percent using female sterilization rose from 39 percent to 51 percent. The new report and a forthcoming analysis show a sharp increase in the percent of contraceptive users who chose female sterilization among several groups that had higher than average proportions using the IUD in 1982: Black, formerly married, less educated, low income, and Hispanic women.[1,4]

Why has female sterilization become the leading method among married and formerly married women? Previous studies of NSFG data have shown that by age 30, three-forths of ever married women have had all the births they want; a typical woman getting a tubal operation was about age 30 and had two or three births. This means that she had 10 or 15 years remaining when she might be able, but did not want, to conceive. About five out of six women in their late 20s have used the pill, which probably means that they want contraception that is as effective and as unobtrusive as the pill. Now that the IUD is no longer widely available, there are only two methods as effective as the pill: female sterilization and male sterilization. The medical techniques of female sterilization have become markedly easier and safer in the past 20 years, so that sterilization often does not require an overnight hospital stay. Also, insurance coverage of sterilization may have become more common in recent years.[4,5]

The Pill

The pill was introduced in the United States 30 years ago, in 1960. Since then, it has been used by the vast majority of women of reproductive age—40.3 million women ages 15–44 in 1988 have used the pill at some time in their lives; 10.7 million of them were using it in 1988. Among women under age 25, the pill was by far the leading method in both 1982 and 1988. The sharp decline in pill use that occurred among married women between 1973 and 1982 did not continue between 1982 and 1988.[1]

Use of Family Planning Services

Sterilization, the pill, and the diaphragm account for 76 percent of current contraceptive use. All of these methods require medical visits to obtain them initially; use of the pill requires regular visits to renew the prescription and check for side effects. Previous research has shown that pill users have a particularly high rate of use of family planning services, even after their other characterizations are taken into account.[6] In addition, young women often make family planning visits to obtain pregnancy tests or to learn about birth control methods.[7] For these reasons and others, it is important to determine how many women use family planning services, and where they obtain those services.

About 20 million women (35 percent of the 57.9 million women ages 15–44) made one or more visits for family planning services in the 12 months before the survey.[8] This was about the same number as in the 12 months before the 1982 survey. Of the 20 million women, about 13 million obtained these services from a private physician, private group practice, or health maintenance organization (HMO); about 7 million used a clinic. Of the 7 million clinic users, about half, or 3.74 million, used a Title X clinic. Black women, teenagers, and low-income were much more likely to use clinics than other women. Women were also more likely to use clinics for their first visit than for their most recent visit: 58 percent used a clinic at their first visit, while only 36 percent used a clinic at their most recent visit. The age group most likely to have had a family planning visit in the last year was women 20–24 years of age—the age group in which use of the oral contraceptive pill is most common.[4,8]

These are only some of the first findings from the 1988 National Survey of Family Growth. Other reports are planned on such topics as contraception, infertility, marriage and cohabitation, and wanted and unwanted births. In addition, a public use computer tape of the 1988 data has been released, so that interested researchers can explore these and other topics further.

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Copies of *Contraceptive Use in the United States, 1973–88*, Advance Data from Vital and Health Statistics No. 182, and *Use of Family Planning Services in the United States: 1982 and 1988*, Advance Data from Vital and Health Statistics No. 184 can be obtained free of charge from the Scientific and Technical Information Branch, Room 1064, NCHS, 6525 Belcrest Road, Hyattsville, MD 20782.

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Consistent Pattern of Elevated Symptoms in Air-conditioned Office Buildings: A Reanalysis of Epidemiologic Studies

MARK J. MENDELL, MPH, AND ALLAN H. SMITH, MD, PHD

Abstract: Published studies of the relation between type of building ventilation system and work-related symptom prevalence in office workers have been contradictory. A reanalysis was performed of six studies meeting specific eligibility criteria, combining published data with unpublished information obtained from study authors. Five eligible studies were from the United Kingdom, and one was from Denmark. Standardized categories of building ventilation type were created to allow comparison of effects across studies. Within each study, prevalence odds ratios (PORs) were calculated for symptoms in each ventilation category relative to a baseline category of naturally ventilated buildings. Air-conditioned buildings were consistently associated with increased prevalence of work-related headache (POR = 1.3-3.1), lethargy (POR = 1.4-5.1), and upper respiratory/mucus membrane symptoms (POR = 1.3-4.8). Humidification was not a necessary factor for the higher symptom prevalence associated with air-conditioning. Mechanical ventilation without air-conditioning was not associated with higher symptom prevalence. The consistent associations found between type of building ventilation and reported symptom prevalence have potentially important public health and economic implications. (*Am J Public Health* 1990; 80:1193–1199.)

Introduction

Health problems and non-specific symptom complaints apparently related to buildings or indoor air, sometimes referred to as "sick building syndrome" or "tight building syndrome," have been recognized for over 15 years[1]. Although particular chemical,[2] biological,[3] physical,[4] or psychological[5] factors have been implicated in some episodes, specific causes have generally not been identified.[6] Studies of this problem to date, generally carried out in buildings identified by worker complaints (referred to herein as "complaint" buildings) have demonstrated mostly what these episodes are *not*. They are not caused by known toxins at concentrations exceeding current health standards nor are they generally associated with known diseases. We still have little idea how frequently the excess symptoms reported in such episodes actually occur among office workers, or what if any chronic health problems in this population are related to office buildings.

It is difficult to study a phenomenon characterized only by self-reported non-specific symptoms with no accepted syndrome definition or objective tests available. A major weakness of investigations in buildings with widely recognized worker complaints is that occupant concerns are likely to upwardly bias symptom reporting, thus distorting the only outcome available for study. Until the development of useful objective tests, it will therefore be preferable to study buildings without recognized worker complaints (referred to herein as "non-complaint" buildings).

Reports on building-related health problems in the United States are almost without exception case studies of complaint buildings;[2-4,7-10] only one study compared the building under investigation to even a single noncomplaint building.[11] A number of recent European studies, however, have provided data from non-complaint buildings on relations between work-related symptoms in office workers and type of building ventilation.[12-16] Preliminary review of the European studies shows that mechanical ventilation, relative to natural ventilation, has been associated with increases,[12,16] with decreases,[17] and with no differences[22] in work-related symptoms; air-conditioning, has been associated with increases[17] and with no differences[16] in symptoms. Previous reviews of the literature on illness episodes in office buildings[6,27-30] have summarized available studies but have not discussed these discrepancies.

This paper presents a reanalysis of data from non-complaint building studies. The purpose was to determine if there were consistent relations between prevalence of specific symptoms and certain building ventilation factors: mechanical ventilation, air conditioning, and humidification. Although differences between the original studies did not allow direct comparison of results, additional information obtained from study authors allowed creation of a standardized set of ventilation categories, calculation of prevalence odds ratios, and comparison of findings across studies.

Methods

Selection of Studies for Reanalysis

Studies of work-related symptoms in office workers were selected from the literature, using specific eligibility criteria. For inclusion in this reanalysis it was required that:

- studies compared multiple non-complaint office buildings;
- data allowed comparison of prevalence for specific work-related symptoms between buildings of different ventilation type; and
- data were available allowing classification of buildings as naturally or mechanically ventilated, as air-conditioned or not air-conditioned, and as humidified or not humidified.

Six studies of worker symptoms in multiple office buildings[12-26] were included in this reanalysis. The studies included were all cross-sectional and compared work-related symptom prevalence in buildings with different ventilation types to a baseline symptom prevalence in naturally ventilated buildings. All studies tested for statistical independence of ventilation categories and symptom prevalence (using chi square or analysis of variance tests) but used no epidemio-

From the Department of Biomedical and Environmental Health Sciences, University of California, Berkeley (both authors) and the California Department of Health Services (Mendell). Address reprint requests to Mark J. Mendell, MPH, Environmental Epidemiology and Toxicology Section, California Department of Health Services, 5900 Hollis Street, Suite E, Emeryville, CA 94608. This paper, submitted to the Journal November 3, 1989, was revised and accepted for publication April 9, 1990.

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logic effect measures (such as prevalence ratios or odds ratios) to assess this relationship. Five studies were conducted in the United Kingdom, and one in Denmark. (Findings of the six studies are summarized in the Appendix.)

Methods of Analysis

Five standard categories of building ventilation type were established, as described in Table 1. Information for this classification was extracted from published materials; where necessary, additional information was obtained directly from the authors, and subsequent classification of buildings was confirmed with them.* Ultimately, sufficient information was available for unequivocal classification of all buildings from the six studies into this common set of categories. Correct conversion of ventilation categories in the original studies to the ventilation categories in this reanalysis was not always straightforward; in such cases, the appropriate conversions are provided in footnotes to Table 2, along with publications from which the data were obtained.

Symptoms were organized into four groups:

- lower respiratory (tight chest, difficulty breathing, shortness of breath, wheeze, and flu-like symptoms);
- upper respiratory/mucus membrane (nose, throat, and eye symptoms);
- central nervous system (headache and lethargy); and
- skin (dry skin, itching skin, and skin rash).

From sample size and prevalence data reported for either individual buildings or groups of buildings in each study, numbers of subjects with and without each specific work-related symptom were calculated within each category of building ventilation type. Prevalence rates were then calculated. "Work-related symptoms" in all studies referred only to symptoms reported as improving on weekends or days away from work (and in study 1 and parts of study 2, also as having begun or worsened since starting work in the current building). Symptom frequency requirements varied between studies.

Baseline symptom prevalence varied widely between studies. Using the naturally ventilated building category as the reference group within each study, prevalence odds ratios and 95% test-based confidence limits[31] were calculated from prevalence rates for each work-related symptom within each of the other ventilation categories. The prevalence odds ratio was chosen as the most informative epidemiologic effect measure for cross-sectional prevalence data.[32,33]

*Personal communications: M. Finnegan, S. Burge, A. Pickering, A. Robertson, J. Harrison, O. Valbjorn.

Results

Table 2 gives the sample sizes for each study reviewed, in terms of number of workers and number of buildings in each ventilation category.

Table 3 gives prevalence data for specific work-related symptoms, as calculated for each study using the new building ventilation categories.

Table 4 gives prevalence odds ratios (PORs) for each work-related symptom in each ventilation category. Symptom prevalence in category II buildings (with simple mechanical ventilation) was comparable to that in category I buildings or lower, with one exception (headache prevalence in study 4, based on only one building in category II). This was in sharp contrast to the general pattern of higher symptom prevalence in air-conditioned buildings (categories III, IV, and V) found in all six studies.

The prevalence of central nervous system and upper respiratory/mucus membrane symptoms was almost without exception higher in the air-conditioned buildings than in the naturally ventilated buildings. Data on lower respiratory problems were relatively sparse and less consistent. Data on skin-related symptoms suggested higher prevalence in air-conditioned buildings.

Data on differences between air-conditioned buildings with different kinds of humidification were mixed. In studies 1 and 3, where these data were available, symptom prevalence in buildings with steam humidification was similar to that in those with no humidification. There was some suggestion in both studies of higher symptom prevalence in buildings with water-based humidification relative to both categories III and IV. For 18 of the 21 symptoms assessed between these two studies, point estimates in category V equaled or exceeded those in both categories III and IV.

In study 2, on the other hand, symptom prevalence in air-conditioned buildings with water-based humidification was generally comparable to or lower than that in air-conditioned buildings without humidification. More complete data from this study, however, were presented at a scientific meeting. They showed consistently *lower* symptom prevalence in air-conditioned buildings with water-based humidification than in those without humidification; symptom prev-

TABLE 1—Building Ventilation Categories for Comparison Across Studies

	Building Ventilation Categories				
	I	II	III	IV	V
Ventilation Characteristics	Natural*	Simple Mechanical**	Air-Conditioned No Humidification	Air-Conditioned Steam Humidification	Air-Conditioned Water-Based Humidification
Mechanical ventilation	No	Yes	Yes	Yes	Yes
Air-conditioning	No	No	Yes	Yes	Yes
Steam humidification	No	No	No	Yes	No
Drip, spray, or evaporative humidification	No	No	No	No	Yes

*operable windows only
**ducted airflow without cooling or chilling

TABLE 2—Study Sample Sizes by Building Ventilation Category

Study	Sampling Unit (Total N)	Building Ventilation Categories I Natural* (N)	II Simple Mechanical** (N)	III Air-Conditioned No Humidification (N)	IV Air-Conditioned Steam Humidification (N)	V Air-Conditioned Water-Based Humidification (N)
1*	Workers (951)	259	—	73	91	528
	Buildings (8)	3	—	1	1	3
2**	Workers (2587)	537	—	507	—	1543
	Buildings (27)	8	—	6	—	13
3***	Workers (4373)	442	944	1017	421	1549
	Buildings (47)	11	7	10	4	15
4†	Workers (1332)	206	118	—	—	1008
	Buildings (5)	2	1	—	—	2
5††	Workers (106)	47	—	—	—	59
	Buildings (2)	1	—	—	—	1
6†††	Workers (2778)	1166	1430	—	—	182
	Buildings (14)	6	7	—	—	1

*Source: Reference 15 was used, as it included an additional, steam-humidified, building not included in the initial report, reference 12, and it omitted two buildings included in the initial report specifically because of worker complaints. Category III buildings were described as having "mechanical ventilation."

**Source: Reference 16. Category III buildings were described as having "mechanical ventilation only." Category V buildings were described as "fully air-conditioned, with or without recirculation," or "humidified."

***Source: Reference 17 was used for data in categories I and II, but reference 19 was used for categories III-V, as it was the only report to include data from air-conditioned buildings by type of humidification, for all symptoms.

†Source: Reference 20. Category I buildings were described as "conventional" and category II as "unconditioned."

††Source: Reference 21.

†††Source: Reference 22. Data used were from the 14 town halls; buildings number 10, 20, 30, 40, 50, and 90 were in category I, number 121 was in category V, and the rest were in category II.

alence was still substantially higher than in naturally ventilated buildings.**

Buildings without natural ventilation may be sealed (i.e., have non-operable windows) or not. Although sufficient information was not available to allow incorporation of this factor into ventilation categories used for reanalysis, enough information was available to show a very strong relation between the presence of air-conditioning and presence of sealed windows in the buildings studied: only one of the 48 non-air-conditioned buildings had sealed windows, whereas 52 of the 57 air-conditioned buildings had sealed windows. Thus, given the limits of these data, symptom increases found to be associated with air-conditioned buildings may more appropriately be regarded as associated with air-conditioned, sealed buildings.

Discussion

This reanalysis suggests that sealed buildings with air-conditioning are associated with higher prevalence of work-related headache, lethargy, and eye, nose, and throat symptoms than unsealed buildings with no air-conditioning, even in the absence of humidification. It also suggests that although air-conditioned buildings with steam humidification are associated with symptom prevalence no higher than air-conditioned buildings without humidification, air-conditioned buildings with water-based humidification *may* be associated with higher prevalence of eye, nose, and throat symptoms than those with steam humidification.

Apparent discrepancies between the original reports were resolved by reclassification based on new information from authors of the studies. Buildings described as mechanically ventilated were actually air-conditioned without humidification in studies 1 and 2, and simply mechanically ventilated in study 3; in study 6, they were simply mechanically ventilated or, in one case, air-conditioned with water-based humidification.

The prevalence of some work-related symptoms was strikingly high in several studies in even the least problematic office buildings, suggesting a measurement problem. High prevalence of lethargy in category I buildings in studies 3 and 5 (50 percent and 62 percent) in fact reflects a very broad definition of "work-related" symptoms in these two studies, the least restrictive such definition in all studies reviewed (see Appendix). (That such high prevalence resulted from specifics of the study protocol is evident from the fact that study 5 remeasured buildings from study 1 with the protocol from study 3, and obtained prevalence estimates two to four times those in study 1, comparable to those in study 3.)[21] Symptom prevalences reported in study 6, also somewhat high, represented combinations of two to three specific symptoms assessed.[10] Such differences emphasize the need for standardized questionnaire design and data reporting.

One must be cautious in generalizing results of this reanalysis. The studies reviewed were all carried out in the United Kingdom or Denmark, and other countries may have differences in climate as well as in building and ventilation system design or operation.

Also, limitations in the data restrict conclusions that can be drawn from this reanalysis, due to a variety of possible biases in each study.

Bias from selection of buildings seems unlikely, as buildings in all studies were selected independently of worker complaints. Response bias among workers also seems unlikely, as response rates were high in all studies (see Appendix). Some selection bias due to cross-sectional study design

**J. Harrison, presented at Indoor Air Quality 89, San Diego, CA, April 17–20, 1989.

TABLE 3—Prevalence of Reported Work-related Symptoms among Office Workers by Building Ventilation Category

		Building Ventilation Categories				
		I	II	III	IV	V
		Natural	Simple Mechanical	Air-Conditioned No Humidification	Air-Conditioned Steam Humidification	Air-Conditioned Water-Based Humidification
Study	Symptoms	(%)	(%)	(%)	(%)	(%)
	Central Nervous System					
1	headache	15.7	—	37.0	29.7	32.0
	Lethargy	13.8	—	45.2	29.7	41.3
2	Headache	20.7	—	39.3	—	35.6
	Lethargy	19.1	—	49.9	—	43.3
3*	Headache	39	33	45	48	47
	Lethargy	50	42	58	60	65
4*	Headache	20	36	—	—	40
5*	Headache	32	—	—	—	66
	Lethargy	62	—	—	—	86
6*	Headache, fatigue, or malaise	33	35	—	—	53
	Upper Respiratory/Mucus Membrane					
1	Nose symptoms	5.8	—	13.7	15.4	19.1
	Dry throat/blocked nose	8.1	—	17.8	16.5	29.9
	Eye symptoms	5.8	—	8.2	13.2	15.9
2	Nose symptoms	13.4	—	30.7	—	22.4
	Throat symptoms	15.5	—	31.6	—	30.8
	Eye symptoms	11.0	—	19.3	—	23.1
3*	Runny nose	19	16	24	25	26
	Blocked nose	40	32	48	48	55
	Dry throat	36	33	49	47	53
	Dry eyes	18	20	31	28	33
	Itching eyes	22	20	31	27	32
4*	Sore throat, coughs, colds	21	21	—	—	44
	Irritated/sore eyes	17	20	—	—	48
6*	Eye, nose or throat symptoms	27	26	—	—	42
	Lower Respiratory					
1	Tight chest	2.3	—	1.4	1.1	6.1
	Short of breath	1.5	—	0.0	0.0	1.9
	Wheeze	3.1	—	0.0	0.0	5.1
3*	Tight Chest	6	5	10	9	12
	Difficulty breathing	6	6	10	8	12
	Flu-like symptoms	15	14	27	27	27
	Skin					
1	Dry skin	5.7	—	5.5	9.9	10.8
	Rash	1.9	—	2.7	3.3	2.1
	Itching skin	2.9	—	2.7	4.4	5.5
2	Dry skin	6.4	—	10.5	—	12.5
6*	Dry skin or rash	6	7	—	—	13

*Prevalence data originally reported as integer values.

is possible as workers who left the workplace due to illness or discomfort were not included, but this would lead to underestimation of any real effects.

Because "negative" (non-significant) studies are generally less likely to be submitted or accepted for publication, reviews of the literature are likely to contain a disproportionate number of studies with positive findings, relative to all studies actually performed on that subject.[34,35] Publication bias may in this way have inflated the magnitude of associations in this review; however, larger studies are considered less prone to this bias,[35] and PORs for air-conditioned buildings were consistently elevated even in the largest studies reviewed. Furthermore, a consistent "negative" finding of this reanalysis was not subject to such bias: symptom prevalence in simply mechanically ventilated buildings was no greater than that in naturally ventilated buildings.

Possible information biases include reporting or interviewer bias and misclassification. Reporting or interviewer bias, due to increased worker or interviewer concerns about health in sealed, air-conditioned buildings, could have increased frequency of reported symptoms in such buildings and thus led to overestimation of odds ratios. This bias cannot be ruled out.

Awareness of the research hypothesis by interviewers or subjects could have had a similar effect. Interviewers were used only in study 1, so subjects in other studies could only have inferred hypotheses from the self-administered questionnaires. In study 1, with the highest PORs of all studies reviewed, interviewers conducted most interviews without knowledge of the buildings where subjects worked, and researchers had no prior hypotheses regarding ventilation type and symptoms other than lower respiratory.[12]

Misclassification bias could have occurred from crude classification of the very complex ventilation systems found

TABLE 4—Prevalence Odds Ratios (and 95% CIs) for Reported Work-related Symptoms among Office Workers in Different Building Ventilation Categories Relative to Naturally Ventilated Buildings

Study	Symptoms	Building Ventilation Categories I Natural POR (95% CI)	II Simple Mechanical POR (95% CI)	III Air-Conditioned No Humidification POR (95% CI)	IV Air-Conditioned Steam Humidification POR (95% CI)	V Air-Conditioned Water-Based Humidification POR (95% CI)
	Central Nervous System					
1	Headache	1.0	—	3.1 (1.7,5.7)	2.3 (1.3,4.0)	2.5 (1.6,3.8)
	Lethargy	1.0	—	5.1 (2.9,9.2)	2.6 (1.5,4.7)	4.2 (2.7,6.5)
2	Headache	1.0	—	2.5 (1.9,3.3)	—	2.1 (1.7,2.7)
	Lethargy	1.0	—	4.2 (3.2,5.5)	—	3.2 (2.6,4.0)
3	Headache	1.0	0.8 (0.6,1.0)	1.3 (1.0,1.6)	1.4 (1.1,1.9)	1.4 (1.1,1.7)
	Lethargy	1.0	0.7 (0.6,0.9)	1.4 (1.1,1.7)	1.5 (1.2,2.0)	1.9 (1.5,2.3)
4	Headache	1.0	2.2 (1.3,3.7)	—	—	2.7 (1.9,3.8)
5	Headache	1.0	—	—	—	4.2 (1.9,9.3)
	Lethargy	1.0	—	—	—	4.0 (1.6,9.9)
6	Headache, fatigue, or malaise	1.0	1.1 (0.9,1.3)	—	—	2.3 (1.7,3.1)
	Upper Respiratory/Mucus Membrane					
1	Nose Symptoms	1.0	—	2.6 (1.1,5.9)	3.0 (1.4,6.2)	3.8 (2.2,6.5)
	Dry throat/blocked nose	1.0	—	2.5 (1.2,5.1)	2.2 (1.1,4.5)	4.8 (3.1,7.6)
	Eye symptoms	1.0	—	1.5 (0.5,3.9)	2.5 (1.1,5.4)	3.1 (1.8,5.3)
2	Nose symptoms	1.0	—	2.9 (2.1,3.9)	—	1.2 (0.9,1.6)
	Throat symptoms	1.0	—	2.5 (1.9,3.4)	—	2.4 (1.9,3.1)
	Eye symptoms	1.0	—	1.9 (1.4,2.7)	—	2.4 (1.8,3.3)
3	Runny nose	1.0	0.8 (0.6,1.1)	1.3 (1.0,1.8)	1.4 (1.0,2.0)	1.5 (1.2,1.9)
	Blocked nose	1.0	0.7 (0.6,0.9)	1.4 (1.1,1.7)	1.4 (1.0,1.8)	1.8 (1.5,2.3)
	Dry throat	1.0	0.9 (0.7,1.1)	1.7 (1.4,2.1)	1.6 (1.2,2.1)	2.0 (1.6,2.5)
	Dry eyes	1.0	1.1 (0.8,1.5)	2.0 (1.5,2.7)	1.8 (1.3,2.4)	2.2 (1.7,2.9)
	Itching eyes	1.0	0.9 (0.7,1.2)	1.6 (1.2,2.1)	1.3 (1.0,1.8)	1.7 (1.3,2.1)
4	Sore throat, coughs, or colds	1.0	1.0 (0.6,1.8)	—	—	3.0 (2.1,4.2)
	Irritated/sore eyes	1.0	1.2 (0.7,2.2)	—	—	4.5 (3.2,6.5)
6	Eye, nose, or throat symptoms	1.0	1.0 (0.8,1.2)	—	—	2.0 (1.4,2.7)
	Lower Respiratory					
1	Tight chest	1.0	—	0.6 (0.1,4.8)	0.5 (0.1,3.8)	2.7 (1.2,6.4)
	Short of breath	1.0	—	0	0	1.2 (0.4,4.0)
	Wheeze	1.0	—	0	0	1.7 (0.8,3.7)
3	Tight chest	1.0	0.8 (0.5,1.3)	1.7 (1.1,2.6)	1.5 (0.9,2.5)	2.1 (1.4,3.2)
	Difficulty breathing	1.0	1.0 (0.6,1.6)	1.7 (1.1,2.6)	1.4 (0.8,2.3)	2.1 (1.4,3.2)
	Flu-like symptoms	1.0	0.9 (0.7,1.3)	2.1 (1.6,2.8)	2.1 (1.5,3.0)	2.1 (1.6,2.8)
	Skin					
1	Dry skin	1.0	—	1.0 (0.3,3.1)	1.8 (0.7,4.4)	1.8 (0.9,3.5)
	Rash	1.0	—	1.5 (0.3,8.0)	1.8 (0.4,7.9)	1.3 (0.4,4.3)
	Itching skin	1.0	—	1.0 (0.2,4.9)	1.6 (0.4,5.6)	1.9 (0.7,4.7)
2	Dry skin	1.0	—	1.7 (1.1,2.7)	—	2.1 (1.5,3.1)
6	Dry skin or rash	1.0	1.3 (0.9,1.7)	—	—	2.5 (1.6,4.1)

in large buildings. The resulting nondifferential misclassification, however, would have led to underestimation of any real effects.

A number of potential confounding factors could have affected results. Some non-building factors known to be related to symptom reporting[17,19,22]—such as particular social and work environments, gender, job types, workplace smoking, and season of measurement—could have introduced confounding if consistently associated with particular ventilation types. There is no evidence that these factors were related to building ventilation type in the buildings studied.

Studies 2, 4, and 5 reported no assessment of potential confounding factors.[16,20,21] Study 1 reported only that smoking prevalence was similar in all buildings studied.[12] For study 3, Hedge, *et al*,[19] performed a multivariate analysis examining simultaneous association of a number of factors with total number of symptoms reported by each individual. They reported that higher overall symptom prevalence was associated with air-conditioned buildings, independently of associations also found between symptom prevalence and various individual, psychological, occupational, and architectural factors.

For study 6, a multivariate analysis found that gender, job category, work activities, and psychosocial job factors were associated with work-related symptoms. Differences in symptom prevalence between buildings, however, remained substantially the same after multivariate adjustment, with symptom prevalence still highest in the single category V building.[23]

Some building-related factors may have caused confounding. Air-conditioned buildings, being on average newer than naturally or simply mechanically ventilated buildings, will be more likely to contain fluorescent lighting,[21,36] inner offices distant from windows, with no natural light,[21] "open-plan" office layouts,[20] newer synthetic materials which emit various organic compounds,[30] and materials with high absorptive surface area (such as carpets and cloth-covered partitions) capable of accumulating and re-releasing physical,

chemical, or biological contaminants.[26,29,30,37] To produce spuriously the associations found, these factors would have to be strongly related to both symptom prevalence and ventilation type.[38] This possibility could not be evaluated in the studies reviewed here.

The patterns of association in this reanalysis suggest that symptom reporting is systematically related to still unidentified factors in sealed, air-conditioned buildings. If bias or confounding factors are not responsible for these patterns, these symptom increases may represent one or more as yet unidentified ventilation-related illness syndromes. Possible ventilation-related risk factors include inadequate fresh air ventilation, reduced thermal comfort, use of chemicals within ventilation systems to kill biologic agents,[27] and recirculation of infectious[39] or allergenic[30] biologic agents. Sufficient environmental data were not available from the studies reviewed to evaluate these factors (see Appendix).

Results of this reanalysis are consistent with increased exposure of workers to biologic aerosols from water within the ventilation system (i.e., on or under air-conditioning cooling coils, which dehumidify ventilation air, and in water-based humidification systems).[17,30] Inconsistent associations found between symptom prevalence and water-based humidification may reflect a dual effect of such systems: reduced risk of respiratory infections from humidification generally,[40] but increased risk of respiratory sensitization to biological contaminants (as in humidifier fever or hypersensitivity pneumonitis) from water-based humidification specifically.[41]

In conclusion, the reanalysis of epidemiologic studies presented here suggests that, at least in some countries, increases in building-related symptoms in offices may not be unusual events, but relatively common events not usually attributed to buildings. Future study of this problem, beyond investigation of complaint buildings, is advisable.

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APPENDIX

Summaries of Findings of Six Studies

Findings of the studies included in this reanalysis are summarized here; terms used to describe ventilation categories are from the studies themselves.

The first study,[12–15] involving eight buildings, found higher symptom prevalence associated with mechanical compared with natural ventilation, even higher symptom prevalence for buildings with air-conditioning, and the highest prevalence in buildings with air-conditioning and humidification. Whether air within air-conditioned buildings was recirculated did not markedly affect symptom prevalence.

The second study,[16] involving 27 buildings, reported higher symptom prevalence in both buildings with mechanical ventilation and those with air-conditioning and humidification, compared to buildings with natural ventilation. Increases in the two groups were similar. In this study, researchers included several buildings they suspected of having problems; they point out, though, that at that time the sick building syndrome was not known in the United Kingdom.*

The third and largest study,[17–19] involving 47 buildings, found lower symptom prevalence in buildings with mechanical ventilation relative to those with natural ventilation, higher prevalence in buildings with air-conditioning, and the highest prevalence in two subsets of buildings with air-conditioning: those with evaporative humidification,[18] and those with "water-based" air-conditioning systems (this refers to method of cooling the building[42,43] and is not related to type of humidification).

The fourth study,[20] involving five buildings, found prevalence of all symptoms measured to be higher in air-conditioned buildings relative to conventional (not defined in report), but prevalence of only one symptom higher in unconditioned buildings relative to conventional buildings.

The fifth study,[21] involving two buildings, found higher symptom prevalence in an air-conditioned, humidified building relative to a naturally ventilated one.

The sixth study,[22–26] involving 14 buildings (minimal data were reported on some additional buildings), found symptom prevalence in mechanically ventilated buildings higher, but not significantly so, than in naturally ventilated buildings. The oldest town halls had the lowest prevalence of symptoms.

The first two studies used physician-administered questionnaires, and defined work-related symptoms as those occurring more than twice in the previous year, and improving on days away from work;[12,16] symptoms were also required to have started or worsened since working in the current building, except for nose, throat, and eye symptoms in the second study.** All the other studies used self-administered questionnaires. Studies 3 and 5 defined work-related symptoms as those occurring more than twice in the previous year and also improving on days away from work.[17,21] Study 4 defined work-related symptoms as those reported to occur frequently at work.[20] Study 6 defined work-related symptoms as those occurring at least weekly and improving on days away from work.[22] Although individual symptoms were assessed in this study, data were reported only on groups of symptoms.

All studies achieved high response rates among workers in buildings studied—in study 1 from 75–97 percent;[12] in study 2 from 86 percent upward;[16] in study 3 from 67–100 percent, averaging 92 percent;[17] in study 5 averaging 97 percent;[21] and in study 6 from 61–93 percent, averaging 80 percent.[22]

No study reported outside air ventilation rates in buildings. In study 1, environmental measurements were taken in only two buildings, one with low and one with high symptom prevalence; no significant differences were found in the parameters measured—dry bulb temperature, globe temperature, relative humidity, air moisture content, air velocity, positive and negative ions, carbon monoxide, ozone, and formaldehyde.[14] In study 2, levels of airborne particles, as well as viable fungal and bacterial microorganisms, were measured in all buildings studied; no associations were found with symptom prevalence.[16] Study 6 reported that increased symptom prevalence was related to factors such as increased temperature and greater amounts of high surface area materials, open shelving, total floor dust, and potentially allergenic floor dust.[26]

Studies 1 through 5 assessed all buildings between November and March; study 6 between February and May.[22]

*Personal communication, J. Harrison.

**Personal communication, A. Robertson.

***Personal communication, A. Hedge.

Alfred Sommer Named Dean of Hopkins School of Public Health

Alfred Sommer, MD, MHS, a distinguished Hopkins ophthalmologist, epidemiologist and specialist in international health, has been named dean of the Johns Hopkins University School of Hygiene and Public Health, effective September 1, 1990. Sommer succeeds Donald A. Henderson, MD, MPH, who has served as dean for 13 years. The School of Public Health is the largest in the world, with more than 300 faculty members and 1,000 students from 75 countries and projects in nearly 40 nations.

Sommer is professor of ophthalmology at the School of Medicine and holds joint appointments in the departments of epidemiology and international health in the School of Public Health. He is internationally recognized for his pioneering work with vitamin A, in which he found that small, inexpensive doses can save the lives of millions of children around the world. Sommer's discovery came as a result of his work using vitamin A to prevent blindness in malnourished children in Indonesia and other developing nations.

A native of New York City, Sommer, 47, received his medical degree from Harvard in 1967 and his master of health science degree from the Hopkins School of Public Health in 1973.

Sommer is an adviser to numerous national and international groups, including the World Health Organization, the National Eye Institute, the Asian Foundation for the Prevention of Blindness, the International Agency for the Prevention of Blindness, Helen Keller International and the Royal Commonwealth Society for the Blind. He is president of the International Federation of Eye Banks and has published more than 180 papers, reviews or book chapters.

The Association of Corneal Arcus with Coronary Heart Disease and Cardiovascular Disease Mortality in the Lipid Research Clinics Mortality Follow-up Study

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Abstract: The relationship between corneal arcus (arcus senilis) and mortality from coronary heart disease (CHD) and cardiovascular disease (CVD) is examined in a prospective study of White men (n = 3,930) and women non-hormone users (n = 2,139), ages 30–69, followed for an average of 8.4 years as part of the Lipid Research Clinics Mortality Follow-up Study. After excluding those with clinically manifest CHD at baseline, corneal arcus was strongly associated with CHD and CVD mortality only in hyperlipidemic men ages 30–49 years, for whom the relative risk for CHD and CVD death was 3.7 and 4.0, respectively, after adjusting for age, total cholesterol, HDL cholesterol, and smoking status using a Cox proportional hazards model. Among 30–49 year old males, corneal arcus appears to be a prognostic factor for CHD, independent of its association with hyperlipidemia in this age-group, of about the same magnitude as other common risk factors, underscoring the usefulness of corneal arcus as a prognostic factor to the practicing clinician. (*Am J Public Health* 1990; 80:1200–1204.)

Introduction

Corneal arcus is an extracellular lipid infiltration of the peripheral cornea. It appears as a yellowish-white ring around the cornea, and is separated from the limbus by a 0.3 to 1 mm lucid zone.[1–4] These deposits consist mainly of cholesterol, cholesterol esters, phospholipids, and triglycerides.[1,5] Corneal arcus has been described in children,[3,6] but it is more prevalent among older people and therefore has been often called arcus senilis.[1–6] It occurs more often among Blacks than Whites[4,6,7] and tends to appear earlier in life in Blacks than in Whites.[1–4,6,7]

Rifkind and Dickerson[8] found an association of corneal arcus with coronary heart disease (CHD), and pointed out that its characteristics are similar to those of atherosclerotic lesions: both accumulate in relatively avascular tissues, both increase with age, and are more frequent among males. Data from the Western Collaborative Group Study indicated an association of corneal arcus with the incidence of coronary heart disease in men below age 50.[9] Moreover, it has been found to be related to risk factors of CHD such as hyperlipidemia and to occur more often among patients with familial dyslipoproteinemias.[1–5,6–18] However, data from several studies showed no independent relationship between corneal arcus and CHD and suggested that both were related to elevated serum cholesterol levels.[7,8,12,15,17]

Segal, *et al*,[18] examined the data from the Lipid Research Clinics (LRC) Prevalence Study, which is a major source of information on the occurrence of hyperlipidemia in North America, and found an independent relationship between corneal arcus and the prevalence of CHD. We here report on the relation between corneal arcus diagnosed in White participants of the LRC Prevalence Study based on the mortality from CHD and CVD during a mean follow-up of 8.4 years.

Methods

The methods of the Lipid Research Clinics Prevalence Study and of the Lipid Research Clinics Mortality Follow-up Study have been described in detail elsewhere.[19,20] In summary, the following points are relevant for the present analysis.

Prevalence Study

The Prevalence Study was conducted in the period 1972–76 in 10 Lipid Research Clinics in the United States and Canada. In the first survey (Visit 1), 60,502 individuals (out of 81,926 eligibles) representing a wide ethnic, geographic, socioeconomic, and age variation had their plasma levels of cholesterol and triglycerides determined. A 15 percent random sample of all Visit 1 participants, plus all those with high plasma cholesterol or triglyceride levels or who were taking lipid-lowering medications, plus varying (by clinic) proportions of those with borderline elevation in cholesterol or triglyceride, were invited for a more extensive examination (Visit 2). See Appendix for the definition of "high" and "borderline elevated." Overall, 85 percent of the 12,595 persons eligible for Visit 2 were actually examined. The data used in this analysis, except for a Visit 1 hyperlipidemia variable, were obtained at Visit 2.

Follow-up Mortality Study

All men and women who were at least 30 years old at Visit 2 had their vital status determined annually from 1977 through 1983. Mean follow-up was 8.4 years, with a range of 5.7 to 9.9 years. In case of death, cause was classified as coronary heart disease (CHD), cardiovascular disease (CVD), or due to other causes, using standard criteria.[20] For deaths suspected of being caused by CVD, the cases were reviewed by a panel of cardiologists. They analyzed the death certificate and information obtained from the next-of-kin and from hospitalization records if the person was hospitalized within 30 days prior to death, or from the personal physician if there had been no hospitalization within 30 days prior to

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death. Vital status is known for over 99.6 percent of the participants.

Exclusions

Excluded from all CVD analysis were participants with possible or definite CHD at baseline, according to the presence of one of the following: angina pectoris diagnosed by the Rose Questionnaire[21] or during the treadmill test; use of angina medications, antiarrhythmics, digitalis or propranolol; exclusion from the treadmill test because of congestive heart failure, R-on-T type premature ventricular contraction, ventricular tachycardia, parasystolic focus, atrial flutter or atrial fibrillation; or resting ECG evidence of diagnostic or equivocal myocardial infarction.[22] Female users of estrogens or progestogens were excluded because of the small number who had corneal arcus. Black participants were excluded because of small numbers. Individuals invited to Visit 2 because of borderline hyperlipidemia were also excluded for statistical reasons, as explained below, as were individuals invited solely because of use of lipid-lowering medication. Finally, those with nonfasting (<12 hours) or missing lipid values and pregnant women were excluded from the analysis.

Corneal Arcus Diagnosis

The interviewers were trained to identify corneal arcus in the Visit 2 using a uniform protocol.[23,24] They stood in front of the participants so that their eyes were at the same level during the examination, which was usually done in natural (rather than artificial) light, the source of which was tangential to the eye. The upper and lower poles of the eyes were examined. Interviewers were directed to look for a whitish fatty material at the periphery of the cornea. The corneoscleral junction had to be observable for a diagnosis of corneal arcus to be made. For these analyses only positive answers were regarded as indicative of corneal arcus and an "uncertain" response was considered as negative.

Laboratory Methods

A detailed description of the laboratory processing procedures is provided in the LRC Program Laboratory Methods Manual[25] and reviewed elsewhere.[26]

Statistical Methods

Visit 2 sample data are analyzed to draw inferences about a population like that of Visit 1. In order to account in analysis for the sampling scheme, the data are treated as a stratified random sample, with two strata: hyperlipidemics, all of whom were invited to Visit 2, and normolipidemics (including borderline hyperlipidemics), 15 percent of whom were invited to Visit 2. For simplification, the oversampling of borderline hyperlipidemics and of users of lipid-lowering medication were excluded. It should be emphasized that borderline hyperlipidemics were not in general excluded, but only the oversampling invited to Visit 2. Because of the higher proportion of hyperlipidemics in the sample relative to the population, weighting techniques were used in the estimation of overall population parameters.

CHD and CVD mortality rates (deaths per 1,000 person-years of follow-up) were calculated for corneal arcus cases and non-cases, for each age/sex group, by lipid strata and weighted across lipid strata. Confidence intervals or tests of statistical significance are not given for these descriptive statistics, but rather are reserved for the models' fit. Age groups are 30–49 and 50–69. Similar mortality rates were also calculated with respect to other possible prognostic factors besides corneal arcus. These factors are given below, along with cutpoints which define the categories (greater than or equal to versus less than cutpoint) for which mortality rates were calculated:

- Serum cholesterol level (240 mg/dl).
- Fasting serum triglyceride level (250 mg/dl).
- Low density lipoprotein (LDL) cholesterol level (160 mg/dl).
- High density lipoprotein (HDL) cholesterol level (40 mg/dl).
- Body mass index (BMI) (30 kg/m^2).
- Smoking status (current versus ex- or nonsmoker).
- Family history of coronary heart disease (either at least one parent died of CHD or experienced angina before the age of 60 or at least one sibling died of CHD before the age of 60).
- Graded exercise test (positive ECG showed a significant ST elevation or depression or a significant change in the ST integral value—for details see Gordon, *et al*[27]).
- Ischemia on resting ECG excluding myocardial infarction (either evidence of left ventricular hypertrophy or other significant ischemia; Minnesota codes 3.1-3.3 with 4.1-4.3 or 1-3-1 through 1-3-6, or 2.1, 4.1, 4.2, 5.1, 5.2, 6.1, 8.1, or 8.2).

In considering the effects of possible confounding variables on the relation in males aged 30–49 between corneal arcus and CHD and CVD mortality, survival models were fit by lipid sampling stratum, using Cox proportional hazards models. In addition to arcus status, survival status and baseline age, the following baseline variables were considered: continuous [total cholesterol, LDL cholesterol, HDL cholesterol, alcohol consumption (g/day), body mass index (kg/m^2), systolic blood pressure, triglyceride]; dichotomous [family history of CHD (yes = 1, no = 0), smoking (yes = 1, no = 0)]. These variables were added one at a time to the "basic" model, with survival status the dependent variable and corneal arcus and age independent variables, and discarded if the corneal arcus survival association was not changed by their inclusion.

Results

For men free of coronary heart disease at baseline, stratified by two age groups and by lipid sampling strata, the prevalence of corneal arcus, the total follow-up time, the mortality rates from CHD and the relative risk are presented in Table 1. In addition, results are given for the two sampling strata combined, with the stratum-specific results weighted to yield estimates of mortality rates for the Visit 1 population. A high relative risk of CHD mortality was associated with the diagnosis of corneal arcus among hyperlipidemic men under 50 years of age but not among older men. Table 2 shows the same trend when mortality from CVD is considered instead of CHD.

In female non-hormone users, the low prevalence of corneal arcus and the small number of CHD and CVD events preclude any kind of analysis in the younger age group; in the older group, where its prevalence and the number of events increase, the same lack of predictive value for CHD and CVD mortality is observed as in the older men (data available on request to authors).

The proportional hazards models of time to CHD or CVD death (Table 3) show that corneal arcus is an independent prognostic factor for mortality from CHD or CVD in men ages 30–49 years in the hyperlipidemic sampling stratum; for normolipidemic younger men its presence does not appear

TABLE 1—Estimated CHD Mortality Rates (deaths per 1000 person-years of follow-up) by Age Group and Corneal Arcus Status, Excluding Those with Prior Manifestations of CHD: Males

Age (years)	Sampling Stratum	Corneal Arcus Status	N	Number CHD Deaths	Total Follow-up Time (person-years)	Estimated CHD Mortality Rate	Estimated Relative Risk
30–49	Hyperlipidemic	No	1315	10	11237.6	0.89	6.7
		Yes	85	4	675.1	5.93	
	Normolipidemic	No	1202	8	10339.5	0.77	3.4
		Yes	46	1	377	2.65	
	Total*	No	2517	18	21577.1	0.79	4.7
		Yes	131	5	1052.6	3.74	
50–69	Hyperlipidemic	No	311	13	2504.2	5.19	0.0
		Yes	68	0	583.1	0.00	
	Normolipidemic	No	597	18	5031.9	3.58	0.9
		Yes	115	3	947.5	3.17	
	Total*	No	908	31	7536.16	3.64	0.8
		Yes	183	3	1530.6	2.99	

*Mortality rates weighted across sampling strata.

TABLE 2—Estimated CVD Mortality Rates (deaths per 1000 person-years of follow-up) by Age Group and Corneal Arcus Status, Excluding Those with Prior Manifestations of CHD: Males

Age (years)	Sampling Stratum	Corneal Arcus Status	N	Number CHD Deaths	Total Follow-up Time (person-years)	Estimated CHD Mortality Rate	Estimated Relative Risk
30–49	Hyperlipidemic	No	1315	13	11237.6	1.16	6.4
		Yes	85	5	675.1	7.41	
	Normolipidemic	No	1202	14	10339.5	1.35	2.0
		Yes	46	1	377	2.65	
	Total*	No	2517	27	21577.1	1.32	3.2
		Yes	131	6	1052.1	4.23	
50–69	Hyperlipidemic	No	311	22	2504.2	8.79	0.0
		Yes	68	0	583.1	0.00	
	Normolipidemic	No	597	26	5031.9	5.17	1.0
		Yes	115	5	947.5	5.28	
	Total*	No	908	48	7536.2	5.31	0.9
		Yes	183	5	1530.6	4.98	

*Mortality rates weighted across sampling strata.

TABLE 3—Estimated CHD and CVD Hazard Ratio, Comparing Subjects with Corneal Arcus to Those Without; Adjusted for Age, Total Cholesterol, HDL-C, and Smoking*, Men Aged 30–49 Years

Lipid Stratum	CHD Hazard Ratio	(95 Percent Confidence Interval)	CVD Hazard Ratio	(95 Percent Confidence Interval)
Hyperlipidemic	3.7	(0.9,14.7)	4.0	(1.2,12.9)
Normolipidemic	2.2	(0.3,20.1)	1.3	(0.2,10.4)

*LDL-cholesterol, alcohol consumption, BMI, systolic blood pressure, and triglyceride were also considered, but had no effect on the C.A./survival association, so were discarded.

to be of the same importance. The covariates included are age and those whose inclusion in the model substantially modified the estimates of relative hazard of CHD or CVD mortality as related to corneal arcus. These covariates are total cholesterol, HDL-cholesterol, and smoking. With or without these additional covariates the relative hazards are similar for normolipidemics, but for hyperlipidemics the relative hazards adjusted for age alone are about 1.6 times those for models presented in Table 3.

The relative risk of the diagnosis of corneal arcus in the 30–49 year old male group is compared in Table 4 to that of other possible risk factors. As can be seen there, corneal arcus appears to be at least as statistically strong a prognostic factor for CHD as obesity, hypo-HDL, ischemia on resting ECG, hypertriglyceridemia and family history of CHD, and not very different in magnitude from the relative risk from smoking habit, hypertension, hyper-LDL, and hypercholesterolemia. Details concerning this relation are given in Segal, *et al.*[18]

Discussion

In men under 50 years of age, corneal arcus has been shown in a prospective study to be a predictor of CHD

TABLE 4—Estimated Mortality Rates (per 1000 person-years) and Relative Risks of Death from CHD in Asymptomatic Men Aged 30–49 Years, by CHD Risk Factor Status

		Hyperlipidemic Stratum		Normolipidemic Stratum		Population Estimates*	
Risk Factor	Status	Death Rate	Relative Risk	Death Rate	Relative Risk	Death Rate	Relative Risk
Corneal Arcus	Yes	5.93	6.66†	2.65	3.44	3.74	4.73
	No	0.89		0.77		0.79	
Current Smoker	Yes	2.00	4.26††	1.72	5.73††	1.78	5.56††
	No	0.47		0.30		0.32	
Quetelet Index	≥30	1.27	1.55	0.96	—	1.00	3.57
	<30	0.82		—		0.28	
Hypertensive	Yes	1.96	2.33	2.68	7.66††	2.48	5.90††
	No	0.84		0.35		0.42	
LDL-C Level	≥160	1.84	3.61††	3.33	9.79††	2.39	6.83††
	<160	0.51		0.34		0.35	
HDL-C Level	<40	1.76	2.71†	1.66	3.95†	1.68	3.73††
	≥40	0.65		0.42		0.45	
Ischemia on Exercise ECG	Present	9.79	9.79	10.00	14.29	9.94	13.25
	Absent	1.00		0.70		0.75	
Ischemia on Resting ECG	Present	7.64	7.64	2.15	2.56	2.70	3.14
	Absent	1.00		0.84		0.86	
Cholesterol Level	≥240	2.19	—	5.21	12.71††	2.59	6.64
	<240	—		0.41		0.39	
Triglyceride Level	≥250	1.73	1.94	2.37	3.04	1.78	2.25
	<250	0.89		0.78		0.79	
Family History of CHD	Present	2.17	2.44	0.96	1.17	1.21	1.46
	Absent	0.89		0.82		0.83	

*Weighted across sampling strata.
†$0.05 < p < 0.10$, two-sided test.
††$0.01 < p < 0.05$, two-sided test.

mortality, independent of presence of hyperlipidemia, and a stronger predictor for hyperlipidemics. Above the age of 50 its diagnosis does not appear to be positively related to the subsequent manifestation of a fatal CHD event. These findings help to clarify previous discussion about this relation,[7,28] confirming the observations made in retrospective,[8,29] cross-sectional,[18,30] and prospective studies.[9] They are in agreement with the interpretation that corneal arcus and coronary atherosclerosis share common determinants in young men, while local factors are more important for its occurrence in older persons.[8,12]

The findings in women confirm the low prevalence of corneal arcus in the young age group,[30,31] which with the low mortality rates rules out the possibility of drawing conclusions about its relation to CHD and CVD mortality. In the older group, where the prevalence of corneal arcus and the mortality rate increase, there is the same lack of relation observed in older men.

The ability to detect patients at high risk of CHD has been of continuous concern, considering the frequent presentation of the disease as sudden death[32] and its devastating consequences, especially in the families of young men. The magnitude of the corneal arcus risk in this age group compared to other usual screening tests (Table 4) highlights the utility of this simple and inexpensive tool as a prognostic factor and reinforces the importance of its diagnosis during the physical examination. This is noteworthy, considering that it had lower prevalence in the baseline of this population-based cohort than in some studies which used hospitalized or referred patients. The fact that, in this study, corneal arcus was detected only by a single examination, without the use of slit lamp, could have resulted in some misclassification.[17] However, such misclassification would be independent of either lipid status or later CHD mortality, so would only bias our results toward the null, that is, toward finding no association between it and CHD mortality. The association between corneal arcus and hyperlipidemia *alone* would suggest evaluation of the lipid profile of young men with corneal arcus. The findings in this paper indicate that it also may be prudent to suggest that all young patients with corneal arcus should undergo a detailed evaluation of their other CVD risk factors.

APPENDIX
Age-Specific Cutpoints Used for Selecting Participants for Visit 2

	Cholesterol (mg/dl)*		Triglyceride (mg/dl)**	
Age Group	Borderline Cutpoint	Elevated Cutpoint	Borderline Cutpoint	Elevated Cutpoint
30–39	220	240	150	250
40–49	225	260	150	300
50–59	245	280	150	300
60+	245	280	150	300

*Conversion factor to SI Units (mmol/L): 0.02586
**Conversion factor to SI Units (mmol/L): 0.1129

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Environmental Tobacco Smoke Exposure during Infancy

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Abstract: We collected information about household smoking habits from 518 mothers when they made their first well child visit with a 6 to 8-week old infant. A urine sample was also collected from the infant, the cotinine concentration measured, and the measurement correlated with data provided by the mother. Eight percent of the infant urine cotinine values fell at or above 10 μg/L in the 305 households where no smoking was reported. Corresponding rates were 44 percent in the 96 households where a member other than the mother smoked, 91 percent in the 43 households where only the mother smoked, and 96 percent in the 74 households where both the mother and another household member smoked. In households where the mother smoked, infant urine cotinine levels were lower in the summer, and higher when the infant was breast-fed. A screening question about family smoking habits in conjunction with well child care could effectively define a group of infants exposed to environmental tobacco smoke and thus be at greater risk for respiratory diseases. (*Am J Public Health* 1990; 80:1205–1208.)

Introduction

Infants and young children chronically exposed to environmental tobacco smoke (ETS) have a higher risk of developing acute respiratory illnesses than their unexposed counterparts, and hospitalization is more often necessary to manage these conditions.[1–4] The studies defining this risk relied upon family reporting (usually by the mother) of numbers of cigarettes smoked in the household and other locations where the infant or child might be exposed. The availability of biochemical markers capable of measuring tobacco smoke products quantitatively in blood, saliva, and urine now offers the opportunity to obtain a more accurate estimate of actual tobacco smoke absorption by infants and children, irrespective of location. Cotinine, a metabolic derivative of nicotine, has proven particularly useful, because it is chemically stable, is not found in nature (thereby avoiding false-positive test results), can be measured reliably in non-smokers exposed to ETS, and has a circulating half-life of about one day.[5,6] Greenberg, *et al*,[7,8] have recently reported their observations on urine cotinine levels in infants exposed to ETS, documenting the relation between self-reported exposure and urine cotinine levels. The present report provides further data on this relation, based on analyses from a large, population-based study focused on 6 to 8-week-old infants receiving routine well child care in private physician offices and clinics in a mid-sized city in the United States.

Methods

Patient Population

Thirty-seven physicians in the greater Portland, Maine area were asked to participate in the study, and 30 (81 percent) agreed to take part; both of the well child clinics also participated. Following informed consent 518 infant/mother pairs were enrolled at their first well child visit between January and October 1988. Each participating mother answered questions about her age, years of completed education, occupation for both herself and the father, ages of other children, breast-feeding, smoking, number of cigarettes smoked, and smoking habits of others in the household. The age of infants at enrollment was between 6 and 8 weeks. The mean age of mothers in the study was 27.4 years, and they had completed, on average, 13 years of school. For 50 percent of the mothers, this was their first child. To examine the extent to which the study sample represented the childbearing population of Greater Portland, Cumberland County data were analyzed in a 70 percent subset of the county's pregnancies in 1987, all of whom had maternal serum alpha-fetoprotein screening. For both groups, 99 percent were White and average age was 27 years; 9 percent of the study population were not high school graduates compared to 11 percent of the county population.

Urine Collection and Cotinine Assay

It was explained that the urine sample was to be analyzed to learn more about environmental exposures, but cigarette smoke exposure was not specifically mentioned. Two cotton rolls were placed in the diaper at the beginning of the office visit. They were either removed and placed in the glass screw-top specimen vial (if wet) at the end of the office visit or mailed back by the mother if urination had not occurred. This method of urine collection in no way alters the concentration of urine constituents, such as creatinine, and the rate of evaporation of liquid is negligible.[9] Approximately half of the samples were obtained during the office visit. Nine percent of the samples were unsatisfactory for analysis, either because of stool contamination or inadequate volume, and in 81 percent of those cases a repeat urine sample was successfully obtained. Before assaying, the urine was transferred to a test tube by compressing the cotton rolls inside a disposable syringe.

The urine was assayed for cotinine by a competitive radioimmunoassay, using cotinine antiserum raised in rabbits and an iodine ^{125}-label described previously.[10,11] The standard curve of the procedure ranged from 0.25 to 12.0 μg/L, with an estimated lower limit of sensitivity of 0.2 μg/L. A dose-response relationship down to 0.3 μg/L has previously been demonstrated between serum cotinine and ETS exposure with this assay.[10] The lower limit of 1.0 μg/L was selected for detecting urine cotinine, however, to allow for possible assay variability within the 0.25 to 1.0 μg/L range that might result from differences in urine composition and

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pH. Consequently, all values below 1.0 μg/L in the present study are assigned a value of <1.0 μg/L.

Results

Figure 1 contains a scatterplot distribution of urine cotinine levels. Among 305 infants who were not exposed to household tobacco smoke, cotinine levels from 97 (32 percent) fell below 1.0 μg/L, and another 185 (60 percent) fell between 1.0 and 9.9 μg/L. Urine cotinine values of 10 μg/L and above were found in only 23 of the infants (8 percent). Overall, the median urine cotinine level in this group was 1.6 μg/L.

Among 96 infants with ETS exposure from other household members only, the median level was 8.9 μg/L; among 43 infants with ETS exposure from the mother only, the median level was 28 μg/L; among 74 infants with ETS exposure from both the mother and other household members, the median level was 43 μg/L. Cotinine values falling at or above 10 μg/L in those categories were: 44, 91, and 96 percent, respectively. The horizontal line drawn at 10 μg/L in Figure 1 represents a reasonable estimate of significant ETS absorption; 94 percent of the infants whose mother smoked had values above this level, as opposed to only 8 percent of the infants in the unexposed category.

Breast Feeding in Relation to Infant Cotinine Levels

Figure 1 also identifies infants who were breast fed (open circles) and those who were bottle fed (closed circles). Among the 401 mothers in the study who did not smoke, 207 (52 percent) breast fed, as opposed to 16 (14 percent) of the 117 mothers who smoked. In households where the mother smoked, breast feeding was associated with significantly higher infant urine cotinine levels, both in the presence of other smokers in the household (urine cotinine medians = 213 μg/L with breast feeding and 39 μg/L without breast feeding, 2-sided t-test on $\log_{10}$ transformed cotinine level, $p = 0.003$), and in the absence of other smokers in the household (urine cotinine medians = 87 μg/L and 25 μg/L, $p < 0.001$).

After excluding breast feeding, regression analyses were performed to examine the relation between number of cigarettes smoked by the mother (exposure) and $\log_{10}$ of the infant urine cotinine level (absorption) both in households where only the mother smoked and in households where others smoked as well. Absorption was related to exposure in both groups (regression coefficient = 0.0155, SE = 0.0068, in the 35 households where only the mother smoked) (regression coefficient = 0.0185, SE = 0.0046, in the 66 households where both the mother and other household members smoked). In both groups, the infant urine cotinine levels differed by a factor of approximately two over the range of exposure.

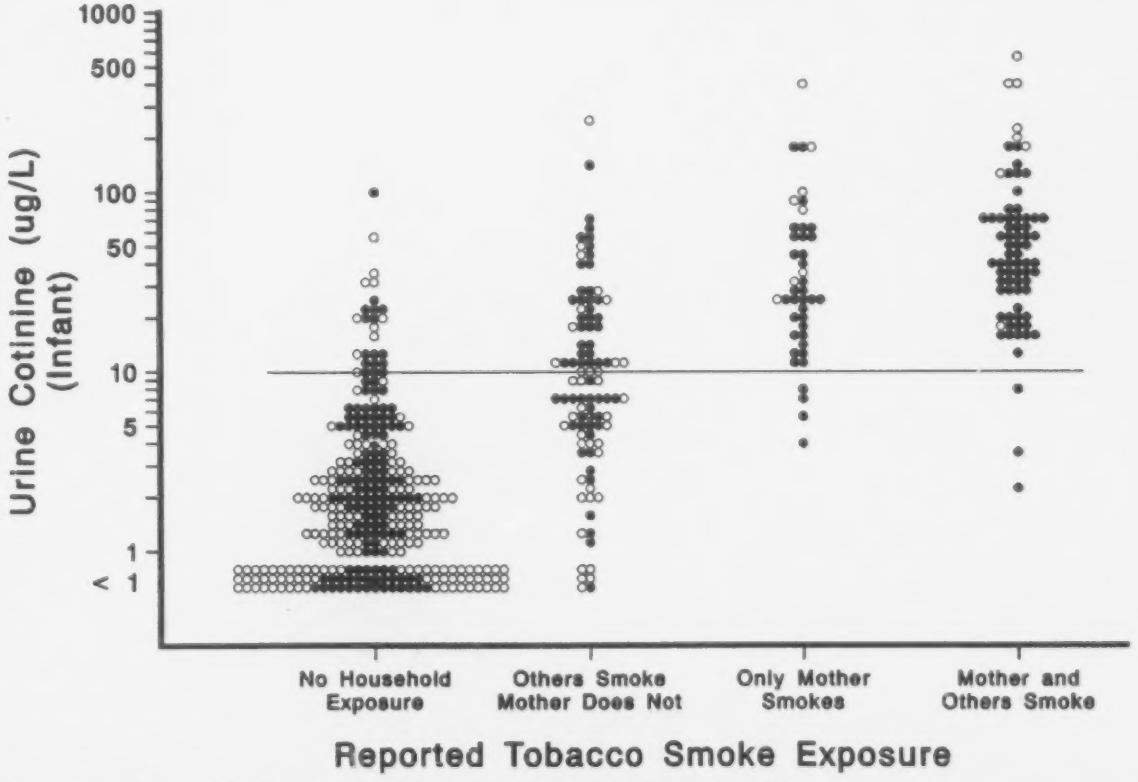

FIGURE 1—Urine Cotinine Levels in Infants, According to Environmental Tobacco Smoke Exposure in the Home.
Cotinine measurements are in spot urine samples. Closed circles indicate that the infant is not breast fed; open circles indicate breast feeding. Cotinine measurements < 1.0 μg/L are considered undetectable, and the horizontal line drawn at 10 μg/L is the cutoff defined from data in the study to indicate significant ETS absorption. Median urine levels in the four groups are as follows: no household exposure—1.6 μg/L; others smoke, mother does not—8.9 μg/L; only mother smokes—28 μg/L; mother and others smoke—43 μg/L.

Influence of Season of the Year on Urine Cotinine Levels in Smoking Households

Table 1 shows, in households where the mother smoked, that the median, 25th, and 75th centiles for infants' urine cotinine levels were significantly lower in the summer (2 sided t-test on $\log_{10}$ transformed cotinine levels, $p = 0.03$); this could not be explained either by the presence of other smokers in the household or by the number of cigarettes smoked by the mother. Preliminary analysis demonstrated no differences between winter, spring, and fall, and so these were combined in the table.

Discussion

Table 2 compares selected characteristics of the present population with those of the population studied by Greenberg, *et al*.[8] The infants in the present study were, on average, 31 days older at enrollment and were primarily Caucasian. The ages, numbers of cigarettes smoked by the mothers, and rate of breast-feeding in mothers who smoked were similar in the two studies. A greater proportion of women in the present study group had finished high school. Greenberg, *et al*, found that infant urine cotinine concentrations were at or above 2 μg/L in 60 percent of their subjects, as compared with 64 percent in the present study, and median cotinine values were also similar (9 and 11 μg/L, respectively) in infants who were not breast fed.

Breast feeding has already been reported to influence infant urine cotinine levels among mothers who smoke,[12,13] and the present study provides further documentation of that effect. Breast milk from mothers who smoke contains high levels of cotinine, and it is likely that ingestion and absorption of cotinine (and also nicotine) explains why breast fed infants of smoking mothers have higher urine cotinine levels than when the mother does not breast feed. Cotinine from this source carries different implications from cotinine produced from inhaled tobacco smoke and needs to be taken into account when using cotinine results to estimate ETS absorption. In the present study household ETS was not reflected in raised infant urine cotinine levels via ingestion in breast fed infants, when the mother did not smoke.

Data from the present study provide evidence, for the first time, of a seasonal influence on infant urine cotinine levels. The lower median cotinine level in infant urine samples during the summer from households where the mother smoked suggests that increased outdoor activity, better ventilation, or a combination, might play a role. When interpreting urine cotinine values, this phenomenon needs to be taken into account, since the average yearly exposure to ETS may be underestimated during the summer months. This variable may behave differently in different climates.

Studies in adults indicate that urine cotinine levels are approximately 10 times higher than those in serum, both in smokers and non-smokers.[10] Since the urine cotinine value reflects exposure over several days, urine cotinine measurements appear suitable both for assessing degree of ETS-related risk and for measuring success following intervention to reduce ETS exposure. The cotton-roll method of urine collection employed in this study is particularly suited for use in the practitioner's office as part of either clinical investigation or ongoing health care counseling.

Our results suggest that a screening question about household smoking habits effectively defines a group of infants with urine cotinine levels of 10 μg/L or above. In our study, 86 percent of all infants with urine cotinine levels at or above 10 μg/L were found in the 41 percent of households where the mother indicated that either she or other household members smoked. Families in which any household member smokes might be considered suitable candidates for further documentation of the extent of infant absorption of ETS. Before implementing this type of approach, however, techniques for teaching families how to avoid infant ETS exposure need to be developed and evaluated.

TABLE 2—A Comparison of Household Smoking Habits and Infant Urine Cotinine Levels in the Present Study and a Recently Published Study

	Greenberg et al[8]	Present Study
Number of Infants	433	518
Average Infant Age at Enrollment (days)	18	49
White (%)	65	99
Mother not High School Graduate (%)	26	9
Mother Smokes (%)	27	23
Average Age of Mother (years)	25	27
Average Cigarettes Smoked/Day By Mother	14	17
Mother Smokes and Breastfeeds (%)	18	14
Household Member Smokes (%) (includes mother)	55	41
Infant Cotinine Value ≥2 μg/L (%)	60	64
Median Cotinine Level for Values ≥2 μg/L (Range)	9 (2–138)	11 (2–261)
Correlation of Mother's Number of Cigarettes versus Infant's Urine Cotinine (r_s value)	0.54	0.39

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TABLE 1—Urine Cotinine Levels in Infants Whose Mothers Smoke, According to Season of the Year*

Season	Number of Infants	Number of Cigarettes Mother Smoked (Mean)	Other Smokers in the Household (%)	Urine Cotinine (μg/L) Centile		
				25th	50th	75th
Winter/Spring/Fall	74	18	68	22	39	64
Summer	27	17	59	15	24	55

*Excludes 16 breast feeding infants

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Drinking Water Contamination and the Incidence of Leukemia: An Ecologic Study

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Abstract: An ecologic study was performed to examine the relation between the incidence of leukemias and the occurrence of volatile organic chemical (VOC) contamination of drinking water supplies within a study area comprised of subpopulations differentially exposed to drinking water VOCs (trichloroethylene and related solvents). Populations served by community water supplies were classified into exposure categories according to VOC contamination status based on 1984–85 sampling data. Leukemia incidence data (1979–84) were collected from a population-based cancer registry. For females, the standardized incidence ratio was elevated only in towns in the highest of three exposure categories. No association was observed in males in any of the exposure categories. A Poisson regression analysis of the data, using finer exposure strata, indicated an increase in risk among females with increasing level of contamination which appeared to be distributed evenly across all age strata. The rate ratio for females at the highest exposure stratum for total non-THM VOCs compared to the least exposed stratum was 1.68. The observed association appears to suggest that drinking water contaminated with VOCs may increase the incidence of leukemia among exposed females, but caution is advised in the interpretation of these results because of the uncertainties inherent in ecologic studies. (*Am J Public Health* 1990; 80:1209–1212.)

Introduction

Since the middle 1970s, there has been considerable epidemiologic interest in the relation between organic contaminants in drinking water and increased cancer incidence in exposed populations. The focus of several studies has been on chlorinated compounds that can form in relatively high concentrations following chlorination of surface water for disinfection. These compounds, some carcinogenic or mutagenic, include the volatile trihalomethanes (THMs) and many nonvolatile substances. There is some evidence that consumption of chlorinated surface water is related to an increased risk of bladder and possibly colorectal cancers.[1–4]

Groundwater was long thought to be relatively protected from such contamination. However, national and state surveys have demonstrated that numerous groundwater supplies throughout the nation have contained volatile organic compounds (VOCs) other than THMs,[5,6] in particular the halogenated solvents trichloroethylene (TCE), tetrachloroethylene (perchloroethylene or PCE), and 1,1,1-trichloroethane (TCA).

In Woburn, Massachusetts, an association was reported between the pattern of VOC water contamination (TCE and others) and the incidence of childhood leukemia.[7] Further epidemiologic analysis of the pattern of non-THM VOC water contamination and the incidence of leukemias is warranted in other geographic areas.

In New Jersey, routine semi-annual testing for 14 VOCs including halogenated solvents has been required of public community water systems since late 1984.[8] Results from 1984 and 1985 showed that about 18 percent of the state's 620 water systems, estimated to serve approximately 20 percent of the state's population, contained detectable levels of non-THM VOCs.[9,10] Data on THM concentrations are also available.

We performed a study of the possible relation between the incidence of leukemia and VOC contamination of drinking water in the state of New Jersey.

Methods

Study Population

In an ecologic study, an ideal geographic area would be completely served by community water systems for which monitoring data are available and would contain subunits that experience a range of contaminant concentrations to ensure exposure differences. The study area that best met these criteria consisted of towns completely within an area bounded by the Lower Passaic River and Saddle River drainage basins. The area is almost completely served by public water supplies, many of which have tapped VOC contaminated groundwater, while others utilize groundwater or surface water free of detectable levels of non-THM VOCs. This portion of the state is primarily urban and residential so that town coding in the Cancer Registry is likely to be accurate, i.e., mailing address and actual residence are likely to be the same.

Towns within the study area were excluded if less than 90 percent of the population was served by public water systems. The age- and sex-specific populations of each municipality were obtained from 1980 US Census summaries.

Exposure Measurement

The New Jersey Department of Environmental Protection compiled all sampling results for each water system serving populations in the study area. The drinking water quality data included the 14 VOCs under the state's monitoring requirements from 1984 and 1985, and THM analyses from 1984 and 1985.

The Department also provided other available sampling data, information on extent of distribution systems, well or reservoir use, and patterns of water purchases among systems so that the water supply of each town could be characterized. Any significant changes in water supply and use patterns in the past decade were noted.

For each water system, we calculated the mean chemical-specific concentrations of VOCs and THMs in distribution system samples for the period 1984–85. Although data were available for later years, the first rounds of monitoring were thought to be more representative of past exposure since corrective actions were taken by many water systems

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TABLE 1—Exposure Classification, Number of Reported Leukemia Cases 1979–84, and Population by Gender

Non-THM VOC rating for SIR	Exposure Stratum for regression (μg/l)*	Leukemia Cases (population) Males	Females	Number of Towns
++	72	1 (5,125)	4 (5,616)	1
	67	5 (11,346)	8 (12,610)	1
	47	0 (1,973)	0 (2,156)	1
	40	8 (12,699)	9 (14,104)	1
	37	11 (8,539)	7 (9,661)	1
		25 (39,682)	28 (44,147)	5
+	12	13 (15,546)	7 (16,683)	1
	9	7 (17,583)	14 (20,738)	1
	7	5 (5,314)	5 (5,488)	1
	5	13 (28,508)	10 (31,078)	4
		38 (66,951)	36 (73,987)	7
−	3	6 (8,358)	3 (8,836)	2
	2	7 (9,385)	4 (10,302)	2
	1	103 (161,195)	74 (182,053)	9
	0	29 (38,478)	19 (44,681)	2
		145 (217,416)	100 (245,872)	15

*Sum of average non-THM VOC concentrations in 1984–85 samples.

following the initial results. The number of distribution system samples for each supply varied from two to 50 in the period. Values for each non-THM VOC, total non-THM VOCs, and total THMs were then assigned to each town based on the water supplies that serve that town.

Disease Measurement

Incident cases of leukemias for the towns within the study area were collected from the New Jersey Cancer Registry for the six-year period 1979–84. Information on each case included date of diagnosis, age at diagnosis, town of residence at time of diagnosis, race and sex, and histologic type according to the International Classification of Diseases for Oncology.[11]

Data Analysis

Based on the 1984–85 contaminant values, towns were categorized as to contaminant status for total non-THM VOCs and for THMs. Categorical exposure levels were defined for each variable, empirically derived from inspection of the distribution of values. For each categorical exposure variable, incidence data for the towns within an exposure category were pooled, and standardized incidence ratios for the combined town groups were calculated. Expected incidence for the grouped areas were derived by applying New Jersey statewide 1982 age-sex-specific rates of total leukemias to the pooled 1980 age-sex-specific populations.[12] (Age-sex-specific rates for specific histologic types are not available.) Standardized incidence ratios (SIRs) were calculated by dividing the observed number of cases by the expected number for each town grouping. The 95% confidence intervals of the SIRs were estimated.[13]

Towns sharing the same values for non-THM VOCs were pooled into exposure strata. For each gender, the number of leukemia cases (total and type-specific) in each exposure stratum and age-group specific population was fitted to a log-linear regression model assuming a Poisson distribution of the counts. Separate regression models were developed for total non-THM VOC and for TCE and PCE, two subsets of that variable.[14,15] Predictor variables included in the models were age group (0–19, 20–49, and 50+) and exposure stratum; interactions were also included. Rate ratios were calculated from the parameters generated by the models. Analysis was performed using the GLIM software package.[16] A hierarchical backward elimination method[17] was used to determine final models.

Results

Three of the 30 towns originally contained in the study area were excluded since approximately 50 percent, 4 percent, and 1 percent of their population, respectively, was served by public community water supplies. Approximately 95 percent to 100 percent of the populations in each of the remaining 27 towns in the study area are served by public community water supplies. These 27 towns contained 688,055 persons (324,049 males and 364,006 females), or 9.3 percent of the population of New Jersey in 1980.

Table 1 includes mean total non-THM VOC values assigned to each town or group of towns for the SIR analysis. TCE, PCE, TCA, and dichloroethylenes (DCE) comprised nearly all of the non-THM VOC. Based on inspection of the average values for each town, three categories of total non-THM VOC status were formed: ++ (72–37 ppb), + (12–5 ppb), or − (3 to less than 1 ppb). Two categories of THM status were also formed: + (76–25 ppb) or − (4–1 ppb).

Figure 1 displays the geographic pattern of this categorization. For THMs, 19 towns were rated plus (+) and eight were rated minus (−).

Over the six-year period 1979–84, 208 leukemia cases occurred among males and 164 among females in the entire study area (Table 1). The most frequently reported histologic type among younger age groups of both sexes was acute lymphocytic leukemia (ALL). Among older age groups, chronic lymphocytic leukemia (CLL), acute granulocytic leukemia (AGL), and chronic granulocytic leukemia (CGL)

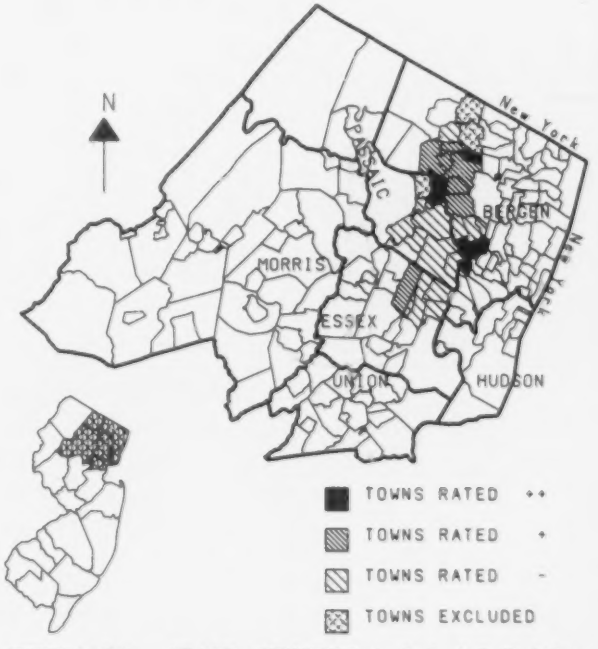

FIGURE 1—Pattern of Total Non-THM VOC Contamination in the Study Area as Categorized for the SIR Analysis

were most frequently reported. Few cases of acute monocytic leukemia were reported, and all were among older males. Twenty-eight percent of the reported cases were coded without specifying histology.

For the entire study area, the observed number of leukemia cases from 1979–84 did not differ from the expected number for either males or females. Among towns classified as ++ for total non-THM VOCs, however, the incidence of leukemia was elevated among females but not in males (Table 2).

No elevation of leukemia incidence was observed for groups of towns classified as either (+) or (−) for THM status among males or females (Table 2).

Regressions were performed for both sexes including categorical age and water quality variables. Results are reported in detail here for females only. The final models of the regression analysis for total leukemias in females (Table 3) do not contain interaction terms between age groups and contaminant status as no p values less than 0.05 were obtained.

The sum concentration of all non-THM VOCs was a statistically significant predictor of total leukemia incidence, adjusted for age. The coefficient for total VOCs was 0.0072 (S.E. =. 0039), and is interpreted as the natural logarithm of the increase in the rate ratio per unit increase in exposure. Thus, for the maximum town average total non-THM VOC concentration observed in the study, 72 μg/l, the rate ratio would be exp[(72 μg/l)(.0072)] = 1.68.

Modeled separately, the age-adjusted coefficients for TCE and PCE concentration were 0.012 (S.E. = .0067) and 0.035 (S.E. = .021), respectively. At the maximum town average TCE and PCE concentrations observed in the study, the rate ratios would be exp[(46 μg/l)(.012)] = 1.74 and exp[(16 μg/l)(.035)] = 1.75, respectively.

When the more common histologic types of leukemia (ALL, CLL, AGL, and CGL) were examined separately, the coefficients for total non-THM VOCs were all of similar direction and magnitude (ALL: 0.016; CLL: 0.0095; AGL: 0.010; CGL: 0.012). There were few cases of each histologic type, hence little power to detect meaningful associations.

TABLE 2—Standardized Incidence Ratios (SIRs) among Males and Females for Groups of Towns Classified According to Various Exposure Categories (Number of cases, 1979–84)

Exposure Category	Number of Cases: Observed	Expected	SIR	(95% Confidence Interval)
Males				
All towns	208	195.7	1.06	(0.92–1.21)
Total non-THM VOCs—1984–85				
++	25	25.0	1.00	(0.65–1.47)
+	38	40.7	0.93	(0.66–1.28)
−	145	130.0	1.12	(0.94–1.31)
Trihalomethanes—1984–85				
+	177	168.5	1.05	(0.90–1.22)
−	31	27.3	1.14	(0.77–1.61)
Females				
All Towns	164	146.8	1.12	(0.95–1.30)
Total non-THM VOCs—1984–85				
++	28	18.3	1.53*	(1.02–2.21)
+	36	30.3	1.19	(0.83–1.65)
−	100	98.2	1.02	(0.83–1.24)
Trihalomethanes—1984–85				
+	140	126.8	1.10	(0.93–1.30)
−	24	20.1	1.20	(0.77–1.78)

*Statistically elevated SIR ($p < 0.05$)

TABLE 3—Poisson Regression Analysis: Final Models for Total Leukemia Cases among Females

Model	Coefficient	Standard Error
	Exposure: Total non-THM VOCS	
Intercept	−1.85	(0.25)
AGE(20–49)	−0.022	(0.32)
AGE(50+)	1.78	(0.27)
TOTAL VOCS	0.0072	(0.0039)
	Exposure: TCE Concentration	
Intercept	−1.83	(0.25)
AGE(20–49)	−0.021	(0.32)
AGE(50+)	1.78	(0.27)
TCE	0.012	(0.0067)
	Exposure: PCE Concentration	
Intercept	−1.85	(0.25)
AGE(20–49)	−0.019	(0.32)
AGE(50+)	1.78	(0.27)
PCE	0.035	(0.021)

Discussion

Results suggest an association between the contamination of drinking water supplies with non-trihalomethane volatile organic chemicals and an increased incidence of leukemias among females. Because of limitations inherent in the study design, a statement of causal inference cannot be made with confidence at this time.

Ecologic studies are practical because they are quick and inexpensive, but because data reflect the group rather than the individual, the results are best used to develop rather than test etiologic hypotheses. Ecologic studies are susceptible to bias in the estimation of effect if there are factors operating at the individual level that are not accounted for in an aggregate exposure assessment. Additionally, at the group level, the degree of correlation among various predictor variables (multicollinearity) is likely to increase, so that the independent effects of predictors may be difficult to isolate. Potential biases and multicollinearity in ecologic studies can be minimized by using narrow exposure strata and regression to estimate effect.[18]

Misclassification of exposure may exist at both the population and at the individual level. In this study, exposure information (1984–85) was collected at the end of the case collection period (1979–84). Although sampling data prior to 1984 are limited, it was our judgment that these data are reasonable proxies for actual levels years earlier. Since the latency period of chemically-induced leukemias can be relatively short—as little as five years or less for benzene[19]—the problems associated with using exposure information gathered after the development of disease may be lessened.

Within a given population classified according to exposure, there may be significant variability of actual exposure to water-borne contaminants experienced by individuals. Individual-level data on personal tap-water use, residential history and migration patterns, and location of workplace or schools compared to residence were not available for this study.

Additionally, confounding variables such as occupational exposures or local toxic air emissions that may be causally related to the development of leukemias, may be correlated with the pattern of water contamination in the study area. However, each town grouping contained towns with both low and high air emission estimates from the recently available USEPA Toxics Release Inventory database.[20]

At least two of the common contaminants, TCE and PCE, have been shown to be carcinogenic in experimental animal studies, indicating the potential to be carcinogenic in humans.[21–25]

Observations in humans exposed to TCE and PCE or related solvents have not demonstrated a consistent association with leukemias. Studies of the mortality experience of dry cleaning workers have variously reported statistically elevated deaths from a variety of cancer sites. Mortality from leukemia was elevated (but not statistically significant) in one of the studies.[26] In a case-referent study of childhood leukemia, fathers' exposure to chlorinated solvents was associated with excess leukemia in the children.[27]

For TCE and PCE, it has been estimated that daily exposures from these chemicals in the ambient air of New Jersey amount to an average of 90 micrograms per day.[28] Using standard assumptions of drinking water consumption,[29] and assuming an equivalent dose from inhaling volatilized contaminants, drinking water concentrations greater than about 20–25 μg/l would add 80–100 μg of exposure, a doubling of the average daily exposure. This estimate illustrates that drinking water can be an important relative source of exposure at levels encountered in some water supplies.

Although we have analyzed total leukemias in this study, we recognize their clinical and etiologic diversity. Known or suspected risk factors include certain genetic traits (particularly for CGL and CLL), ionizing radiation (apparently most types but CLL), infectious agents, and chemicals such as benzene (primarily AML).[30,31] Combining histologic types together in this ecologic analysis may have obscured an effect if the hypothesized etiologic agent, VOC-contaminated drinking water, is specific to a particular histologic type. A detailed analysis of type-specific incidence in this study was precluded by the small number of cases.

The inconsistency of association with respect to gender in this study cannot be explained given the data and level of analysis used in this study.

Our results suggest that a relation may exist between leukemia incidence and VOC contamination of drinking water, at least among females, although this ecologic study cannot lead to a causal inference. Since the observed association may reflect an underlying causal relation, the potential public health importance warrants further study. In particular, a case-referent epidemiologic study would allow an analysis of several potential risk factors and several histologic types that could not be assessed in this ecologic study.

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Health Status of Migrant Farmworkers: A Literature Review and Commentary

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Abstract: I made a computerized search of MEDLINE files from 1966 through October 1989 followed by a review of this literature. Four hundred eighty-five articles were scanned; 152 were found specifically related to migrant families, while another 51 articles addressed the health of agricultural workers or farmers in general. Solid data exist on dental health, nutrition and, to a lesser extent, childhood health. Data also were prominent in several disease categories including certain infectious diseases, pesticide exposures, occupational dermatoses, and lead levels in children.

Estimates of the size of the migrant and seasonal farmworker population vary widely. Basic health status indicators such as age-related death rates are unknown. Prevalence rates of the most common cause of death in the United States have yet to be studied. More research is needed into the health problems and health status of migrant and seasonal farmworker families. (*Am J Public Health* 1990; 80:1213–1217.)

Introduction

Migrant farmworker families are believed to experience poor health compared to the general population.[1] However, their health status has not been well measured, and many studies have not been published in peer reviewed journals. As a result, large gaps exist in the data on basic health status indicators in the migrant population. The contributing factors to this poor health status are even less well documented. I undertook a literature review to assess the state of our knowledge on migrant farmworker health.

Methods

MEDLINE files from 1966 through October 1989 were searched, using the subject headings of migrants and transients (US), health, agriculture, and agricultural workers' diseases; all articles relating to US populations containing the title word "migrant" or "farmworker" were also scanned. A total of 485 articles were identified through this search, and 203 were judged after further review to make specific reference to the health of migrants, farmworkers, or farmers in the United States.* The bibliographies of these articles were then scanned to identify any relevant articles missed by the computer search. In addition, I had access to one unpublished literature search undertaken for the Bureau of Health Care Delivery and Assistance (USPHS-DHHS) in 1984,[2] and a second literature search first published by the Farmworker Justice Fund in 1985[3] and revised in 1988.[4] The outcomes of these previous literature searches are dealt with in the discussion section of this paper.

Subject matter was divided into 12 categories, including both adult and pediatric health status, health care utilization patterns, health care delivery systems, and eight specific categories of illness by type of disease or organ system. Articles were also analyzed by year of publication.

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Results

Table 1 provides data on the outcome of the search. A further breakdown of the types of articles found by topic and by year of publication (Table 2) illustrates the changing focus of research in migrant health over the past 24 years. The early years were a time of interest in nutritional status, health care delivery systems, and obstacles to health care for the migrant farmworker population. Interest in the risk of pesticides and in the prevalence of infectious diseases showed evidence of continually increasing activity. The first article on AIDS (acquired immunodeficiency syndrome) and HIV (human immunodeficiency virus) seroprevalence in this population appeared in 1987.[5] No articles prior to 1980 dealt with specific psychiatric or behavioral illness patterns, but in the 1980s quantitative studies have begun to appear in the areas of family violence,[6] substance abuse,[7] and specific psychiatric symptomatology.[8]

Topics on which a significant volume of data were found include pesticide risk (including carcinogenic potential), infectious diseases (especially parasites, enteric pathogens, and tuberculosis), and health care delivery. The disciplines of clinical nutrition, dentistry and, to a lesser extent, pediatrics have displayed continuing interest in migrant workers' health.

As early as the 1960s, nutritionists were active in assessing the overall nutritional health status of adults,

TABLE 1—Overview of the Medical Literature in Migrant Health, 1966–89

Subject Area	Migrant-Specific Articles	Farm & Farm Labor (general)	Total
Health Status (Adult)	4	3	7
Health Status (Children)	14	—	14
Nutritional Status	10	—	10
Dental Health	18	—	18
Health Care Utilization	9	—	9
Health Care Delivery & Obstacles to Care	43	—	43
Psychiatric or Behavioral Conditions	7	—	7
Cardiovascular Disease	2	—	2
Infectious Disease	17	1	18
Cancer	—	12	12
Pesticides	22	23	45
Occupational Hazards	6	12	18
Total	152	51	203

TABLE 2—Subjects of Interest in Migrant Health Literature, by Year of Publication

Subject Area	Year of Publication 1966–73	1974–81	1982–89
Health Status (Adult)	2	0	5
Health Status (Children)	4	4	6
Nutritional Status	5	3	2
Dental Health	5	7	6
Health Care Utilization	0	5	4
Health Care Delivery & Obstacles to Care	23	14	6
Psychiatric or Behavioral Conditions	1	1	5
Cardiovascular Disease	2	0	0
Infectious Disease	3	5	10
Cancer	1	2	9
Pesticides	4	17	24
Occupational Hazards	4	5	9
Total	54	63	86

children, and pregnant women in the migrant farmworker population.[9] While under-nutrition (protein-calorie malnutrition, vitamin deficiency, and iron-deficiency anemia) was the focus of previous surveys,[10] future studies will be needed to assess areas of over-nutrition such as obesity and hyperlipidemia.

The dental profession has published many studies assessing the dental health of migrant children and adults using a community-based screening approach.[11]

The results of several community-based, health screenings of migrant children have been published.[12] While most of the studies published in the 1970s screened only for lead poisoning, the 1980s have seen some broader assessments. However, none have been performed on a large scale, with unbiased sampling methods and attention to differences among various ethnic groups as well as between the three US migrant streams. Table 3 lists a variety of basic questions on migrant health to which we find virtually no answers in the peer-reviewed medical literature. These are discussed in the following section.

TABLE 3—Unanswered Questions in Migrant Health

A. Population Characteristics
   1. Size of the Migrant and Seasonal Farmworker Population
   2. Age Distribution, Ethnicity, and Family Size of Farmworker Families in East, Central, and West Coast Migrant Streams
B. Mortality and Survival Data
   1. Median Survival
   2. Crude Death Rates
   3. Age Distributions (Mortality curve)
   4. Age-Specific Causes of Death
C. Peri-Natal Outcome Data
   1. Birthweight and Prematurity
   2. Neonatal vs Post-Neonatal Infant Mortality*
   3. Fetal wastage (Miscarriage & Stillbirth)
   4. Congenital Anomalies
   5. Maternal Morbidity/Mortality
D. Chronic Diseases
   1. Prevalence of Chronic Diseases
   2. Rates of Hospitalization or Bed-Disability Days
   3. Incidence of Various Types of Cancer
   4. Controllable Medical Risks for Cardiovascular Events
      a) Hypertension
      b) Diabetes
      c) Hyperlipidemia
      d) Smoking
      e) Previous Cardiac Ischemia or Angina
E. Occupational Risks
   1. Annual Highway Mileage (Risk of Motor Vehicle Accident)
   2. Pesticide Exposure (Frequency of Acute Poisonings and Effects of Chronic Low-Level Exposures)
   3. Compliance with Field Sanitation Laws
   4. Accidental Injury (Falls, Farm Machinery, etc.)
   5. Incidence of Occupational Dermatoses by Crop & Chemical Exposure
   6. Effects of Poor Housing
   7. Frequency & Severity of Heat & Cold Exposures
F. Nutritional Factors
   1. Obesity
   2. Serum Cholesterol and Lipid Profiles
   3. Protein/Calorie Malnutrition and Nutritional Anemias in New Migrant Sub-Populations (Haitian and Central American Refugees)
G. Health-Related Behaviors
   1. Tobacco Usage
   2. Alcohol Consumption
   3. Other Substance Abuse
   4. Seat-Belt Use
   5. Risk Factors for AIDS
   6. Suicide/Homicide and Family Violence
H. Accessibility to Health Care
   1. Utilization of Peri-Natal Care
   2. Immunization Status
   3. Treatment of Acute Illness Events (i.e., Strep pharyngitis)
   4. Utilization of Recommended Cancer Screening
   5. Delays in Diagnosis/Treatment Prior to Hospitalization

Discussion

The Missing Denominator—How Many Migrants Are There?

In migrant health we lack even such basic data as crude death rates, median survival, infant and maternal mortality, and incidence of permanent disability. One of the major difficulties in computing these is the lack of a precise denominator. We do not know how many migrants there are, because of the transient nature of the population, their migrations into and out of the US (both randomly and seasonally), undercounting of workers who meet the legal definition of a migrant but do not fit ethnic or demographic stereotypes, and the desire of many immigrant workers (documented or not) to avoid contact with government agencies.

The Office of Migrant Health estimates that there are three million "migrant and seasonal farmworkers and their dependents" in the United States.[13] Estimates of the number of "hired farmworkers" in 1985 from the National Agricultural Statistics Service varied from about 1 million (using one method of counting) to over 2.5 million (using a different method).[14] Their surveys were only able to come up with 159,000 "migrant farmworkers" in 1985, a number well below most other estimates.[15]

The Bureau of Labor Statistics asserts that "the size of the hired farm working force declined from an average of 3.6 million in the 1950s to 3.2 million in the 1960s and has been fairly steady at 2.7 million in recent years."[16] The same study notes a decline in the migrant farmworker population from 400,000 in the 1960s to 200,000 in the 1970s.

Several reasons exist for these discrepancies. The first is a question of defining terms. The numbers quoted above do not refer to the same population, but rather to various sub-groups. The Office of Migrant Health's definition is the broadest ("migrant and seasonal farmworkers and their dependents") and therefore gives the largest estimate. The Department of Labor and the Department of Agriculture surveys generally count only employed farmworkers over age 14 (each department using different methodologies).

There are no published estimates of the number of days

worked by migrant workers in a typical year, but with the common use of day-laborers the farm labor force may be expected to have a significant number of days without work. Methods such as those used by the Department of Labor that only count "hired farmworkers" at a given point in time may underestimate the true size of the farmworker population. Similarly, the methodology published by the Office of Migrant Health[17] based on crop acreage and the number of person-hours required to harvest a given crop would also be expected to undercount the farmworker population by an amount proportionate to the number of farmworkers unemployed during that harvest season.

In describing a study population it must always be noted whether a given population estimate is referring only to migrant farmworkers, or to migrant and seasonal farmworkers, or to a composite of farmworkers, crew chiefs, and farm managers. The estimate must also be examined for its inclusion or exclusion of family members and dependents, as well as unemployed farmworkers. Characteristics of the overall population will not necessarily be representative of workers in different geographic streams (East, West, and Central), of workers in different seasons (upstream and downstream) or on different crops, or of the various ethnic and other sub-populations of the farmworker population. Current estimates of the migrant population just do not have the needed accuracy or reliability to be used as denominators in the calculation of morbidity and mortality figures.

Mortality Data

In the study period (1966–89) there was no published study which effectively measured median age of survival, crude death rates, or age-specific mortality in the migrant farmworker population. A literature search commissioned by the Department of Health and Human Services in 1984 reviewed over 400 articles in 60 journals and uncovered only "sporadic documentation" of health problems, none of which included specific mortality or survival data.[2] The Farmworker Justice Fund undertook a similar literature review in 1985 and again in 1988 which documented a variety of occupational and lifestyle-associated hazards, but found no hard data on health indicators.[3,4]

Death certificate data in general has limitations,[18] but there are additional problems specific to migrant farmworkers. In no state is migrant status recorded on the death certificate, so deaths of agricultural workers would include seasonal and homebased farmworkers, thus diluting out the specific health effects of migratory lifestyle. Another problem is the "healthy worker effect," the exclusion of former farmworkers who have returned to their country of origin or who have become unemployed or disabled prior to their death. Members of the extended family, including children, who undoubtedly engage in farm labor may or may not be identified as having a primary occupation.

Perinatal Outcomes

A nutritional survey done by Chase, *et al,* in 1971 in Colorado derived from their maternal interviews an estimate of infant mortality among Mexican American farmworkers in Colorado that was nearly three times that of the general population (63 per 1,000).[19] A more recent study (1978) randomly surveyed 145 married women in migrant farmworker households in Wisconsin.[20] Of these, 132 women had given birth to a total of 629 children (excluding stillbirths). The reported infant mortality rate was 29 per 1,000, while early childhood deaths (age 0–5 years) occurred at a rate of 46 per 1,000. Cause of death was not reported. Large-scale maternal interview studies to define infant mortality in the farmworker population have not been undertaken.

Unfortunately, a large sample of perinatal outcome data from migrant health centers has never been analyzed for publication in the peer-reviewed literature. Migrant Health Centers have been estimated to reach approximately 17 percent (500,000) of the estimated 3 million who live in migrant and seasonal farmworker families.[13] Women receiving comprehensive perinatal care in a migrant health center might be expected to have lower infant mortality rates than those who receive no prenatal care prior to hospital delivery or those whose babies are delivered by a traditional birth attendant, but this has never been documented.

Occupational Risks

In 1987 agriculture surpassed mining as the nation's most hazardous occupation with 1,700 work-related deaths (52 per 100,000 workers). The US Bureau of Labor Statistics estimates that there are 12.7 cases of injury and illness per 100 fulltime workers per year.[21] Several reviews of the specific occupational hazards of agricultural labor are available.[3,22] Hazards include chronic and acute pesticide exposures, lack of safe drinking water and toilets, occupational dermatoses, acute injuries (falling from heights and farm machinery injuries), and chronic low-grade back and joint trauma. Most of these risks have been poorly quantified. One study of emergency room visits for farm equipment injuries suggests a methodology for future studies and monitoring of acute agricultural illnesses and injuries.[23]

The difficulty in quantifying pesticide exposures has been documented best in California, which has the nation's strongest mandatory reporting legislation. The state reported a 30 percent reduction in pesticide poisoning from 1985 to 1986, not from an actual reduction in risk but rather from a change in the criteria for documenting exposures.[24] The scant data that have been published on chronic or low-level pesticide exposure, suggest that limb-reduction birth defects,[25] childhood leukemias[26] and brain tumors,[27] adult lymphomas and lymphosarcomas[28] may be linked with occupational exposure to pesticides.

The Council on Scientific Affairs of the American Medical Association recently reviewed findings of the US Environmental Protection Agency and of the International Agency for Research on Cancer with regard to the carcinogenicity of agricultural chemicals. They report two of 53 chemicals to be *definitely* carcinogenic (arsenic and vinyl chloride). In addition they found 13 of 53 to be *probably* carcinogenic, and 16 of 53 *possibly* carcinogenic.[29] Sharp, *et al,* have also published an excellent review of the carcinogenic and neurotoxic effects of chronic low-level exposure to pesticides.[30]

Disability

A 1974 survey found that 44.5 percent of migrant farmworker households had one or more members who described themselves as disabled, but this study did not obtain clinical correlation of the degree or cause of the disability.[31] No survey of workers for the prevalence of disabling musculoskeletal conditions was found in this review.

Infectious Diseases

A recent study of 709 migrant farmworkers on the Delmarva Peninsula showed a high rate (37 percent) of PPD positivity (10 mm or greater) on TB skin testing, but found only one case of active tuberculosis,[32] suggesting that the

prevalence of tuberculosis among migrant farmworkers may have been overestimated in previous studies which relied on skin testing. The complete absence of articles on the most common infections seen in clinical practice, such as otitis media or streptococcal pharyngitis in children, was striking. Similarly the most common infectious causes of hospitalization among migrants in this author's experience—such as cellulitis, diabetic foot infections, pneumonia, and appendicitis—all are absent from the literature on migrant health.

Chronic Diseases and Premature Mortality

A basic problem with the predominance of research on conventional topics of migrant health—such as pesticides, TB, and parasites—is that it may give disproportionate attention to diseases that are more common among migrants, but still may not be the major cause of their mortality, e.g., cardiovascular disease and complications of diabetes.

There have been a plethora of health care utilization studies documenting the most common diagnoses among migrants seeking health care.[2] What all these studies tell us is that while some uncommon diseases are more prevalent among migrants, common diseases such as diabetes and hypertension in adults still lead the list. From the current literature base it is impossible to calculate the incidence, prevalence, or risk factors for cancer, heart disease, or stroke in the migrant farmworker population, even though these are the most common causes of death in the US.[33]

Socioeconomic Status

Clearly the migrant population is poor—the average migrant farmworker earned only $3,295 per year (1985) from farm labor, and income from all sources was only $6,194.[34] Few studies compared the health problems of farmworker families to other low-income minority groups rather than to the general population. An example of this more meaningful comparison is one which compared blood levels of migrant children to those of a population of urban poor children.[35]

Conclusions

Some progress has been made. Professionals in dentistry and clinical nutrition have made good baseline assessments of the health status of their clients and are now ready to move into on-going monitoring and intervention programs to improve dental and nutritional health status. In other areas we have only small foci of data, often in stereotyped migrant-specific disease categories. New areas such as psychiatric and behavioral morbidities are beginning to be explored, and methodologies are becoming more precise and quantitative. Such studies will need to be undertaken across a broad range of farmworker sub-populations to determine the extent to which results can be generalized to all farmworkers. Other basic indicators of health and disease, as well as the size of the total population and its sub-sets, remain totally unknown.

While services provided by migrant health centers may have improved the overall health of migrant farmworker families by improving their access to health care, there are very limited data with which to prove this point. Neither can it be proven whether the health problems of migrant farmworkers are the same as those of other groups suffering from poverty and powerlessness, or whether there are migrant farmworker-specific factors in their occupation and lifestyle which contribute to death or disease. The fields of undiscovered knowledge in migrant health are vast and ready for harvest. More research is needed.

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IVACG Releases Statement on Role of Vitamin A in Child Health and Survival

The XIII International Vitamin A Consultative Group (IVACG) Meeting in November 1989 reviewed the evidence available to date on the relationship of vitamin A depletion and deficiency to childhood morbidity and mortality. Results from completed studies as well as preliminary findings of others underway were presented and discussed by experts from several disciplines. The IVACG Steering Committee realized the need for an interim statement summarizing the weight of evidence available because results from several large, randomized masked trials are unlikely to be available for one to two years. The following statement, released in June 1990, represents the steering committee's careful consideration of the available evidence.

- An adequate vitamin A status prevents nutritional blindness and contributes significantly to child health and survival.
- The role of vitamin A in preventing nutritional blindness is well documented. Evidence is accumulating that it also reduces mortality. The mechanism(s) by which it has this effect is unclear.
- The impact of improved vitamin A nutrition will vary with the severity of vitamin A deficiency and the contributions of other ecological factors.
- It is therefore imperative to improve the diet and raise the level of vitamin A nutrition where a current intake is inadequate.

The IVACG Steering Committee hopes that this statement will be useful in the process of formulating policies and programs to control and combat vitamin A deficiency. The International Vitamin A Consultative Group was established in 1975 to guide international activities aimed at reducing vitamin A deficiency in the world. For further information, contact the IVACG Secretariat, The Nutrition Foundation, Inc., 1126 Sixteenth Street, NW, Washington, DC 20036. Tel: (202) 659-9024.

Paternal Military Service in Vietnam and the Risk of Late Adverse Pregnancy Outcomes

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Abstract: To investigate the relationship between paternal military service in Vietnam and the risk of late adverse pregnancy outcomes, we conducted a case-control study of women who delivered infants from August 1977 until March 1980 at Boston Hospital for Women. Paternal military service history among 857 congenital anomaly cases, 61 stillbirth cases, and 48 neonatal death cases were compared with that of 998 normal controls. Military service veterans were identified by crossmatching identifying information from obstetric records with state and national military records. After controlling for confounding variables, we found that the Vietnam veterans' relative risk of fathering an infant with one or more major malformations was 1.7 (95% CI = 0.8, 3.5) compared to non-Vietnam veterans. The increased risk was present in several organ systems and did not seem to be related to a particular type of defect. No associations or highly unstable associations were found between paternal military service in Vietnam and the occurrence of congenital anomalies overall, minor malformations, normal variants, stillbirths, and neonatal deaths. These findings should be viewed with caution since maternal and delivery characteristics appear to have contributed to the etiology of several of the major malformations among the Vietnam veterans' children. (*Am J Public Health* 1990;80:1218–1224.)

Introduction

Many Vietnam war veterans have expressed concern that their children are at increased risk of adverse health effects, particularly congenital anomalies, because of wartime exposure to the herbicide Agent Orange.[1–3] The results of studies published to date suggest that the overall risk of congenital anomalies is not greater among Vietnam veterans but that the risk of specific defects or the risk among specific subgroups may be increased.[4–7] We undertook the present case-control study to investigate the relation between paternal military service in Vietnam and a wide range of late adverse pregnancy outcomes including congenital anomalies, stillbirths, and neonatal deaths.

Methods

Study Population

The present analysis used subjects identified for a prior investigation on maternal risk factors for late adverse pregnancy outcomes.[8–9] In the previous study, we enrolled 14,130 obstetric patients who delivered infants at least 20 weeks' gestation from August 1977 until March 1980 at Boston (Massachusetts) Hospital for Women Division of Brigham and Women's Hospital. This represented 82.5 percent of all delivery patients during the study period; lack of personnel was the main reason for not being enrolled.

For the current study, we selected three case groups and one control group from the enrolled patients. The congenital anomaly case group consisted of 1,314 women who delivered live- or stillborn infants with one or more congenital anomalies. The stillbirth case group consisted of 121 women who delivered stillborn infants without anomalies, and the neonatal death case group was made up of 76 women who delivered infants without anomalies but who died shortly after birth. The 1,490 controls were randomly selected from the remaining women who delivered infants without anomalies who were alive upon discharge to achieve an approximate 1:1 case-control allocation ratio.

The original study interview obtained information on maternal demographic characteristics, medical and obstetric conditions, and habits. In addition, the medical records were reviewed to gather information on the delivery and infant characteristics. For the current study, we re-reviewed the medical records for identifying paternal information not previously obtained and for more detailed information on the pregnancy outcome. For women with stillbirths and neonatal deaths, we obtained the underlying cause of death. For women delivering infants with congenital anomalies, we recorded up to five anomaly diagnoses using a modification of International Classification of Diseases 9th Edition developed by the Centers for Disease Control (CDC) (unpublished coding manual). Anomaly coding decisions followed CDC guidelines and were reviewed for accuracy by a dysmorphologist. In addition, follow-up information was sought from pediatricians and hospitals to confirm the cardiovascular defects. Review of medical records and coding of anomalies were done before military service history was determined.

The medical records of 2,599 women (86.6 percent) were located for re-review. Both cases and controls whose records were lost were more likely to be Black, less educated, and younger than women whose records were reviewed. In addition, 507 women whose records were reviewed were unmarried. Among 84.0 percent of the unmarried women as well as 18 married women, we found no identifying paternal information in their medical records.

In the current study, we excluded women whose records were not located for review, unmarried women, married women without identifying paternal information and women with various diagnoses, and the second pregnancy of those with more than one pregnancy during the study period (see Appendix A for details). This left 857 congenital anomaly cases, 61 stillbirth cases, 48 neonatal death cases, and 998 controls for the final analysis.

Military Service Information

We identified military veterans by crossmatching identifying paternal information from the medical record with: the Massachusetts veterans' bonus list and Massachusetts military records, and the military records at the National Personnel Records Center (NPRC) Military Records Division in St. Louis, Missouri. The identification process and review of

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Massachusetts and NPRC records were performed without knowledge of case or control status of subjects.

Potential Massachusetts veterans were identified by manually searching the bonus list of 250,000 for husbands' names. Potential matches were confirmed by reviewing more complete identifying information in individual military discharge papers. The discharge papers also provided information on the branch, rank, place and dates of service.

Under the Freedom of Information Act, we were able to obtain information on branch, last rank, dates of active service and, if applicable, dates, first rank, and occupational specialty for Vietnam service of all military veterans in our study population. We were denied information on the exact geographic locations of Vietnam service.

In addition to names, Social Security numbers, needed to identify veterans in national records, were obtained from the bonus list for identified Massachusetts veterans and from Massachusetts Registry of Motor Vehicle drivers' license records for the remainder. We located Social Security numbers for 1,641 husbands (83.9% of case husbands and 83.2% of control husbands). Those without numbers were more likely to be Black and born in a foreign country and were included only in the Massachusetts bonus list review.

Analysis

We performed three analyses basing military service history on: 1) the Massachusetts veterans' bonus list alone; 2) national records alone; and 3) both record sources combined. Military service history was categorized as: 1) Vietnam veteran; 2) non-Vietnam veteran; and 3) no known military service.

Husbands were classified as Vietnam veterans if they received a $300 bonus and/or their military records noted the receipt of a Vietnam service medal or listed Vietnam as a place of foreign service. For US Navy personnel, Vietnam veterans were identified by linking individual ship assignments to Naval ship location records. Husbands were classified as non-Vietnam veterans if they received a $200 bonus and/or their military records contained no information on Vietnam service. Husbands were classified as having no known military service if they were not found on the Massachusetts bonus list or if they had no identifiable military records nationally. A national military record was identified but could not be reviewed for four individuals. These husbands were considered veterans with unknown place of service and, when appropriate, were excluded from the analysis.

In the analysis that combined military service information from both Massachusetts and national records, husbands identified as Vietnam veterans in either or both sources were classified as Vietnam veterans. Husbands identified as non-Vietnam veterans in both sources or as a non-Vietnam veteran in one source and as a non-veteran in the other source were classified as non-Vietnam veterans. Husbands were classified as having no known military service if they were classified as such according to both record sources.

To identify Vietnam veterans who might be at increased risk of adverse pregnancy outcomes because of possible exposure to phenoxy herbicides during combat, Vietnam veterans were classified as having probable combat experience on the basis of their branch and occupational specialty by the Director of the Massachusetts Agent Orange Program without knowledge of case or control status of subjects.

We calculated the exposure odds ratio (relative risk) to measure the strength of association between paternal Vietnam service and each type of adverse pregnancy outcome. The odds of each adverse outcome among Vietnam veterans and non-Vietnam veterans was compared to that among men with no known military service. In addition, the odds of each pregnancy outcome among Vietnam veterans were compared to that among non-Vietnam veterans to control for confounding by factors related to entry into military service. Ninety-five percent confidence intervals (CI) for the crude odds ratios were computed using Miettinen's test-based method[10] if there were more than five exposed cases and Fisher's exact method[11] if there were five or fewer exposed cases.

We examined paternal military service history in each case group and then divided the congenital anomaly cases into several subgroups. We categorized anomaly cases according to severity: 1) one or more major malformations; 2) only minor malformations; or 3) only normal variants using a scheme developed by the Metropolitan Atlanta Congenital Defects Program (unpublished procedure manual). Anomalies were also categorized into 13 organ system groups and into specific diagnostic categories. Infants with more than one anomaly diagnosis could contribute to more than one organ system or diagnostic group.

We assessed confounding by entering 44 potential confounding variables into a logistic regression model one at a time and by comparing the adjusted and crude odds ratios[12] (see Appendix B for details). In the regression analyses, the dependent variables included all congenital anomalies, major malformations, minor malformations, and normal variants. Because the number of exposed subjects was small, no adjusted analyses were performed on the stillbirth, neonatal death, and most organ system case groups.

Final logistic regression models were developed for the anomaly cases and severity subgroups that adjusted for several variables simultaneously. The final models included variables that had altered the crude odds ratios by at least 10 percent as well as the following variables of a priori interest: maternal race, educational level, average alcohol consumption during the first trimester, prior infant with a congenital anomaly, and year of pregnancy.

Results

Table 1 displays military service history based on the combined Massachusetts and national data among husbands of cases and controls, and the crude odds ratios of each adverse pregnancy outcome. Compared to men with no known military service, Vietnam veterans, and, to a lesser extent, non-Vietnam veterans had an increased risk of fathering infants with one or more major malformations (ORs = 1.8 and 1.4, respectively). The relative risk of major malformations among Vietnam veterans fell to 1.3 when they were compared to non-Vietnam veterans (95% CI = 0.7, 2.4).

No material increases in the crude relative risks of minor malformations, normal variants, or neonatal deaths were observed among the military service groups. Vietnam veterans had an increased risk of stillbirths but confidence intervals are very wide. The underlying causes of death among stillbirths did not differ among the military service groups.

Most results were similar when military service history was based either on the Massachusetts bonus list or NPRC records alone. However, the relative risks of major malformations and stillbirths fluctuated according to information source. These variations may be accounted for by the small number of exposed cases (data available from author).

Compared to non-veterans, the crude relative risks of defects from several organ systems were elevated both among Vietnam veterans and non-Vietnam veterans (Table 2). The largest increases in risk among Vietnam veterans

TABLE 1—Military Service History, Crude Odds Ratios and 95% Confidence Intervals for All Congenital Anomalies, Congenital Anomaly Severity Subgroups, Stillbirths, and Neonatal Deaths

	Military Service History*			Odds Ratios** Comparing		
	Number of Vietnam Veterans	Number of Non-Vietnam Veterans	Number of Men with No Known Military Service	Vietnam Veterans to Men with No Known Service	Non-Vietnam Veterans to Men with No Known Service	Vietnam Veterans to Non-Vietnam Veterans
All Congenital Anomalies (N = 857)	55	146	656	1.3 (0.9, 1.9)	1.0 (0.8, 1.3)	1.2 (0.8, 1.9)
One or More Major Malformations (N = 214)	18	45	151	1.8 (1.0, 3.1)	1.4 (1.0, 2.0)	1.3 (0.7, 2.4)
Only Minor Malformations (N = 232)	11	32	189	0.9 (0.5, 1.7)	0.8 (0.5, 1.2)	1.1 (0.5, 2.4)
Only Normal Variants (N = 411)	26	69	316	1.2 (0.8–2.0)	1.0 (0.7–1.4)	1.2 (0.7–2.1)
Stillbirths (N = 61)	5	5	51	1.5 (0.4–3.9)	0.5 (0.1–1.2)	3.2 (0.7–14.5)
Neonatal Deaths (N = 48)	3	9	36	1.2 (0.2–4.2)	1.2 (0.6–2.5)	1.1 (0.2–4.5)

*Military service history was based on the combined Massachusetts and NPRC data.
**Odds ratios were computed by comparing paternal military service history of each case group to that of the 995 controls; 52 control husbands were Vietnam veterans, 167 were non-Vietnam veterans, and 776 had no known military service. Three control husbands who were identified as veterans but whose place of service was unknown were excluded.

TABLE 2—Military Service History and Crude Odds Ratios According to Major Malformation Organ System

	Military Service History*			Odds Ratio# Comparing		
Organ System	Number of Vietnam Veterans	Number of Non-Vietnam Veterans	Number of Men with No Known Military Service	Vietnam Veterans to Men with No Known Service	Non-Vietnam Veterans to Men with No Known Service	Vietnam Veterans to Non-Vietnam Veterans
Nervous System (N = 24)	3	3	18	2.5	0.8	3.2
Eye (N = 2)	0	0	2	0.0	0.0	0.0
Ear, Face, Neck (N = 1)	0	0	1	0.0	0.0	0.0
Cardiovascular (N = 27)†	4	7	16	3.7	2.0	1.8
Respiratory (N = 14)	0	4	10	0.0	1.9	0.0
Gastrointestinal (N = 22)	1	4	17	0.9	1.1	0.8
Genital (N = 16)	2	4	10	3.0	1.9	1.6
Urinary Tract (N = 13)	1	4	8	1.9	2.3	0.8
Musculoskeletal (N = 98)	7	21	70	1.5	1.4	1.1
Endocrine (N = 2)	1	1	0	∞	∞	3.2
Autosomal Chromosome Defect (N = 17)	1	3	13	1.2	1.1	1.1
Congenital Syndrome (N = 7)	0	2	5	0.0	1.9	0.0

*Military service history was based on the combined Massachusetts and NPRC data.
#All confidence intervals for the odds ratios include 1.0.
†Cardiovascular defects among infants with Trisomy 21 are excluded.

were for malformations of the nervous system, cardiovascular system, genital organs, and urinary tract.

The organ system results were similar but somewhat attenuated when Vietnam veterans were compared to non-Vietnam veterans. The organ system risk estimates should be interpreted cautiously since they were based on a small number of exposed subjects.

When we examined the specific anomaly diagnoses and clinical notes among the 18 major malformation cases whose husbands served in Vietnam (Appendix C), we found that no one diagnosis predominated within an organ system. In fact, only two defects were observed more than once: two infants had a ventricular septal defect, and three infants had talipes (clubfoot).

Maternal and delivery characteristics appeared to be important contributory factors in the etiology of some malformations. Pediatricians consider that the congenital dislocation of hip and clubfoot were secondary to fetal position; both the mother and prior sibling of the case of adrenal hypoplasia had a history of adrenal insufficiency. None of the medical records of these major malformation cases noted that the husband was a Vietnam veteran.

We also examined military service history among five diagnostic categories of skin and related anomalies: abnormal palmar creases (N = 24), sacral dimples (N = 78), hemangiomas (N = 83), benign neoplasms of the skin (N = 25), and other specified skin anomalies (N = 152). The crude relative risks of abnormal palmar creases and sacral dimples were

increased among Vietnam veterans when compared either to non-veterans or non-Vietnam veterans (OR 3.7, 95% CI = 0.8, 12.1 and OR 2.5, 95% CI = 1.2, 5.3, respectively, for the comparison between Vietnam veterans and non-veterans; OR 3.2, 95% CI = 0.6, 17.8 and OR 1.8, 95% CI = 0.8, 4.3, respectively, for the comparison between Vietnam veterans and non-Vietnam veterans). The remaining categories of skin anomalies were not materially elevated among Vietnam veterans nor were any skin anomalies materially increased among non-Vietnam veterans. Because normal variants like abnormal palmar creases can be markers for more serious defects, we reanalyzed these data excluding infants with major malformations. Upon reanalysis, the relative risks of palmar creases and sacral dimples among Vietnam veterans remained elevated.

In Table 3 we have presented the military service characteristics of Vietnam veteran case and control husbands. A greater proportion of congenital anomaly and major malformation case husbands versus control husbands served in the Marine Corps and had longer durations of Vietnam service. Anomaly case husbands were also more likely to have enlisted and have had initial occupational specialties in Vietnam likely to involve combat.

Because Vietnam veterans who were in combat had a greater likelihood for herbicide exposure than veterans who were not,[13] we recalculated the odds ratios for all congenital anomalies and the severity subgroups considering as exposed only Vietnam veterans with probable combat experience. Compared to non-Vietnam veterans, the relative risks of congenital anomalies overall, one or more major malformations, only minor malformations, and only normal variants among Vietnam veterans with likely combat exposure were similar to those risks among all Vietnam veterans (1.4, 1.1, 1.4, and 1.5, respectively). The number of Vietnam veterans with probable combat exposure among the stillbirth and neonatal death case groups was too small to permit meaningful analysis.

Many available characteristics of the study population were potential confounders because of their association with paternal military service history and a particular pregnancy outcome. When we adjusted simultaneously for confounding variables using logistic regression models, we found that the relative risk of one or more major malformations rose to 2.2 for the comparison between Vietnam veterans and non-veterans (crude OR 1.8) and to 1.7 for the comparison between Vietnam veterans and non-Vietnam veterans (crude OR 1.3) (Table 4). The adjusted relative risks for congenital anomalies overall and the other severity subgroups differed little from the crude relative risks. Confidence intervals are again very wide.

Discussion

In our study population, we have observed positive associations between paternal military service in Vietnam and the risk of fathering an infant with one or more major malformations even after controlling for available confounding variables. The increased risk was present in several organ systems and did not seem to be related to a particular type of defect. We also found positive associations between two normal variants of the skin and Vietnam service. No associations or highly unstable associations were observed between Vietnam service and congenital anomalies overall, minor malformations, normal variants, stillbirths, and neonatal deaths.

The positive findings warrant cautious interpretation since maternal and delivery characteristics appeared to be important factors in the etiology of the endocrine and some of the musculoskeletal malformations observed among the children of the Vietnam veterans. In addition, most risk estimates for the organ systems were unstable because they were based on a small number of subjects.

One might also argue that cautious interpretation is needed because of the wide variety of major malformations seen among the Vietnam veterans' children. However, the etiologic implications of the lack of a predominant defect or discernible pattern of defects are unclear, because so little is known about the possible biologic mechanisms by which paternal exposures might influence pregnancy outcomes and because many well-known maternal exposures have been associated with a wide range of defects (e.g. maternal prenatal ingestion of thalidomide has been associated with limb, ear, eye, heart, gastrointestinal tract, and genital organ defects).[14]

TABLE 3—Distribution (%) of Paternal Military Service* Characteristics among Vietnam Veterans According to Outcome

Characteristics**	Congenital Anomaly Cases (N = 55)	1+ Major Malformation Cases (N = 18)	Still-Birth Cases (N = 5)	Neonatal Death Cases (N = 3)	Controls (N = 52)
Military Branch					
Army	49.1	50.0	60.0	66.7	50.0
Navy	18.2	16.7	40.0	0.0	25.0
Air Force	10.9	11.1	0.0	33.3	15.4
Marines	21.8	22.2	0.0	0.0	9.6
Year Started Vietnam Service					
1964–1966	8.2	7.6	20.0	0.0	10.2
1967–1969	71.4	61.5	40.0	100.0	61.2
1970–1972	20.4	30.8	40.0	0.0	28.6
Duration of Vietnam Service (months)					
1–6	18.8	8.3	20.0	0.0	25.5
7–12	56.3	58.3	40.0	100.0	59.6
13–27	25.0	33.4	40.0	0.0	14.9
First Rank in Vietnam					
Enlisted	83.6	77.8	80.0	66.6	76.9
Officer	16.4	22.2	20.0	33.3	23.1
Probable Combat Exposure	56.4	44.4	20.0	66.7	50.0

*Military service history was based on the combined Massachusetts and NPRC data.
**Individuals with unknown values were excluded.

TABLE 4—Adjusted Odds Ratios and 95% Confidence Intervals for Late Adverse Pregnancy Outcomes According to Military Service History

	Adjusted Odds Ratio	95% Confidence Interval
Vietnam Veterans Compared to Non-Veterans		
All Congenital Anomalies	1.2*	0.8, 1.8
1+ Major Malformations	2.2**	1.2, 4.0
Only Minor Malformations	0.7***	0.3, 1.5
Only Normal Variants	1.1*	0.7, 1.9
Stillbirths	1.5‡	0.6, 3.8
Neonatal Deaths	1.2‡	0.4, 4.2
Non-Vietnam Veterans Compared to Non-Veterans		
All Congenital Anomalies	1.1*	0.8, 1.4
1+ Major Malformations	1.4*	0.9, 2.2
Only Minor Malformations	0.7†	0.5, 1.2
Only Normal Variants	1.1*	0.8, 1.5
Stillbirths	0.5‡	0.2, 1.1
Neonatal Deaths	1.2‡	0.6, 2.5
Vietnam Veterans Compared to Non-Vietnam Veterans		
All Congenital Anomalies	1.1*	0.7, 1.8
1+ Major Malformations	1.7††	0.8, 3.5
Only Minor Malformations	0.9†††	0.4, 2.3
Only Normal Variants	1.0*	0.6, 1.8
Stillbirths	3.2‡	0.7, 14.5
Neonatal Deaths	1.1‡	0.2, 4.5

*Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy.

**Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy, and parity.

***Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy, and employment status at delivery.

†Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy, and prenatal urinary tract infection.

††Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy, prior use of an intrauterine contraceptive device, and month prenatal care began.

†††Adjusted for maternal race, educational level, alcohol use during the first trimester, history of another child with an anomaly, year of pregnancy, employment status at delivery, prenatal urinary tract infection, DES exposure, and cigarette and marijuana smoking during pregnancy.

‡Unadjusted results.

In the current investigation, congenital anomaly diagnoses were made usually within a few days of birth by pediatric residents who were not specialists in teratology. Thus, it is quite likely that some anomalies were missed. We believe that this misclassification was not related to military service history and so probably biased some results towards the null.

We believe it is unlikely that the positive association observed for major malformations was due to more complete ascertainment of these congenital anomalies among the children of Vietnam veterans. Anecdotal reports of adverse reproductive outcomes among Vietnam veterans potentially exposed to Agent Orange began appearing in the medical literature in late 1979 after most of the study pregnancies had occurred.[1–3] Most of these malformations had very noticeable cosmetic or medical effects and many were life threatening. Thus, it is unlikely that the diagnosis or notation of a defect would be affected by the father's military service history. Furthermore, none of the medical records of the major malformation cases noted that the husband was a Vietnam veteran.

We also regard it as unlikely that these results simply reflect a differential tendency for Vietnam veteran versus non-Vietnam or non-veteran cases and controls to have participated in the original study. For practical reasons, enrollment in the current study was limited to married women whose records contained identifying paternal information. Enrolled women were predominantly White and college educated while the excluded women were more likely to be Black and less educated. Because the demographic characteristics of both excluded cases and controls were similar, it is unlikely that these differences led to bias. However, generalizations from this study should be limited to veterans who are demographically similar to the enrolled population.

Observation bias was improbable because identification of veterans was done blindly using military records. To control potential confounding, we excluded high-risk women and, when possible, used multivariate techniques to adjust for other variables. In addition, we compared Vietnam veterans to other veterans in order to control for factors related to entry into military service. However, since we lacked specific data on paternal exposures and conditions, it is possible that confounding was not completely controlled.

Studies have been conducted among North Vietnamese military veterans who were possibly exposed during the Vietnam war.[15,16] Positive associations were generally found but their validity is difficult to assess since they have never been published but have only been described briefly by scientists who attended the 1983 International Symposium on the Effects of Phenoxy Herbicides.

In contrast, two case-control studies conducted among Australian Army and US Vietnam veterans found no overall increase in the risk of major malformations (ORs 1.03 and 0.97, respectively).[4,5] The Centers for Disease Control study found that US Vietnam veterans with greater estimated opportunities for Agent Orange exposure had stable increases in the risk of fathering infants with spina bifida (but not anencephaly), cleft lip with or without cleft palate, congenital neoplasms, and

coloboma.[5] The Australian study found that Army veterans had unstable increases in the risk of fathering children with ventricular septal defects and Down's Syndrome.[4]

Preliminary results from a retrospective cohort study among the US Air Force personnel who conducted the aerial herbicide spraying missions in Vietnam revealed an elevation in the risk of fathering infants with major and minor birth defects combined (OR: 1.3) that was due primarily to minor skin anomalies. Unlike our study, the skin anomalies reported among the Air Force children were mainly birthmarks and other skin discolorations. Investigators also observed no increase in the risk of stillbirths but a 3.8-fold increase in the risk of neonatal deaths. Recall bias cannot be ruled out in the Air Force data since they were based solely on interviews; collection of birth records to verify these associations is currently underway.[6]

Another US study further examined reproductive and other health outcomes among a cohort of Army veterans. At interview Vietnam veterans were more likely to report that they fathered a child with a birth defect than were non-Vietnam veterans (OR: 1.3, 95% CI:1.2, 1.4). In contrast, a sub-study using only diagnoses recorded in hospital records found similar rates of major, minor and "suspected" birth defects among Vietnam and non-Vietnam veterans (OR's: 1.1, 1.0, 0.9, respectively).[7]

In summary, current and previous study results are not in agreement concerning the risk of major malformations among Vietnam veterans. No consistent pattern of defects has emerged. We conclude that large studies, preferably with individual herbicide exposure data, would be needed to clarify the risk of late adverse pregnancy outcomes among Vietnam veterans overall as well as the subgroup of veterans who were exposed to Agent Orange.

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APPENDIX A

Selection of Cases and Controls for Study of Paternal Vietnam Service and the Occurrence of Late Adverse Pregnancy Outcomes

	Number of Congenital Anomaly Cases	Number of Stillbirth Cases	Number of Neonatal Death Cases	Number of Controls
Selected	1,314	121	76	1,490
Medical Record Reviewed*	1,159	87	66	1,287
Exclusions:				
Unmarried woman**	253	20	14	238
Diabetes	29	4	—	27
Epilepsy	4	3	2	7
Drug Abuse	6	—	—	—
Herpes, Toxoplasmosis, Rubella Infection	10	—	2	12
Second Pregnancy During Study Period	16	—	1	15
Total Number Excluded***	302	26	18	289
Total Number in Vietnam Service Study	857	61	48	998

*These figures also reflect changes in outcome status after record review and follow-up.

**Unmarried women are single, separated, divorced, or widowed. In addition, 18 married subjects whose husbands' names were not recorded in the medical record were excluded here.

***Since some subjects fell into more than one exclusion category, the total number of women excluded is less than the sum of women in each exclusion category.

APPENDIX B

Potential Confounding Variables Considered in Analysis

Paternal age
Maternal age
Paternal educational level
Maternal educational level
Paternal race
Maternal race
Paternal birthplace
Maternal birthplace
Paternal employment status at delivery
Maternal employment status at delivery
Medical insurance
Religion
Maternal history of prior oral contraceptive use
Maternal history of prior IUD use
Intent to become pregnant
Trimester prenatal care started
Maternal history of hypertension
Maternal history of cervical incompetence
Maternal history of pelvic inflammatory disease
Maternal history of gonorrhea
Maternal history of DES exposure
Maternal history of gynecologic surgery
Maternal history of other chronic medical conditions
Prenatal pre-eclampsia
Prenatal hypertension
Prenatal urinary tract infection
Gravidity
Parity
Number of prior livebirths
Number of prior pregnancy losses
Number of prior induced abortions
Number of prior ectopic pregnancies
Maternal history of cigarette smoking
Average number of cigarettes smoked per day during first trimester
Maternal history of alcohol intake
Average number of alcoholic drinks consumed per week during first trimester
Maternal marijuana use during pregnancy
Maternal coffee consumption during first trimester
Maternal tea consumption during first trimester
Maternal use of nausea medication during pregnancy
Maternal use of prescription drugs during first trimester
Family history of a prior malformed child
Gender of infant
Calendar year of pregnancy

APPENDIX C

Congenital Anomaly Diagnoses among Major Malformation Cases Whose Husbands Served in Vietnam

Case Number	*Anomaly Diagnoses Coded* (Relevant Clinical Notes)
1	Congenital hydrocephalus (secondary to intraventricular hemorrhage)
2+	Reduction deformity of brain, cleft palate without cleft lip, perineal hypospadias
3	Encephalocele, rockerbottom feet*
4	Probable ventricular septal defect (too early to tell for certain, murmur, tachypnea, tachycardia, acrocyanosis)
5	Ventricular septal defect, undescended testes,* flame nevi*
6+	Patent ductus arteriosus, patent foramen ovale, simian crease* (congestive heart failure, intracranial hemorrhage, pulmonary artery hypertension)
7	Transposition of the great vessels
8	Congenital dislocation of the hip (breech presentation)
9	Talipes equinovarus (severe tibial torsion)
10	Talipes equinovarus
11	Talipes calcaneovalgus (secondary to fetal position)
12	Hypoplasia of mandible and zygomatic bone
13	Arthrogryposis (30° contracture of elbows, 15° contracture of knees)
14	Split hand deformity
15	Congenital hydronephrosis
16	Ovarian cysts
17	Congenital adrenal hypoplasia (adrenal insufficiency in mother and sibling)
18	Trisomy 21 (membraneous ventricular septal defect)

*These diagnoses were classified as minor malformations or normal variants.
+Died

Lyme Disease in New Jersey Outdoor Workers: A Statewide Survey of Seroprevalence and Tick Exposure

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Abstract: To evaluate the spread of Lyme disease in New Jersey, we conducted a statewide cross-sectional study of Lyme disease seroprevalence in a high-risk occupational group of outdoor employees. Of the 689 employees who participated in the study, 39 (5.7 percent) were positive for antibody to *B. burgdorferi*, the causative agent of Lyme disease. Seroprevalence varied markedly by county; unexpectedly high seroprevalence rates were found in several northern counties (Sussex, Hudson, and Hunterdon). Furthermore, some southern counties (Atlantic, Cape May, and Ocean) with large tick populations (as measured by self-reported exposure to ticks) had low seroprevalence rates which were inversely correlated with self-reported preventive practices. These data suggest that lyme disease, as measured by seroprevalence of antibody to *B. burgdorferi*, may be spreading beyond the southern portion of the state where it had been previously well documented and that preventive behaviors may play an important role in minimizing the risk of the disease. (*Am J Public Health* 1990; 80:1225–1229.)

Introduction

The incidence of Lyme disease is increasing in many areas of the United States. The disease is caused by the spirochete *Borrelia burgdorferi* and transmitted by the *Ixodid* ticks. Early reports of the disease in the United States were from Connecticut in the late 1970s[1] but the disease is now a public health concern in the surrounding states (New Jersey, New York, and Massachusetts) as well, and has been reported in at least 43 states.[2]

A series of investigations in New Jersey were performed during the period 1978–82 by Bowen and Schulze, *et al.*[3–7] They found that, of 117 reported Lyme disease cases during the period, 48 percent (57) had occurred in one centrally located county (Monmouth). Almost all cases (98 percent) had occurred in the central and southern portions of the state.[5] Furthermore, the statewide distribution of *Ixodes dammini* correlated with the location of reported cases and revealed a high density of *Ixodes dammini* in the southern two-thirds of the state and a particularly high concentration in Monmouth County. It should be noted that while *Ixodes dammini* is the primary vector of Lyme disease in the northeast United States, the causative agent, *B. burgdorferi* has been identified in a number of species of ticks including *Amblyomma americanum*, and *Dermacentor variabilis*.[8]

In an attempt to further evaluate the spread of Lyme disease in New Jersey, we conducted a statewide cross-sectional study of Lyme disease seroprevalence in a high-risk occupational group. We measured: 1) seroprevalence of antibody to *B. burgdorferi*, 2) self-reported tick exposure and, 3) self-reported preventive practices among outdoor workers in the Natural and Historic Resources divisions of the New Jersey Department of Environmental Protection.

Methods

Study Population

The New Jersey Natural and Historic Resources (NHR) Section consists of four divisions: Parks and Forestry; Coastal Resources; Green Acres; and Fish, Game and Wildlife. Employees work primarily in outdoor jobs in a variety of habitats including mountainous regions, grass lands, forests, and shoreline. Geographically, they are distributed across the state from Stokes State Forest on the state line with New York in the north to Cape May County across the bay from Delaware in the south. During October 1988, we conducted a Lyme disease evaluation of employees at 12 different sites across the state on 15 dates. The summer months of May through August are the highest risk months and antibody seroconversion generally occurs three to six weeks after infection.[9] Participation was voluntary and all participating employees gave informed consent. A total of 689 (69 percent) of an estimated 1,000 eligible employees participated in the study.

Data Collection

The evaluation consisted of venipuncture for a serum specimen and administration of a questionnaire. Whole blood specimens were centrifuged and then divided into as many as five equal aliquots. An initial fresh specimen was sent to a commercial laboratory for immunofluorescent antibody (IFA) titer to *B. burgdorferi*. A titer of 1:128 or greater was reported as positive by the laboratory. The remaining samples were frozen and then after the initial fresh specimen results were reported, all positives and a random sample of the negatives were sent to as many as three other laboratories for analysis. Samples were analyzed at one department of health laboratory and at two university research laboratories by IFA in one case and enzyme linked immunosorbent assay (ELISA) in the other two. To be defined as positive for antibody to *Borrelia burgdorferi* in the study, the worker had to have at least two positive tests in two independent laboratories.

All employees participating in the study completed a self-administered questionnaire. The administration of the questionnaire was supervised by the authors and checked for accuracy after completion. Study variables included demographic data, counties of work and residence, self-reported measures of tick exposure, and frequency of performance of six behavioral practices of potential benefit in minimizing the risk of *B. burgdorferi* infection.

For purposes of data analysis and description, the counties of the state have been defined as northern or southern based on a line which cuts New Jersey geographically in half. The line runs from the lower border of Mercer

From the Center for Occupational Medicine, New Jersey Department of Environmental Protection Trenton (Goldstein, Maccarillo, Borbi, Tuccillo); the Clinical Epidemiology Unit, Section of General Internal Medicine, University of Pennsylvania, Philadelphia (Schwartz); and the Occupational Medicine Program, Columbia University School of Public Health, New York City (Friedmann). This paper, submitted to the Journal October 11, 1989, was revised and accepted for publication April 2, 1990.

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County through the middle of Monmouth County (Figure 1). Since the dividing line runs through Monmouth County, and because that county is unique in that it has a particularly well documented history of elevated Lyme disease risk, Monmouth will be discussed separately and not included with the northern or southern groupings.

Results

Seroprevalence

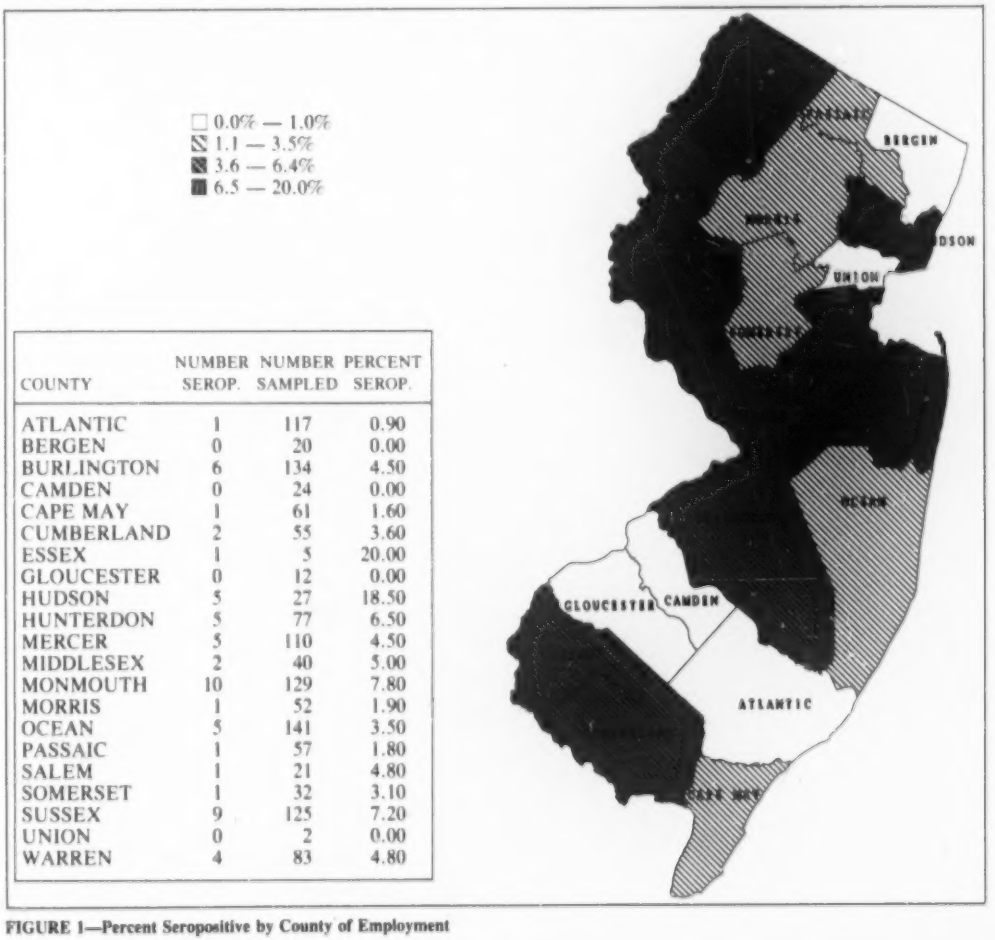
Statewide: A total of 689 employees of the NHR divisions of the NJ Department of Environmental Protection participated in the study. Thirty-nine employees (5.7 percent) had at least two positive tests for antibody to *B. burgdorferi*, the causative agent of Lyme disease. There were no differences in seropositivity based on years of outdoor state employment. The 39 seropositive subjects were found to have a mean (SD) of 16.5 (28.9) years of outdoor state employment while the seronegatives had a mean (SD) of 19.0 (28.0) years. There were no important differences based on job title or primary job habitat.

Geographic Distribution: Figure 1 displays the distribution of seropositives based upon county(s) of employment (some employees worked in more than one county on a regular basis). Seroprevalence ranged from a low of zero percent in four counties (Bergen, Camden, Gloucester, and Union) to highs of 6.5, 7.2, 7.8, and 18.5 percent in Hunterdon, Sussex, Monmouth, and Hudson counties, respectively. The 20 percent seroprevalence found in Essex County is based upon only five workers sampled.

Self-Reported Tick Exposure

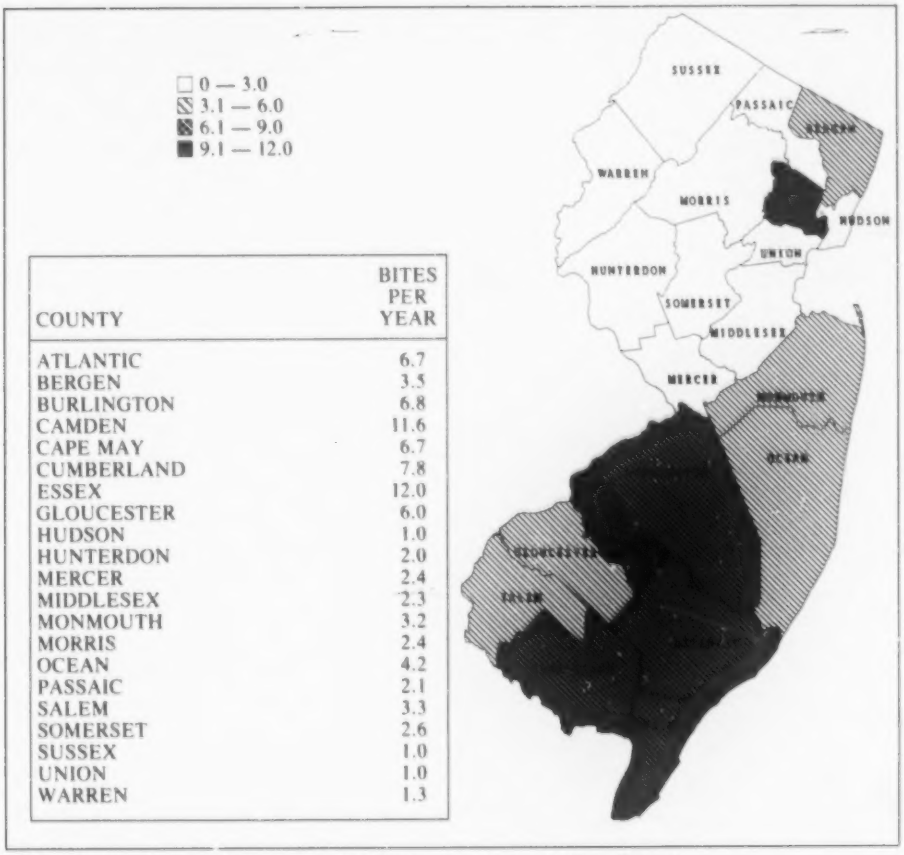
Statewide: Based on self-reports from the 689 employees who participated in the study, the workers reported a mean (SD) of 3.5 (7.6) and a median of 1.0 tick bites (ticks embedded in the skin) during the past year. The number of ticks removed from skin or clothing per week (during May–August 1988) on the primary job (some employees had other non-DEP jobs) was reported as a mean of 2.5 (SD ± 5.4) and a median of 2.0

Geographic Distribution: Figure 2 displays the mean number of tick bites over the past 12 months by county of employment. The mean yearly bite totals ranged from less than 1.5 in Hudson, Sussex, Union, and Warren counties to greater than 7.5 in Essex, Cumberland, and Camden counties. The mean number of ticks removed from skin or clothing per week on the primary state job by county of employment ranged from less than 1.5 ticks per week in Bergen, Passaic, Sussex, and Union counties to greater than 4.0 ticks per week in Burlington, Cape May, Cumberland, and Salem counties.



COUNTY	NUMBER SEROP.	NUMBER SAMPLED	PERCENT SEROP.
ATLANTIC	1	117	0.90
BERGEN	0	20	0.00
BURLINGTON	6	134	4.50
CAMDEN	0	24	0.00
CAPE MAY	1	61	1.60
CUMBERLAND	2	55	3.60
ESSEX	1	5	20.00
GLOUCESTER	0	12	0.00
HUDSON	5	27	18.50
HUNTERDON	5	77	6.50
MERCER	5	110	4.50
MIDDLESEX	2	40	5.00
MONMOUTH	10	129	7.80
MORRIS	1	52	1.90
OCEAN	5	141	3.50
PASSAIC	1	57	1.80
SALEM	1	21	4.80
SOMERSET	1	32	3.10
SUSSEX	9	125	7.20
UNION	0	2	0.00
WARREN	4	83	4.80

FIGURE 1—Percent Seropositive by County of Employment



COUNTY	BITES PER YEAR
ATLANTIC	6.7
BERGEN	3.5
BURLINGTON	6.8
CAMDEN	11.6
CAPE MAY	6.7
CUMBERLAND	7.8
ESSEX	12.0
GLOUCESTER	6.0
HUDSON	1.0
HUNTERDON	2.0
MERCER	2.4
MIDDLESEX	2.3
MONMOUTH	3.2
MORRIS	2.4
OCEAN	4.2
PASSAIC	2.1
SALEM	3.3
SOMERSET	2.6
SUSSEX	1.0
UNION	1.0
WARREN	1.3

FIGURE 2—Mean Number of Self-reported Tick Bites per Year by County of Employment

Preventive Measures: Self-reported behavioral practices are displayed in Table 1 on a statewide basis and by selected counties in Table 2. These behaviors were performed more frequently on the job than in non-occupational settings. Additionally, although workers frequently checked themselves for ticks, a minority of workers reported the other behaviors. In an effort to account for the unexpectedly high seroprevalence rates found in three northern counties (Sussex, Hudson, and Hunterdon) we chose to compare the preventive behavior performance frequencies in these counties with the frequencies found in three southern counties with relatively low seroprevalence rates. Table 2 reveals that these preventive behaviors were performed more frequently by employees in Monmouth County and three selected southern counties (Atlantic, Ocean, and Cape May) than they were in the three northern counties (Sussex, Hudson, and Hunterdon). The results for the other northern and southern counties were similar to those for the selected northern and southern counties reported.

TABLE 1—Percent of Workers Who Report Performance of Preventive Behaviors Statewide

Specific Behavior	Percent Who Perform Behavior While Outdoors		
	Frequently (Usually or Always)	Sometimes	Never
Wear long pants in the summer			
At work	85.7	10.7	4.3
Not at work	47.9	44.3	7.7
Wear long sleeves in the summer			
At work	13.7	37.8	48.5
Not at work	6.7	48.7	44.6
Tuck pants into socks or shoes			
At work	8.2	21.4	71.0
Not at work	3.6	17.3	79.2
Use insect repellent on skin			
At work	22.6	47.0	30.6
Not at work	17.7	50.4	31.8
Use insect repellent on clothes			
At work	19.3	43.2	37.5
Not at work	16.2	44.6	39.1
Check self for ticks			
At work	74.9	14.4	10.7
Not at work	65.9	22.8	11.3

Discussion

To our knowledge, the present study is the first to investigate the seroprevalence of antibody to *B. burgdorferi* on a statewide basis.

Several studies have investigated the seroprevalence or the seroconversion rate of populations residing or working in endemic areas. An early investigation in 1982 reported a one-year clinical Lyme disease cumulative incidence rate of

1.1 percent in 1,364 residents and employees living on or around the grounds of Naval Weapons Station Earle in Monmouth County, New Jersey.[10] A 1984 study of 176 residents of a Fire Island, New York summer community revealed that 9.7 percent had serological evidence of exposure to the Lyme spirochete in the initial cross-sectional portion of the study.[11] A 1986 study of long-term residents on Great Island, Massachusetts reported a seroprevalence of 8 percent among 121 asymptomatic residents.[12] More recently, an evaluation of 190 residents living within 5 kilometers of a nature preserve in Ipswich, Massachusetts from 1980–87 revealed a Lyme disease attack rate of 35 percent based on a clinical case definition.[13] Seventy-three percent of those with clinical disease and 23 percent of all residents tested were found to be seropositive for *Borrelia burgdorferi*-specific antibody. A 1982 comparison of outdoor versus indoor workers at Naval Weapons Station Earle revealed a five-fold higher cumulative incidence rate for Lyme disease among outdoor workers.[3] Finally, a recent study by Smith, *et al*, of 414 outdoor workers in southeastern New York State revealed a Lyme disease seroprevalence of 6.5 percent among these employees.[14] This was 5.9 times higher than the seroprevalence of a comparison group of anonymous blood donors from the same region.

It should be noted that participation in the study was voluntary and therefore unequal participation by county is a potential source of bias. Rates of participation on a county-by-county basis were not possible to determine. We did, however, find that the main determinant of participation was whether or not the employee's primary work location was one of the screening sites. These sites included an equal number of randomly distributed northern and southern locations. Job duties were similar in all the counties and the vast majority of all subjects statewide had not previously been tested. Therefore we feel it is unlikely that there was any specific county characteristic which resulted in a differential degree of study participation.

Our statewide seroprevalence of antibody to *B. burgdorferi* (5.7 percent) in a high-risk group of outdoor workers is comparable to the 6.5 percent seroprevalence found by Smith, *et al*, in New York State. When examined on a county-by-county basis, however, there were some unexpected findings. Earlier studies by Bowen and Schulze of Lyme disease distribution in New Jersey (1978–82) had reported a large number of case reports, as well as a high density of *Ixodes dammini*, in Monmouth County and the southern half of the state. Their results revealed a paucity of cases and ticks in the northern half of the state.[3–7] The present study revealed that one of five counties with seroprevalences greater than the statewide mean of 5.7 percent was Monmouth County and the others—Sussex, Hunterdon, Hudson, and Essex—were all counties in the northern half of the state (Figure 1). The Essex finding can be discounted because of a very small denominator (n = 5) but denominators for the other counties were much larger (n = 27 to 129). Similar (unpublished) analyses of distribution based on county of residence revealed seven counties with seroprevalence rates greater than the statewide mean. One was Monmouth, but again five of the remaining six (Sussex, Hudson, Middlesex, Somerset, and Mercer) were in the northern half of the state.

The preponderance of northern counties with elevated seroprevalence was not matched by an increased level of self-reported tick exposure in these counties. In fact, as displayed in Figure 2, the vast majority of counties with tick exposures greater than the statewide mean were southern counties; for "total tick bites in the past year," seven out of eight were southern counties. Similarly for "ticks removed from skin or clothing per week on primary job," eight out of 10 were southern counties.

In three southern counties, where seroprevalence was low but tick exposure was high, the employees performed the preventive measures on a "frequent" basis more often than their colleagues statewide (based on mean results from all 689 employees). In Monmouth County, the traditional hotbed of Lyme disease in New Jersey where only a moderately elevated seroprevalence rate was found, the employees also reported performance of preventive measures at a greater frequency than the statewide mean. Finally in three northern counties with relatively high seroprevalence rates and relatively low tick exposure, the employees were performing preventive measures less frequently than other employees statewide.

These findings suggest that the performance of preventive behaviors may reduce the risk of exposure to *B. burgdorferi*. These results do not establish a causal relationship but do merit further investigation. It may be postulated that there has been a relatively recent spread of infected ticks to the northern counties and, although their numbers are still not great, the northern ticks are feeding on a more susceptible human population.[13] The well-publicized history of Lyme disease in the southern counties may have led to a better educated public in these counties and therefore a greater awareness of ticks and appropriate risk reduction behaviors. It is possible that a certain number of individuals from southern counties where Lyme disease had been better publicized responded with expected behavior rather than

TABLE 2—Percent of Workers Reporting Frequent Preventive Behavior Performance for Selected New Jersey Counties

	Specific Behaviors					
County	Long Sleeves (13.7)*	Tuck Pants (8.2)*	Skin Repellent (22.3)*	Clothing Repellent (19.3)*	Tick Check (74.9)*	Number of Behaviors Greater than Statewide Mean
Atlantic	14.0	15.6	32.5	34.2	86.9	5/5
Ocean	16.8	12.4	29.2	27.7	89.0	5/5
Cape May	13.1	16.6	42.6	41.0	93.4	4/5
Monmouth	17.5	10.4	27.8	27.8	84.9	5/5
Sussex	13.4	4.2	10.9	9.2	53.8	0/5
Hudson	7.8	11.5	22.0	11.5	69.2	1/5
Hunterdon	10.7	2.7	10.7	8.0	78.6	1/5

*Statewide mean percent for frequent performance (usually or always)

actual behavior leading to a potential source of bias. This differential Lyme disease awareness may have also resulted in an overreporting of tick exposure in the southern counties and a relative underreporting in the northern counties. A comprehensive statewide study of tick concentrations would help to resolve this last possibility. However, some limited statewide data (Schulze TL: unpublished data) suggest that tick concentrations are much lower in the northern part of the state and that differential reporting of tick exposure is, in fact, not likely to be the explanation for the observed differences in seroprevalence.

In summary, the findings suggest that Lyme disease, as measured by seroprevalence of antibody to *B. burgdorferi*, appears to be spreading beyond Monmouth County and southern portions of the state where it had been previously identified. This spread into the northern counties may not necessarily be solely from southern New Jersey but could also reflect the spread of disease from bordering Orange and Rockland counties in New York. Unexpectedly high seroprevalences were found in several northern New Jersey counties (Sussex, Hudson, and Hunterdon). These high seroprevalence rates in regions of relatively low tick exposure, coupled with low rates (Atlantic, Ocean, and Cape May) and surprisingly moderate rates (Monmouth) in areas of higher tick exposure may be related to preventive behaviors. These results suggest that simple preventive behaviors may have a significant impact on minimizing the risk of Lyme disease.

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Evaluating the Impact of Municipal Water Fluoridation on the Aquatic Environment

JOHN W. OSTERMAN, MD, ScD

Abstract: Although highly beneficial for dental health, low concentrations of fluoride in environmental waters may be toxic to several organisms. In an era of heightened public awareness about the environment, this may lead city officials to withhold implementing water fluoridation for environmental reasons. This paper presents a mass balance approach to evaluate this perceived risk. Generally speaking, fluoridated water loss during use, dilution of sewage by rain and ground water infiltrate, fluoride removal during secondary sewage treatment, and diffusion dynamics at effluent outfall combine to eliminate fluoridation-related environmental effects. In Montreal, water fluoridation would raise average aquatic fluoride levels in the waste water plume immediately below effluent outfall by only 0.05–0.09 mg/l. Downstream, these changes would be only 0.02–0.05 mg/l at 1 km, and 0.01–0.03 mg/l at 2 km below outfall. Overall river fluoride concentrations theoretically would be raised by 0.001–0.002 mg/l, a value not measurable by current analytical techniques. All resulting concentrations would be well below those recommended for environmental safety and would not exceed natural levels found elsewhere in Quebec. A literature review did not reveal any examples of municipal water fluoridation causing recommended environmental concentrations to be exceeded, although excesses occurred in several cases of severe industrial water pollution. (*Am J Public Health* 1990; 80:1230–1235.)

Introduction

The beneficial effects of water fluoridation for human health are widely recognized.[1–3] Optimally fluoridated drinking water significantly reduces dental caries in both children and adults.[2–5] Fluoride may also reduce the incidence of osteoporosis and hip fractures, although the evidence is controversial.[6–8] No side effects occur.[1–3] Although the prevalence of mild dental fluorosis may increase, the esthetic effects of this mild tooth whitening are greatly outweighed by the improved esthetics of fewer decayed, missing, and filled teeth.[9] By far the most cost-effective intervention in dentistry,[10] optimally fluoridated water is provided in more than 10,000 North American communities and in countries around the globe.[11–13]

The toxic environmental effects of fluorides are also well documented, however. Airborne fluoride pollution from industrial sources may cause severe effects in plants by inhibiting photosynthesis, chlorophyll production and carbohydrate metabolism, with resulting defoliation or death.[14–16] Most plants accumulate fluoride which may lead to bony and dental fluorosis, lameness, arthritis, and other symptoms in wild and domestic herbivores foraging downwind from fluoride air pollution sources.[17]

As a water pollutant, elevated concentrations of fluoride may affect a number of organisms, including fish, amphibians, insects, snails, shellfish, protozoa, and some aquatic plants.[18] Fresh water fish, especially rainbow trout and other trout species, appear to be particularly sensitive and may show growth and behavioral changes, decreased survival, and prolonged hatching time when exposed to moderate levels.[19] Under negligible water hardness conditions, lethal effects for rainbow trout may occur at fluoride concentrations as low as 2.7–4.7 mg/l[20] and one undocumented observation reported delayed hatching of trout eggs at 1.5 mg/l.[21] Small amounts of calcium carbonate found in soft or moderately hard water, however, greatly reduce these effects,[22] as do water hardness generally, alkalinity, the presence of metallic ions (particularly calcium and magnesium) and chloride.[22–24] Toxicity increases with water temperature, by increasing fish metabolism.[25]

To protect aquatic organisms, several authorities recommend an upper limit between 1.0 mg/l and 1.8 mg/l of fluoride for fresh water systems.[18,26–28] Others feel that the environmental literature on fluoride toxicity is incomplete. They recommend that levels protecting drinking water (generally 1.2 to 2.0 mg/l) also be used to protect the environment, arguing that detectable effects occur in humans at lower concentrations than in other species.[29–32] Nevertheless, some states allow concentrations as high as 5–10 mg/l if the contaminated water is not subsequently used for human consumption.[33] No effluent standards exist.

Industrial waste and municipal sewage may add to the fluoride which is naturally present in all surface water.[33] In an era of heightened public awareness about environmental issues, concerns of public interest groups may lead city officials and politicians to withhold water fluoridation for environmental reasons, as occurred in Montreal in 1988.

To evaluate the environmental impact of water fluoridation, this paper uses a mass balance (material balance) approach to develop a series of mathematical equations which describe the fate of fluoride added to drinking water in a typical municipal water management system. In this approach, the ionic mass of fluoride entering the aquatic system from all sources is calculated, its distribution followed, and its fate examined; by balancing fluoride inputs and outputs, intervening concentrations and changes may be determined. This approach, widely used in environmental impact analyses,[34] is analagous to the study of physiologic or pharmacologic agents in living organisms.

The equations described below may be applied to readily available data from relevent water authorities to predict potential fluoridation-related changes in environmental waters receiving municipal effluent. These changes may be compared to natural variations in local ambient fluoride concentrations, the scientific literature concerning fluoride toxicity to aquatic organisms, and to recommendations from

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scientific and government agencies. The city of Montreal is presented as an example.

Methods

Fate of Fluorides in Municipal Water Systems

For most municipal water management systems, drinking water is drawn from a lake, river, or subterrain well and is pumped to a purification plant. There it may undergo aeration, flocculation, coagulation and sedimentation, filtration and be disinfected with ozone or chlorine dioxide. Usually a small amount of residual chlorine is maintained in the drinking water to avoid bacterial regrowth in the distribution system. Ambient fluoride concentrations are generally measured in the final stages of water purification. Fluoride is then added to obtain optimal levels, between 0.7 and 1.2 mg/l depending on climatic conditions. Although several fluoride-containing compounds may be used, all dissociate on admixing, thereby liberating free fluoride ion ($F-$).[35] Occasionally, when drinking water sources contain high concentrations of fluoride, the water may be "defluoridated" to optimal levels by one of several methods.[36]

Drinking water is distributed to a variety of residential, industrial, commercial, and municipal clients. Waste water from all these sources is collected by the city sewer system and is transported to the sewage treatment plant. Industrial and human wastes increase sewage fluoride levels. Some fluoride is lost, however, when drinking water is used to water gardens, wash cars, fill pools, extinguish fires, etc. Infiltrating ground water, underground streams, and rain may add to sewage flow and cause considerable dilution.

Primary sewage treatment removes suspended solids and phosphates by screening, filtration, flocculation, coagulation and sedimentation. Several common processes using iron salt coagulation, lime, or activated alumina may remove fluoride at high concentrations; however, these effects are minor at the low concentrations of fluoride found in municipal sewage.[36,37] After initial treatment, waste water undergoes secondary (biological) treatment in order to destroy unwanted organisms and reduce nitrogen levels and biochemical oxygen demand (BOD). The microorganisms present in most secondary treatment systems absorb large quantities of fluoride and may reduce fluoride concentrations in effluent by up to 50 percent.[38,39] Effluent may then undergo further disinfection with chlorine before discharge.

Effluent is generally discharged from an outfall pipe at a lake, river, or ocean bottom. The warmer effluent rises quickly to the surface creating physical turbulence and an immediate dilution which may be as high as 5–15 times. If natural dilution is low, a diffusion apparatus may be installed at effluent outfall which distributes effluent evenly over a wide area to ensure similar dilution levels.[40] Natural currents then carry the waste water downstream, creating a waste water "plume" and secondary dilution over several miles.

Mathematical Equations Evaluating Fluoridation Impact

From this overview of the fate of fluorides used in water fluoridation, mathematical equations may be derived to estimate the impact of fluoridation on the aquatic environment downstream of municipal sewage outfall. Terms used in these equations are defined in Table 1.

Global Impact

An estimate of global environmental impact may be obtained by comparing the fluoride needed to optimally fluoridate drinking water to the amount naturally present in the aquatic system receiving municipal effluent. Adjusted fluoride equals the difference between optimal and pre-existing drinking water fluoride concentrations (ΔF_M) multiplied by the rate per unit time of drinking water production (Q_M). Environmental fluoride equals the sum of fluoride naturally present in drinking water ($F_{M1}Q_M$) and fluoride naturally present in the waters receiving municipal effluent ($F_{E1}Q_E$). Thus,

TABLE 1—Definition of Symbols

*Fluoride Concentrations**		
F_M	=	fluoride concentration in municipal drinking water
F_R	=	fluoride concentration in raw sewage
F_T	=	fluoride concentration in treated sewage
F_E	=	fluoride concentration in environmental water receiving municipal effluent
F_X	=	fluoride concentration in waste water plume at point X, downstream from effluent outfall site
Flow Rates		
Q_M	=	flow rate (production) of municipal drinking water
Q_S	=	flow rate of sewage at treatment plant
Q_E	=	flow rate of environmental water receiving municipal effluent
Portions		
P_M	=	portion of municipal drinking water destined for sewers
P_D	=	portion of sewage originating as drinking water
PE_X	=	portion of environmental water affected by waste water plume at point X
Diffusion Factor		
DF_X	=	a unitless number describing cumulative dilution occurring in the waste water plume from effluent outfall to point X downstream

*Fluoride concentrations before and after fluoridation (see text) are respectively indicated by subscripts 1 and 2.

$$\frac{\text{Municipal F}}{\text{Environmental F}} = \frac{\Delta F_M Q_M}{F_{E1}Q_E + F_{M1}Q_M} \quad (1)$$

If Q_M is very small relative to Q_E, in the case of a small municipality discharging into a large river, then Equation 1 simplifies to:

$$\frac{\text{Municipal F}}{\text{Environmental F}} = \frac{\Delta F_M Q_M}{F_{E1}Q_E} \quad (2)$$

Multiple municipalities and river systems may be considered in an additive fashion using the general formula:

$$\frac{\text{Municipal F}}{\text{Environmental F}} = \frac{\Sigma_i \Delta F_{Mi} Q_{Mi}}{\Sigma_i F_{E1i} Q_{Ei} + \Sigma_i F_{M1i} Q_{Mi}} \quad (3)$$

The change in environmental fluoride concentrations due to fluoridation (ΔF_E) may be obtained by dividing the fluoride added to drinking water by the sum of municipal and receiving water flow rates.

$$\Delta F_E = \frac{\Delta F_M Q_M}{Q_E + Q_M} \quad (4)$$

In a multiple city or river situation, this relationship becomes:

$$\Delta F_E = \frac{\Sigma_i \Delta F_{Mi} Q_{Mi}}{\Sigma_i Q_{Ei} + \Sigma_i Q_{Mi}} \quad (5)$$

Obviously, the post-fluoridation concentration in water downstream from municipal sewage outfall equals the sum of the calculated change and the pre-existing concentration. The ratio of these two values ($\Delta F_E/F_{E1}$) is identical to Equation 1.

While conceptually simple, these models overestimate the real effects of fluoridation on the environment by assuming that all drinking water is discharged as waste, by excluding the effects of sewage treatment, and by discounting eventual fluoride deposition into sediments. Moreover, estimates from these models are valid only at some theoretical point downstream where municipal effluent and receiving waters have become thoroughly mixed.

The first of these objections may be overcome by including in the numerator of Equations 1–5 only the percentage of drinking water which is eventually discharged as effluent (P_M). Thus, for example, Equation 1 would be corrected to:

$$\frac{\text{Municipal F}}{\text{Environmental F}} = \frac{\Delta F_M Q_M P_M}{F_{E1}Q_E + F_{M1}Q_M} \quad (6)$$

Local Impact

To estimate fluoridation-related changes in fluoride concentrations in the waste water plume downstream from municipal effluent outfall, the effect of sewage treatment must first be evaluated by comparing fluoride concentrations in treated (F_{T1}) and raw (F_{R1}) sewage. The ratio of these values (F_{T1}/F_{R1}) shows the portion of fluoride remaining after sewage treatment and is not affected by fluoridation status.[38,39] The concentration of fluoride in post-fluoridation treated municipal effluent (F_{T2}) may then be estimated.

$$F_{T2} = F_{T1} + \frac{\Delta F_M P_D F_{T1}}{F_{R1}} \quad (7)$$

P_D is the portion of sewage originating as drinking water and equals $Q_M P_M/Q_S$. From the rate of diffusion of waste water in the effluent plume (DF_X), the concentration of fluoride in the plume (F_{X2}) may now be estimated at outfall and for points downstream.

$$F_{X2} = \frac{F_{T2} + F_{E1}(DF_X - 1)}{DF_X} \quad (8)$$

The change in fluoride concentration at any point downstream from outfall (ΔF_X) equals:

$$\Delta F_X = \frac{\Delta F_T}{DF_X} \quad (9)$$

Waste water is unequally distributed in the plume. Thus, diffusion, and DF_X, is greatest at the edge where effluent concentrations are least and, conversely, least at the center where concentrations are highest. Also, turbulence may create eddies of poorly diluted effluent at the outfall site, which disappear downstream. It should be noted that DF_X has a maximum value equal to flow in the receiving water (Q_E) divided by flow of municipal sewage (Q_S); generally this limit is important only when large volumes of effluent are discharged into small bodies of water.

The effluent plume affects only a portion of the receiving waters. This portion (P_X) at point "X" may be calculated as follows:

$$P_X = \frac{Q_S(DF_X)}{Q_E} \quad (10)$$

Results

An Example: The City of Montreal

Montreal's drinking water, serving 1,6 million people, is drawn from the St. Lawrence River, near the Lachine Rapids slightly upstream from the city, and is returned some 35 km downstream at Ile-aux-Vaches. From data provided by municipal, provincial, and federal agencies,[41–47] potential global changes in fluoride levels related to water fluoridation were estimated (Table 2). At the Lachine Rapids the daily water flow of the St. Lawrence averages 883 (range 659–1028) million cubic meters, and natural fluoride levels average 0.13 (range 0.11–0.17) mg/l. On average, therefore, 115 tonnes (tonne = 1000 kg = 2,204.6 pounds) of naturally present fluoride (theoretical range 73–175) flow past Montreal daily. Assuming all municipal drinking water eventually returns to the river, fluoridation would add an additional 1.93 tonnes to this water, or 1/60 (1.7 percent) of naturally occurring amounts. On average, however, only 57 percent of Montreal's drinking water returns to the river, making the additional fluoride load closer to 1.09 tonnes, or 1 percent of natural amounts. This additional charge is well within natural variations in fluoride concentration (±15 percent), river flow (± 16 percent), and fluoride ion mass (±37 percent). Overall, the average concentration of fluoride in the river downstream from effluent outfall would increase by 0.001–0.002 mg/l, a value which is not measurable by current analytical methods.

Excluding rain, Montreal produces an average of 2.4 million cubic meters of sewage per day, of which 42 percent originates as unfluoridated drinking water, 2 percent as fluoridated drinking water from neighboring suburbs, and 56 percent from infiltrating ground water. This latter value is very high, due to Montreal's island geography, and an old combined sewer system in one sector. Optimal fluoridation in Montreal would increase drinking water fluoride concentrations from 0.11–0.15 mg/l to 1.2 mg/l which would in turn increase the average concentration of fluoride in Montreal sewage by 0.45 ± 0.01 mg/l. Daily fluoride concentrations in

TABLE 2—Average Daily Water Flow and Fluoride Levels in the St. Lawrence River and Montreal Water Supplies, before and after Fluoridation (range in parentheses)

	Flow Rate ($m^3 \times 10^6$)	Fluoride Ion Concentration (mg/l)	Fluoride Ion Mass (metric tons)
St. Lawrence River	883 (659–1028)	0.13 (0.11–0.17)	115 (73–175)
Water supply before fluoridation	1.8 (1.5–2.2)	0.13 (0.11–0.15)	0.23 (0.16–0.33)
Water supply after fluoridation	1.8 (1.5–2.2)	1.2	2.16 (1.80–2.64)
Fluoride added to water supply	1.8 (1.5–2.2)	1.07 (1.05–1.09)	1.93 (1.64–2.31)
Fluoride added to waste water from water supply	1.02*	1.07 (1.05–1.09)	1.09 (1.02–1.11)

*Range not available

Montreal sewage average 0.34 ± 0.07 mg/l (mean ± SD) and range from 0.22 to 0.58 mg/l.[45] Thus, after fluoridation, concentrations in Montreal sewage would become 0.79 ± 0.08 mg/l (mean ± SD) and range from 0.66 to 1.04 mg/l. Because Montreal sewage does not undergo biological treatment, little or no fluoride removal occurs at the sewage treatment plant.

The diffusion of Montreal's waste water in the St. Lawrence river was measured in 1983 as part of a feasibility study for the new municipal sewage treatment plant.[46] At sewage outfall a minimum initial diffusion factor of 5 occurred at the center of the waste water plume, and initial diffusion averaged 15 over the plume's entire cross-sectional area. After adjusting for current modifications which will increase outfall capacity and thereby decrease diffusion slightly, an average increase of 0.05 mg/l in fluoride concentration would be predicted across the plume immediately below effluent outfall (Table 3) and an average increase of 0.09 mg/l at the plume's center (Table 4). The plume's average concentration immediately below outfall would be 0.20 mg/l and the average level in the plume center at outfall would be 0.26 mg/l. Given known variations of fluoride ion concentrations in Montreal's sewage, the greatest daily average concentration expected would 0.34 mg/l at the outfall center. These values would decrease rapidly downstream. At 1 km from outfall an increase of only 0.02–0.05 mg/l would be expected. The predicted change at 2 km would be only 0.01–0.03 mg/l and would affect only 3.5–6.3 percent of the river.

Discussion

Using a mass balance approach, mathematical equations may be derived to evaluate the environmental impact of fluoride added to optimally fluoridate municipal water supplies. Generally, it will be shown that fluoridation has little impact on the surrounding aquatic environment. Some fluoride is lost when drinking water is used to water lawns, fill pools, etc; this usually ends up in the soil which may already contain large amounts (100–300 ppm).[3,33] Although fluoride concentrations in the sewer system may increase with the addition of industrial and human wastes, they are diluted by rain and infiltrated ground water. Up to 50 percent of fluoride may be removed during secondary sewage treatment. Initial diffusion rapidly dilutes the remainder at the effluent outfall site. Secondary diffusion causes further reductions downstream. Eventually, the remaining fluoride is incorporated into river sediments, or carried to the ocean which already contain large amounts (1.2–1.4 mg/l).[33] Any toxicity from this residual fluoride would be severely inhibited by other substances present in surface and waste waters, notably water hardness, calcium ion, and chloride.[22–24]

TABLE 3—Estimated Average Daily Fluoride Concentrations (mg/l) across Effluent Plume before and after Municipal Water Fluoridation in Montreal (range in parentheses)

Distance from Outfall (km)	Diffusion Factor*	Concentration before Fluoridation	Concentration after Fluoridation	Change	Portion of River Affected %
0.3	10	0.15 (0.12–0.21)	0.20 (0.16–0.26)	0.05	2.0
1	19	0.14 (0.12–0.19)	0.16 (0.14–0.22)	0.02	3.9
2	31	0.14 (0.11–0.18)	0.15 (0.13–0.20)	0.01	6.3

*Adjusted for current modifications to increase effluent capacity.

TABLE 4—Estimated Average Daily Fluoride Concentrations (mg/l) at Effluent Plume Center before and after Municipal Water Fluoridation in Montreal (range in parentheses)

Distance from Outfall (km)	Diffusion Factor*	Concentration before Fluoridation	Concentration after Fluoridation	Change	Portion of River Affected %
0.3	5	0.17 (0.13–0.25)	0.26 (0.22–0.34)	0.09	1.0
1	10	0.15 (0.12–0.21)	0.20 (0.16–0.26)	0.05	2.0
2	17	0.14 (0.12–0.19)	0.17 (0.14–0.22)	0.03	3.5

*Adjusted for current modifications to increase effluent capacity.

Montreal is a typical example. Due to its island geography, infiltrating ground water in the sewer system is probably greater than elsewhere, but this effect is offset by the lack of a secondary sewage treatment plant. Water fluoridation in Montreal would result in a slight increase in the fluoride concentration of the waste water plume directly below the effluent outfall. These changes would return to pre-existing levels within two kilometers. Global changes would not be measurable. Rain water, not included in this analysis, would reduce sewage concentrations and these findings even further.

The maximum predicted daily concentration at the center of the outfall site (0.34 mg/l) would be well below recommended levels for environmental safety,[18,26–32] and would be similar to natural levels occurring elsewhere. A survey in Quebec showed that average daily fresh water fluoride levels across the province varied from 0.07 to 0.24 mg/l and maximum levels of 1.7 mg/l occurred.[47] The average concentration in US rivers measured at 343 stations of the National Stream Quality Accounting Network in 1975 was 0.33 mg/l[48]; six streams in the southwest US had concentrations of 1.4 to 1.8 mg/l. Other investigators report that fluoride concentrations in US rivers average 0.2 mg/l and range from 0.0 to 6.5 mg/l.[49] In a polluted section of the Illinois River around Peoria, fluoride concentrations averaged 1.08 mg/l and ranged from 0.17 to 2.06 mg/l.[50] The source of the pollution was unspecified. Ocean concentrations range from 1.2 to 1.4 mg/l.[33] One result is that marine and estuarine organisms are relatively immune to the toxic effect of fluoride.[33,51]

The predicted increase of 0.45 mg/l in the fluoride concentration of Montreal sewage due to water fluoridation is similar to findings elsewhere. Fluoride levels in raw sewage from seven fluoridated Ontario communities contained 0.47 mg/l more fluoride than raw sewage from 11 non-fluoridated communities (0.96 vs 0.49 mg/l), and treated sewage differed by 0.33 mg/l (0.62 vs 0.29 mg/l).[38] For 56 California cities, raw and primary treated sewage from fluoridated communities contained an average of 1.80 mg/l (range 1.4–3.2) or 0.37 mg/l more fluoride than non-fluoridated communities (average 1.43; range 0.6–3.6 mg/l).[39] Both studies showed the important effects of secondary sewage treatment which reduced effluent fluoride concentrations by 35 percent in Ontario and by 56 percent in California in both fluoridated and non-fluoridated communities. In California, domestic waste accounted for far more sewage fluoride than water fluoridation.

The fate of fluoride in waste water from municipal and industrial sources has been evaluated by a number of researchers. In 1973, fluoridated Bozeman, Montana (population 20,000) discharged an average of 8.5 m^3/min of primary treated municipal effluent into the East Gallatin River (average flow: 146.7m^3/min). This raised average fluoride levels from 0.33 mg/l, nine meters above outfall, to 0.62 mg/l at 0.3 km downstream.[52] A maximum level of 2.0 mg/l was recorded. Natural fluoride levels were reestablished 5.3 km downstream. Another study at the same site found that fluoride concentrations quadrupled at effluent outfall but returned to natural levels 4 km downstream.[53] These changes were not considered to have any effect on the environment.[29]

In 1977, fluoridated Minneapolis-St. Paul produced effluent at the rate of 9.6 m^3/sec with a fluoride concentration of 1.21 mg/l, which was added to the Mississippi River (flow rate 279 m^3/sec). Water collected 10 air miles below outfall contained the same fluoride concentration as that collected upstream from the sewage disposal plant.[54]

In 1986, Kudo, *et al*,[55] evaluated fluoride concentrations along four small rivers in an industrialized area of the French Alps near unfluoridated Grenoble. This city (population 350,000) added about 1.8 tonnes of fluoride daily in untreated sewage to the Isere River (flow rate 300 m^3/sec) which increased fluoride concentrations from 0.19 mg/l above the city to 0.26 mg/l some 70 km downstream. This study underlined the importance of industrial water pollution sources. On the small Arc River (flow rate 50 m^3/sec) fluoride concentrations increased from 0.13 mg/l to 0.62 mg/l over a 50 km portion during which it received industrial effluents from three aluminum factories, a factory producing silica, and a factory producing phosphates. Severe fluoride-containing air pollution from these companies had destroyed much of the area's vegetation.

In another study underlining the importance of industrial water pollution, Martin and Salvadori[56] found that industrial effluents from a fluorophosphate fertilizer plant in Rouen, France increased fluoride concentrations in the Seine River from 0.25 to 2.0 mg/l which eventually fell to 0.47 mg/l many miles downstream. Fluoride discharge was estimated to be 37 tonnes per day, a quantity which would fluoridate a city 20 times the size of Montreal!

In conclusion, by using a mass balance approach, fluoridation-related changes in environmental concentrations of fluoride may be estimated from knowledge of municipal water management systems and data which are usually readily available from appropriate water authorities. Generally speaking, these changes will be minimal and, except when accompanied by serious industrial pollution, will remain below toxic levels recorded in the literature and recommendations by scientific authorities for the protection of the environment and human health.

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Crop Duster Aviation Mechanics: High Risk for Pesticide Poisoning

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Abstract: A cross-sectional medical survey was conducted among 63 Nicaraguan aviation mechanics exposed to organophosphate and other toxic pesticides. Thirty-one (49 percent) reported having been acutely poisoned on the job. Also, seven of 14 novice mechanics, with less than one year on the job, reported that they had been poisoned. Thirty-eight (61 percent) had cholinesterase levels below the lower limit of normal, including three workers with levels less than 20 percent of the lower limit of normal. Risk factors for low cholinesterase included recent hire and recent poisoning. Workers did not use protective equipment, nor were there facilities for bathing on site. As a result of this survey, the government has prohibited the mixing and loading of pesticides at this airport and requires the washing of planes prior to maintenance work; coveralls and thin, pesticide impermeable gloves are to be issued to mechanics handling pesticide-contaminated parts. Closed system mixing and loading systems have been installed at satellite airstrips. (*Am J Public Health* 1990; 80:1236–1239.)

Introduction

Pesticide poisoning has been a major public health problem in Nicaragua for the last 30 years.[1] The country's principal traditional export is cotton, a crop which is sprayed up to 35 times a season with organophosphate insecticides for control of the boll weevil. Most of this spraying is done from crop dusting airplanes, and poisoning of highly exposed mixers and loaders at the airports and landing strips which service these airplanes has been recognized as a problem, both in Nicaragua and in the developed countries.[2]

The airplane mechanics who service these airplanes are a little studied population who have high exposure to pesticide-contaminated parts. To characterize exposure of these workers to pesticides and to assess any acute health effects, we evaluated mechanics at Nicaragua's largest crop dusting airport in late December 1987.

Methods

The 28 crop dusters owned by the six companies operating out of the airport made approximately 10 flights daily during the peak cotton spraying season (October 15 through early January). It was estimated that as much as 130,000 gallons were sprayed daily on flights originating from this airport, accounting for almost 30 percent of all pesticides sprayed in the department of Chinandega, the principal cotton growing region (Mario Pais, Ministry of Industry, personal communication). The mechanics were under pressure to do minor service and adjustment as quickly as possible, because the companies and pilots were reimbursed for each flight. There was little time to wash planes before minor maintenance. Most exposure was to the hands, since the manipulation of small mechanical parts was necessary for the routine work of changing pesticide filters and adjusting spray nozzles, and difficult to perform while wearing clumsy protective gloves.

The mechanics often did not know what pesticide was loaded in the plane they were servicing. One employee from each company, with responsibility for recording the pesticide used in each flight, characterized frequency of spraying as daily or as less frequently for the various pesticides used at the airport during the month prior to the study.

The 71 mechanics (all men) employed at the airport were invited for a questionnaire interview which asked about history of use of personal protective equipment, history of training regarding poisoning prevention, about previous poisoning, and about symptoms compatible with cholinesterase inhibitors (organophosphate and carbamate insecticides). The questionnaire asked about specific symptoms, such as headache, lightheadedness, blurred vision, lack of appetite, vomiting, stomach ache, and frequent spitting (sialorrhea). Erythrocyte cholinesterase is inhibited by organophosphate insecticides and is an early indicator of overexposure. It was measured using the Ellman assay (adapted for use with a new battery operated field kit), as previously described[3]:

> Briefly, 5 µl of blood was pipetted into a cuvette containing 200 µl of sample buffer containing Tris-HCl, Triton X-100 surfactant, and trace azide. Two ml of reagent solution containing acetylthiocholine iodide substrate, DTNB [5,5'-dithiobis(2-nitrobenzoic acid)] indicator, quinidine sulfate (to inhibit plasma cholinesterase), and phosphate buffer were added to the cuvette. The solution was incubated for three minutes and the change in absorbance noted between one minute and three minutes. The activity was adjusted for temperature and converted to international units. The results were not adjusted for hemoglobin. Forty-six healthy, unexposed male Nicaraguan health workers were sampled in order to establish a lower normal limit of 3.7 international units/minute/ml of blood. (This is the mean minus 1.65 standard deviations).
>
> Each worker was given the result of his cholinesterase assay at the time of the test.
>
> Confidence intervals for relative risk of low cholinesterase for various risk factors were calculated using the test-based method of Miettinen.[4] Multivariate analysis of mean cholinesterase was done using SPSS-PC.[5]

Results

All pesticides sprayed at the airport during the month prior to the survey are listed in the Appendix. Methamidophos, mephosfolan, methyl and diethyl parathion are all US Environmental Protection Agency (EPA) category I (acutely toxic) organophosphate insecticides. Chlorpyrifos (category II) and acephate (category III) are less toxic organophosphates. Cholinesterase depression reflects overexposure to these insecticides. Unfortunately, there are no good field methods for evaluating exposure to the non-

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organophosphate pesticides, although there are several pesticides with chronic effects. A metabolite of chlordimeform was recently reported to be associated with a 73-fold excess risk of bladder cancer in exposed workers.[6]

Questionnaire

Sixty-three mechanics (88 percent) of the 71 who worked at the airport came to the health screening. Those who did not participate had been assigned temporarily to smaller satellite airports, were sick, or refused to participate. This is a relatively mature work force with an average of almost 10 years in the current job (Table 1). Nevertheless, there was a sizable proportion of novices (22 percent) with less than one year of experience, including one 11-year-old and one other worker under age 16, the minimum legal age for work with pesticides.[7] Almost half the workers reported having required medical attention for acute symptoms resulting from pesticide exposure at some time during work at the airport. Twelve (19 percent) of 63 reported that they were poisoned in the previous 12 months, and half of the 14 workers with less than one year of experience had been poisoned. Some older workers reported having been poisoned as many as seven times at the airport. The most common protective equipment used were boots (used by 14 percent), followed by coveralls (11 percent). No worker used protective gloves, a respirator, or protective glasses.

Cholinesterase Measurements

Thirty-eight (61 percent) of the 62 workers sampled had low erythrocyte cholinesterase levels (Table 1). The mean cholinesterase (3.1 international units [IU]/minute/ml blood) for all mechanics was less than the lower limit of normal of 3.7 IU. The lowest level recorded was 0.61 IU, one of three samples less than 20 percent of the lower limit of normal. That worker complained of headache and nausea, had pinpoint pupils and sialorrhea, and was diaphoretic, all of which responded to treatment with atropine sulfate.

We evaluated the following risk factors for depressed cholinesterase levels among these workers: recent poisoning, recent hire, no training about safe use of pesticides, less than daily change of clean work clothes, and symptoms compatible with organophosphate insecticide poisoning. In addition, the mean cholinesterase for workers with each risk factor was compared with the mean for all workers without the risk. Almost all workers poisoned in the previous year and almost all mechanics hired within the previous year had low cholinesterase values at the time of the screening (see Table 2). The presence or absence of more than two symptoms compatible with organophosphate insecticide poisoning was not reflected either in higher prevalence of low cholinesterase values (52 percent compared with 67 percent among those with two symptoms or less) or in lower mean cholinesterase levels.

Paradoxically, there was a higher prevalence of low cholinesterase among mechanics who reported a daily change of clean work clothes (87 percent) compared with mechanics who changed work clothes less often (53 percent).

There was a slightly lower prevalence of low cholinesterase levels among workers who had received some previous training about the safe handling of pesticides, and the mean cholinesterase level among workers who had received training (3.6 IU) was higher than among workers without any training (2.9 IU). However, this beneficial effect of training was confounded by the high exposure tasks of recently hired workers, who were also less likely to have received training. After controlling for a dummy variable representing recent hire (within the last year) in a multiple regression procedure, there was still a slight beneficial average effect on cholinesterase of having received training, but the effect was reduced (regression coefficient = 0.41 I.U.; 95% CI = −0.28, 1.13).

TABLE 1—Demographic Characteristics, History of Poisoning, and Cholinesterase Levels among Aviation Mechanics, Chinandega, Nicaragua, December 1987 (N = 63*)

Mean age (SD)	29.1 (10.1)
Mean duration (years) of employment (SD)	9.6 (8.2)
Number employed less than 1 year (%)	14 (22)
Number poisoned during employment at airport (%)	31 (49)
Number poisoned in previous 12 months (%)	12 (19)
Number poisoned in previous 12 months among mechanics with less than 1 year employment (%)	7/14 (50)
Number who have received pesticide safety training on current job (%)	16 (26)
Number with low erythrocyte cholinesterase (%)	38 (61)
Mean cholinesterase in international units/minute/ml blood (SD)	3.1 (1.3)

*Because of missing data the denominator varies slightly within this and subsequent tables.

Discussion

To our knowledge mechanics engaged in routine maintenance of crop dusters have not been studied previously. At this airport, they were at extraordinarily high risk for pesticide overexposure and for poisoning. Many of these workers reported being hospitalized for treatment, almost exclusively with signs and symptoms compatible with the cholinesterase inhibitors used at the airport. The high prevalence of cholinesterase depression is probably underestimated, because pre-exposure baseline levels of cholinesterase were not available. We therefore used a normal range based on inter-individual variability in cholinesterase. A more appropriate criterion for a low cholinesterase level is a drop of 30 percent from baseline, as recommended by the World Health Organization[8] and the US National Institute for Occupational Safety and Health.[9] Because the inter-individual normal range is almost a two-fold increase over the lower limit of normal, a decrease of well over 30 percent for someone with a high baseline cholinesterase level would still leave that person in the normal range in this study.

In workers with daily exposure to organophosphate pesticides, cholinesterase may be depressed without clinical signs and symptoms of poisoning.[10] The absence of association between symptoms and cholinesterase depression probably reflects the importance of the rate of decline in cholinesterase in producing symptoms.[2] A gradual inhibition of cholinesterase from continued low level exposure to organophosphates is less likely to produce symptoms than the same inhibition from a single large exposure. Our experience is not dissimilar to that reported in other studies, where symptoms sometimes have been found to be associated with chronic depression of cholinesterase and sometimes have not.[2,11–13]

It is likely that experienced workers tolerate the hazards of this job, because it is highly skilled and it pays well. In addition, the most hazardous work is done by the newly hired apprentice mechanics, many of whom do not return the following season. The anomalous increase in proportion of workers with low cholinesterase levels among those who changed to clean work clothes daily probably occurred because these young workers are in the dirtiest, most highly

TABLE 2—Mean Cholinesterase and Frequency of Low Cholinesterase among Aviation Mechanics by Selected Risk Factors, Chinandega, Nicaragua, December 1987

Risk Factor	Number with Low Cholinesterase (%)	Relative Risk (95% CI)	Mean Cholinesterase	Difference of Means (95% CI)
Poisoned in the previous year (N = 12)	11 (92)	1.7 (1.1, 2.6)	1.5 IU	−2.0 (−1.3, −2.7)
Not poisoned in the previous year (N = 50)	27 (54)		3.5 IU	
Less than 1 year in job (N = 14)	13 (93)	1.8 (1.2, 2.7)	2.0 IU	−1.4 (−0.7, −2.1)
One or more year in job (N = 48)	25 (52)		3.4 IU	
Has not received pesticide safety training at airport (N = 45)	30 (67)	1.5 (0.91, 2.5)	2.9 IU	−0.7 (−0.07, −1.3)
Has received training (N = 16)	7 (44)		3.6 IU	
Changes work clothes less often than daily (N = 47)	25 (53)	0.61 (0.40, 0.93)	3.2 IU	0.6 (1.3, −0.1)
Changes work clothes daily (N = 15)	13 (87)		2.6 IU	
More than 2 symptoms compatible with cholinesterase inhibitors (N = 23)	12 (52)	0.78 (0.50, 1.2)	3.0 IU	−0.1 (0.6, −0.8)
Two or fewer symptoms compatible with cholinesterase inhibitors (N = 38)	26 (67)		3.1 IU	

exposed jobs and are also more likely to need to change work clothes daily.

Pesticide hazards at this airport are characteristic of Central American and other developing countries where regulations or their enforcement are not adequately developed to protect workers from poisoning. Even for these highly trained workers at a work place of strategic importance for one of the country's principal sources of foreign exchange (cotton), there were no bathing facilities and the companies did not provide protective equipment. To have removed all mechanics with low cholinesterase levels from exposure would have severely compromised production at a time when it was critical to spray to control the boll weevil. Only workers with the lowest cholinesterase levels were automatically removed from production. Where possible, workers with less depressed levels were assigned to less exposed tasks, hardly an ideal solution, as all mechanic jobs involve potential pesticide exposure.

In addition, the pesticide multinationals have introduced products which are prohibited or severely restricted in the developed world, or which are not registered in developed countries and for which there is little health information available to national regulatory authorities. Although lowered cholinesterase levels reflect overexposure to, and poisonings result from, cholinesterase inhibitors, overexposure to cholinesterase inhibitors can be considered a marker for overexposure to other pesticides with chronic toxicity. Ethyl and methyl parathion are almost always mixed with the animal carcinogen toxaphene[15] in Nicaragua and were often applied with chlordimeform. Until recently, methyl parathion was involved in almost half of all poisonings reported in certain regions of the country.[11] The common practice of promoting the use of personal protective equipment (gloves and respirators) in a country where there is little personal protective equipment available, and where temperatures commonly reach 38 degrees centrigrade, is an inadequate solution to the problem of pesticide overexposure.

Nevertheless, Nicaragua appears to be taking the problem of pesticide health hazards seriously. In the past five years the government, with support from various nongovernmental organizations, has initiated programs to install closed systems (which mix and load the crop dusters without the workers having to directly handle the pesticide), and has initiated training programs to protect these workers. The Ministry of Health has prohibited the mixing and loading of pesticides at this airport; the airport is to serve as a base for storage, maintenance, and repair of previously washed planes. All mixing and loading is to be done at outlying satellite airstrips, where the closed mixing and loading systems have been installed. Although in practice this may be difficult to enforce, the Ministry's order (based on concern about contamination of ground water as well as worker health) is proving to be a useful tactic in forcing the companies to cooperate with remedial action. Coveralls have been distributed to all mechanics, and free laundry service is available to most of the mechanics working at the airport. Prior to the spray season in 1988, all mechanics were released from work for a two-day training program on safe handling of pesticides. A worker health and safety committee has been established and is being trained further in basic preventive measures and in emergency treatment of poisoning from cholinesterase inhibitors. Children under 16 years old are no longer employed at the airport, and funding has been obtained for a pilot program to evaluate a new, thin (but pesticide impermeable) glove to see if it can be used for the fine manipulation of parts by the mechanics. Perhaps the most important change which may result in decreased pesticide poisoning by these workers is the decreasing amount of land planted in cotton in Nicaragua. The result may be less demand for aerial spraying and less pressure to get the maximum production from each crop duster, making it possible to enforce the washing of each plane before any maintenance is done. Finally, despite strong objections by the cotton producing industry, the importation of chlordimeform was recently halted, and a new regulation gives a commission headed by the Ministry of Health the right to veto the importation of pesticides deemed to be dangerous to public health.

APPENDIX

Pesticides Used at Crop Dusting Airport, Chinandega, Nicaragua, December 1987

Pesticides with Long-term Health Effects

Chlordimeform** (metabolite is human bladder carcinogen)[6]
Methamidophos** (human delayed peripheral neurotoxin[14])
Toxaphene** (animal carcinogen[15])
Maneb/mancozeb** (animal carcinogen[9])

Other Pesticides Restricted or Prohibited in Other Countries[16]

Methomyl** Mephosfolan** Methyl** and diethyl* parathion

Other Pesticides not Registered in the US

Dominex** Bendocet** Jupiter**

Other Pesticides of Lesser Toxicity

Acephate**	Chlorpyrifos**	Deltamethrin**	Bacillus Thuringiensis**
Propargite**	Talstar*	Propiconazole*	Lambda Cyhalothrin**

*Sprayed at one or more companies during the 30 days prior to the study but not sprayed daily by any company.
**Sprayed daily by one or more companies during the 30 days prior to the study.

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Health and Environmental Outcomes of Traditional and Modified Practices for Abatement of Residential Lead-Based Paint

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Abstract: We evaluated traditional and modified practices for abating lead-based paint in homes of children with blood-lead concentrations (PbB) > 1.4 μmol/L (> 29 μg/dl). Traditional abatement resulted in acute increases in: 1) lead contaminated house dust (generally 3 to 6-fold over pre-abatement levels, but at abated sites typically 10 to 100-fold); and 2) the PbBs of nearly half of the occupant children. Modified practices represented modest short-term improvement compared to traditional practices but were also inadequate. By six months, it was clear that neither form of abatement resulted in long-term reductions of PbB or house dust lead levels, leaving children at continued risk of excessive exposure to lead and permanent adverse neurobehavioral effects. Windows were found to be high sources of lead contaminated house dust. Recommendations are made for improved abatement practices including more complete abatement of window units and more effective clean-up to remove lead-bearing dust. Thirteen million US children live in lead-painted dwellings. Research is needed to identify abatement strategies that will be practical and well suited to the current understanding of low-level lead toxicity. (*Am J Public Health* 1990; 80:1240–1245.)

Introduction

Abatement of residential lead-based paint hazards was required more than 40 years ago in response to cases of symptomatic lead poisoning in children (generally, blood lead concentrations (PbB) > 3.86 μmol/L).* These cases were often associated with the ingestion of chips of lead-containing paint in dilapidated dwellings.[1] Together with chelation therapy, abatement of lead-based paint hazards by traditional practices in homes of children with symptomatic lead poisoning contributed to the drastic reduction in the numbers of reported lead-related deaths and recurrences of acute encephalopathy in Baltimore by the late 1960s.[2,3]

More recently, clinical and epidemiological studies have revealed that body burdens of lead previously considered safe are associated with significant long-term adverse health effects which can occur even in the absence of clinical symptoms.[4–7] Since the 1960s, the PbB considered unacceptable has been progressively lowered from 2.90 μmol/L to the current level of 1.21 μmol/L (25 μg/dl).[8–11] New concerns are being raised about the risk of adverse neurodevelopmental effects as PbBs increase above the 0.48–0.72 μmol/L range.[12–15] Lead-contaminated house dust (PbD) ingested via repetitive hand-to-mouth activity is now recognized as a major contributor to the total body lead burden of children.[16–18]

Studies in the 1980s suggest that traditional abatement practices are inadequate for reducing exposure so that children's PbBs can be maintained within acceptable levels. Chisolm, *et al*,[19] found a recurrence rate of 47 percent of PbBs greater than 2.36 μmol/L in children following chelation therapy and reoccupancy of older dwellings abated by traditional methods. Charney, *et al*,[20] showed that repeated dust removal following lead-paint abatement was associated with a modest reduction in children's PbB whereas lead-paint abatement alone was not. Various case reports indicate that abatement and refinishing activity in homes with lead-based paint can result in increased PbBs in workers and children.[21–25]

Despite changes in our understanding of the epidemiology of childhood lead toxicity and recognition of low-level adverse health effects, practices for abating residential lead-based paint have remained essentially unchanged for the last 40 years. Only recently have a small number of states established upgraded abatement requirements (Maryland, Massachusetts, Minnesota).[26–28] This paper reports on an observational prospective study in which traditional abatement practices were evaluated and compared with modified practices intended to address some of the deficiencies of traditional practices. The study was a natural experiment in which the investigators had no control over the implementation of either form of abatement.

Methods

Study Design

Serial measurements of lead in interior surface dust (PbD, mg/m^2) and children's blood were made pre-abatement, post-abatement, and six months post-abatement. A subset of dwellings abated by traditional methods had additional PbD measurements at one and three months post-abatement. Households were identified through the Baltimore City Health Department, which inspected the dwellings of children identified with lead toxicity and enforced abatement requirements. During the enrollment period (March 1984 to January 1985), property owners either performed required lead-paint abatement themselves using traditional practices or elected to have city crews perform the abatement using modified traditional practices. City abatements were being offered for the first time and were done according to principles similar to those subsequently outlined by the Centers for Disease Control (CDC).[11]

Appendix A describes and compares the two abatement practices. Unlike traditional practices, modified practices included more than minimal efforts to clean the dwelling following abatement, dispose of debris off-site, and to protect workers, occupants and their belongings. Unlike traditional practices, modified practices excluded the use of open-flame burning and sanding techniques and included the repainting of abated surfaces.

Local abatement requirements consisted of addressing deteriorated paint on surfaces up to four feet from the floor

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*To convert to μg/dl multiply by 20.72

and all paint on easily accessible "biting" surfaces where the lead content of the paint was > 0.7 mg/cm^2 by x-ray fluorescence or 0.5 percent by weight by wet chemical analysis. At the time of enrollment, toxicity was defined by the CDC as a.PbB > 1.40 μmol/L (> 29 μg/dl) of whole blood together with a free erythrocyte protoporphyrin > 49 μg/dl of whole blood.[10] This study was approved by the Committee on Human Volunteers at the Johns Hopkins School of Hygiene and Public Health.

Study Sample

Seventy-one (71) study dwellings (53 traditional abatement, 18 modified abatement) had PbD measurements from pre-abatement and at least one point in time post-abatement. Attrition during the six months post-abatement was the same in both groups (17 percent).

Most study dwellings were pre-1940 row homes occupied by low-income tenants. Prior to abatement, all dwellings had multiple interior surfaces coated with lead-based paint and at least one child with PbB > 1.40 μmol/L. Based on walk-through inspections of 66 of the 71 study dwellings at the end of the study, dwellings were rated as follows using a classification system designed by housing professionals for use in this study: fairly well maintained (traditional = 8, modified = 6); substandard (poorly maintained yet maintainable: traditional = 30, modified = 10); and grossly substandard (not maintained and in need of renovation: traditional = 12 modified = 0).

Children in the 71 study dwellings abated by traditional or modified practices from which dust data were obtained, and children in an additional 25 dwellings abated by modified methods for which there were no dust-lead measurements, were included in the analysis of PbB change if they met the following criteria: age: 9 to 72 months; not known to be mentally retarded; spent 75 percent of his/her time living in the abated dwelling; resided in the abated dwelling pre- and post-abatement and had PbB determinations on venous blood at pre-abatement and at least one time post-abatement. Due to the confounding influence of chelation therapy on PbB and body burden of lead, children with histories of therapy at the time of enrollment were excluded from PbB analyses (except for the analysis of the association between PbB and PbD). PbB data were obtained by medical record review of children in the 25 dwellings abated by modified practices for which there were no dust-lead measurements.

Data Collection and Laboratory Methods

Over 3,100 dust samples were collected for lead analysis from nearly 900 sites in 71 study dwellings. From 12 to 24 dust samples were collected from the same floor, window sill, and window well (portion of window that receives the sash when window is closed) surfaces at each home visit. Samples were collected from most rooms, with particular attention to areas where children spent most of their time as reported by parents. As in previous studies[20,29,30] sample collection and preparation was done by the method of Vostal, *et al*,[31] except that no attempt was made to avoid collecting loose chips of paint and samples were analyzed using flame atomic absorption spectrophotometry at the Kennedy Institute's Trace Metals Laboratory. PbD results were expressed as mg Pb/m^2. The methods of this study underestimate the total amount of lead per surface sampled, particularly on window wells where the largest quantities of visible particulate matter and loose chips of paint were collected and the highest PbD levels were measured (see Appendix B).

Venous blood specimens were collected in low-lead vacuum tubes by the subjects' usual health care providers. Specimens were analyzed by atomic absorption spectrophotometry[32] by the Maryland Department of Health and Mental Hygiene or by anodic stripping voltammetry[33] at the Kennedy Institute's Trace Metals Laboratory using the rapid blood lead analyzer (Environmental Sciences Associates, Inc. Model 3010A, Bedford, MA). Inter-laboratory variation was minimal (data available from authors). Both laboratories served as reference laboratories for the CDC's blood lead proficiency testing program. After considering the precision of the analytical method using anodic stripping voltammetry (1 SD = 0.08 μmol/L, and modified Delves cup (1 SD = 0.16 μmol/L)[34], a change in PbB was defined as an increase or decrease of 0.24 μmol/L (5 μg/dl) or more (i.e. a change of which exceeds 1.5 to 3 standard deviations).

Data Analysis

In the absence of a federal standard for lead in interior surface dust, a target PbD of < 1.5 mg/m^2 was set for evaluation purposes. This target was based on an earlier study which reported all but one PbD level in modern suburban homes under 1.18 mg/m^2 (mean 0.29 mg/m^2).[30] Using a different method of sample collection and analysis, similar levels were reported (0.04 to 1.23 mg/m^2, GM = .20 mg/m^2) in public housing units where children were found to have acceptable PbBs (mean = 0.48 μmol/L).[35]

PbD values were found to be log-normally distributed. Units of analysis were individual PbB values and PbD values transformed using natural logarithms. PbB distributions were not log-normalized due to the restricted range of values. Multiple regression models based on maximum likelihood estimates were fit to PbB and to PbD at various points in time. Important covariates were included in the models. Since PbD values within dwellings were correlated (first order correlation coefficient for pre-PbD data ordered by household and surface type was 0.39), a correction for correlated residuals was made by adjusting the standard errors of the beta coefficients using an in-house program and retesting the significance of the variables.

Results

Dust-Lead Findings

Pre-Abatement Dust-Lead: Pre-abatement PbD levels ranged from less than 0.27 mg/m^2 to: 272 mg/m^2 on floors, 1076 mg/m^2 on window sills, and 4059 mg/m^2 on window wells. Dwellings abated by traditional and modified practices had comparable PbD before abatement (Table 1) despite differences in housing condition. Surface type and season were significant predictors of pre-PbD levels. Knowledge of which surfaces were identified for abatement was not a predictor of pre-PbD (see Table 2).

Pre-Post Change in Dust-Lead: Increased PbD levels were measured immediately following traditional abatement (usually within 48 hours) on abated window sills and on floors in close proximity to abated surfaces (Table 1). Increases at individual sites were typically 10 to 100-fold over pre-abatement levels. Most window sills (31/39) abated by the modified practices had lower PbD levels compared to pre-PbD levels. However, most window sill and floor surfaces remained outside the target range (<1.5 mg/m^2) even after final clean-up. Window wells, which were not treated during abatement by either group, had post-PbD levels which remained high in both groups (Table 1). Immediate post-abatement PbD levels in dwellings abated by modified prac-

TABLE 1—Geometric Mean (GM) Dust Lead Levels (mg/m²) Over Time by Type of Surface and by Abatement Group

Surface Type	Abatement Group	Pre-Abatement GM	Pre-Abatement 95% CI	Pre-Abatement N	Post-Abatement[a] GM	Post-Abatement[a] 95% CI	Post-Abatement[a] N	6 Mos Post-Abatement GM	6 Mos Post-Abatement 95% CI	6 Mos Post-Abatement N
Floors	Traditional	2.7	(2.4,3.1)	280	15.5	(12.9,18.5)	271	3.4	(2.9,3.9)	234
	Modified	3.1	(2.2,4.2)	82	7.0	(4.9,9.9)	50	3.4	(2.6,4.5)	57
Window Sills	Traditional	14.4	(11.8,17.4)	249	38.7	(31.1,48.0)	246	16.6	(13.2,20.9)	199
	Modified	19.4	(14.6,25.9)	95	6.5	(4.8,8.8)	64	17.6	(12.4,25.0)	66
Window Wells[b]	Traditional	166.8	(124.7,223.3)	150	154.5	(120.8,197.5)	139	134.2	(97.0,185.6)	100
	Modified	196.7	(121.8,317.7)	37	87.0	(52.6,143.9)	24	267.8	(164.7,435.4)	32

[a]Post-abatement levels include values from abated and non-abated surfaces.
[b]Window wells were not abated by either group.

TABLE 2—Multiple Regression Analyses: Pre- and Post-Abatement Interior Dust Lead (mg Pb/m²)

Independent Variables	Pre-Abatement Beta	Pre-Abatement (se)[b]	Post-Abatement[a] Beta	Post-Abatement[a] (se)[b]
Intercept	0.37	(0.57)	5.59	(0.56)
ln (Pre-PbD)[c]	-		0.44	(0.05)
Abatement Group				
Traditional	ref		ref	
Modified	0.31	(0.32)	−2.09	(0.36)
Surface Type				
Floor	ref		ref	
Window sill	1.54	(0.18)	−0.17	(0.17)
Window well	4.00	(0.18)	0.64	(0.21)
Knowledge of Abatement Status				
Abated surface	ref		ref	
Proximate to abated	−0.23	(0.18)	−0.55	(0.21)
Neither of the above	−0.52	(0.25)	−1.55	(0.24)
Housing Condition				
Maintained	ref		ref	
Substandard	0.25	(0.34)	0.59	(0.28)
Grossly substandard	0.52	(0.40)	0.90	(0.34)
Don't know	−0.20	(0.44)	0.79	(0.31)
Season				
Winter	ref		ref	
Spring	1.49	(0.94)	1.24	(0.27)
Summer	0.98	(0.43)	0.02	(0.30)
Fall	0.48	(0.47)	−0.21	(0.29)
Interaction				
Modified * Proximate to abated surface			1.36	(0.38)
Modified * Not abated/ not proximate to abated surface			1.94	(0.41)
n	858		726	
R^2	.56		.71	

[a] Post-PbD for modified abatements is post final clean-up level where available.
[b] Standard errors are corrected for correlated residuals.
[c] Pre-PbD distribution was centered around its geometric mean.

tices were significantly lower than post-abatement levels in dwellings abated by traditional methods after adjusting for Pre-PbD and covariates (Table 2).

Data from a subset of 19 dwellings with traditional abatement and more frequent PbD monitoring showed downward trends in PbD on floors and window sills which were greatest during the first month. Window well PbD levels remained high. By three months, PbD levels at floors, window sills, and window wells were similar to levels observed in these homes at six months and in all study homes at six months (Table 1).

Six Month Dust-Lead: At six months, PbD levels were similar to, or greater than, their respective pre-abatement levels in both abatement groups (Table 1). Also, at six months, PbD tended to increase as housing condition worsened.

Blood-Lead Findings

Of 151 children eligible for PbB analysis, 78 (52 percent) (n = 46 traditional abatement; n = 32 modified abatement) were included in at least one component of the PbB analysis. The other 73 children did not have PbB determinations on venous blood at pre-abatement and at least one point in time post-abatement due to inadequate follow-up by their health care providers. No evidence was found for differences in PbB follow-up by abatement group, sex, age, race, season, housing condition, history of chelation, or location of child during abatement. Insufficient data precluded a comparison of pre-PbB levels by follow-up status.

Pre-Abatement Blood-Lead: Children from dwellings abated by traditional and modified practices had comparable pre-abatement PbBs (see Table 3) and comparable ages at abatement (traditional: 34.6 months, modified: 34.0 months). No differences were found in Pre-PbB by sex or by housing condition.

Post-Abatement Blood-Lead: Within one month of abatement, children in traditionally abated dwellings had significantly higher PbBs than children living in dwellings abated by modified practices (see Table 3). Individual increases in PbB were observed across a range of pre-PbB values from 1.16 to 2.51 μmol/L) (24 to 52 μg/dl). Overall, 13/27 (48 percent) of children in dwellings abated by traditional practices had PbB increases (defined as 0.24 μmol/L or more). Of these, nine were hospitalized for the first time for chelation therapy (PbB > 2.36 μmol/L). Of 19 children in dwellings abated by modified practices, three (16 percent) had PbB increases, two of whom were hospitalized for the first time for chelation therapy. Children with reported home contact during abatement had significantly higher post-PbB

TABLE 3—Pre- to Post-Abatement Change in Blood Lead Concentration for Children Never Chelated Pre-Abatement

	Blood Lead (μmol/L) Pre-Abatement $\bar{X}$	Pre-Abatement (se)	Post-Abatement[a] $\bar{X}$	Post-Abatement[a] (se)	Difference (95% CI)
Traditional Abatement (n = 27)	1.78	(.07)	2.11	(.13)	+0.33 (0.13, 0.53)
Modified Abatement (n = 19)	1.66	(.10)	1.71	(.12)	+0.05 (−0.09, 0.19)

[a] Post-abatement – within 1 month

than children with no reported contact (mean difference +0.35 μmol/L). In regression analysis (n = 46 children, R^2 = .65), abatement group was a significant predictor of post-abatement PbB after controlling for pre-PbB, season and whether or not the child had contact with the home during abatement.

Six Month Blood-Lead: At six months, children (traditional = 14, modified = 15) with no history of chelation therapy (PbB < 2.36 μmol/L) continued to have elevated PbBs (mean = 1.48 μmol/L) which were not significantly different from their pre-abatement PbBs (mean = 1.57 μmol/L). By six months post-abatement, five other children (three traditional abatement, two modified abatement) had experienced PbB > 2.36 μmol/L and had received chelation therapy.

Correlation between PbD and PbB: At pre, post, and six months post-abatement, children's PbBs were significantly correlated with floor and window sill PbD in their homes (Table 4).

Discussion

High levels of lead in dust were measured in homes of children with PbB > 1.4 μmol/L. Before and after abatement of lead-paint, 71 study dwellings had geometric mean PbD on floors, window sills, and window wells that were approximately 10 to 600 times greater than the PbDs reported by others in modern suburban homes and in public housing units where children had acceptable PbBs.[30,35] Study homes in the most deteriorated condition tended to have the highest PbD levels over time and the highest risk of chelation therapy (PbB > 2.36 μmol/L) among occupant children. These homes had multiple problems such as roof and plumbing leaks, holes in walls, deteriorated floors, and defective heating systems.

Most components of windows were not abated of lead-paint, and window wells remained reservoirs of very high levels of PbD during the entire study period regardless of housing condition. Most windows in study homes were deteriorated and within easy reach of young children. We observed that repeated opening and closing of old windows resulted in further deterioration of lead-painted surfaces on interior and exterior window components and contributed to the accumulation of visible dust and chips of paint on window wells. Higher interior PbD levels observed during spring and summer may be related, in part, to more frequent opening and closing of windows by occupants during these seasons. Others have also reported differences in PbD levels by season, housing characteristics, and location within rooms.[29,36,37] Soil lead was not measured in this study, although others have reported significant correlations between concentrations of lead in surface soil and interior house dust.[16,37]

Despite the implementation of improved practices, modified abatements, like traditional abatements, did not result in any long-term reductions of levels of lead in house dust or the blood of children with pre-abatement PbBs between 1.16 and 2.51 μmol/L. Traditional abatement practices also resulted in acute increases in PbD and the PbBs of children. Some received chelation therapy (PbB > 2.36 μmol/L) for the first time following abatement. With alternative abatement practices children were less likely to experience acute increases in PbB. However, post-abatement PbD levels overall exceeded the target range (<1.5 mg/m^2) despite improvements in PbD levels at most abated sites. Household respondents reported that one-third of children in both abatement groups had some contact with the home during abatement (day, night, or both) against the advice of the city health department. Home contact was associated with significant increases in children's PbB in this and other reports.[24,25] As in previous studies, a significant relationship was found between PbB and dust-lead levels.[36–38]

Inskip and Atterbury have reported high rates of deposition of lead on surfaces in close proximity to surfaces abated by methods used in this study as follows: heat gun and torches: 1 to 5 mg Pb/m^2/hour, sanding: 50 to 200 mg Pb/m^2/hour.** One dwelling in this study abated by open-flame burning and sanding was noteworthy because two study children and a third who moved into the dwelling after abatement had a total of five admissions for chelation therapy (PbB > 2.36 μmol/L) during the entire post-abatement study period.

In addition to the use of high dust-generating methods, poor containment, clean-up, and disposal practices associated with traditional abatements (Appendix A) exacerbated the problem of exposure to lead-bearing dust on household surfaces. Exposure to lead-bearing dust is particularly hazardous for children because hand-to-mouth activity is recognized as a major route of entry of lead into the body and because absorption of lead is inversely related to particle size.[39] Another study[40] showed that 21 percent of lead in house dust was found in particles of size less than 44 microns. This suggests that standard vacuums used by city crews in performing modified abatements may not have been adequate to trap lead-bearing particulates which may have been

**Inskip M, Atterbury N: The legacy of lead-based paint: potential hazards to "do-it-yourself" enthusiasts and children. Proceedings of the International Conference on Heavy Metals in the Environment, Heidelberg, West Germany, 1983. CEP Consultants, Ltd., Edinburgh, UK, pp. 286–289.

TABLE 4—Pearson Correlation (r) Over Time: Children's Blood Lead Concentrations and Geometric Mean Dust-Lead Levels in Their Homes

	Floor PbD			Window Sill PbD		
Blood Lead	r	95% CI	n	r	95% CI	n
Pre-Abatement[a]	0.35	(.08, .57)	50	0.31	(.04, .54)	50
Post-Abatement[b]	0.48	(.20, .70)	37	0.40	(.07, .66)	33
6 Months Post-Abatement[c]	0.54	(.23, .76)	30	0.53	(.18, .75)	27

[a] Includes children chelated pre-abatement (admission PbB value used) and children with no history of chelation therapy pre-abatement.

[b] Includes children chelated and discharged to abated dwellings who had re-exposure defined as PbB rebound followed by an increase in PbB to or above prior admission level with subsequent readmission (readmission PbB used for six children in floor PbD:PbB correlation and for four children in window sill PbD:PbB correlation).

[c] Includes children with no history of chelation and children six months post-chelation.

exhausted, re-entrained into the air, and redeposited.

PbD levels and the ability to reduce them appear to be related to the condition of household surfaces. Within and across study dwellings, smooth floor surfaces (e.g., vinyl tile, linoleum) tended to have lower PbD levels than wooden floors, which in our study sample were usually pitted, splintered, and worn. A relationship between surface condition and effectiveness of clean-up was found in subsequent studies which covered or coated old floors prior to final clean-up.***

The lack of long-term effectiveness for reducing lead exposure may have been due to the reliance upon abatement of certain surfaces up to four feet from the floor that children could easily reach, mouth, and chew. In this respect both the traditional and modified abatement practices reflected an outdated understanding of the etiology of childhood lead poisoning. These limited forms of abatement did not address high sources of lead-contaminated dust such as windows and left the potential for ongoing exposure to lead in paint and dust. Poorly maintained housing was observed to deteriorate further after abatement. Poor containment and clean-up practices during abatement may also have contributed to increased and ongoing exposures to lead-bearing dust on upholstered furnishings and in air ducts and vents.

Lead in paint, dust, and soil now constitute the major sources and pathways of lead exposure for US children. Recent estimates are that 42 million US dwellings contain lead-based paint, and 1.8 million US children live in deteriorated homes with lead-paint.[18] The public health goal vis-a-vis lead toxicity in children is to maintain PbBs of children and fetuses below levels associated with adverse health effects (i.e. PbB below 0.48–0.72 μmol/L).

There is a need for a new approach[41] to abating residential lead-based paint which would include: 1) more extensive, if not complete, abatement of lead-paint on interior and exterior surfaces using replacement, paint removal and encapsulation methods which prove to be safe, practical and effective in the long-term; 2) the reduction of lead-bearing house dust by the application of the following: (a) more effective clean-up measures which may well require the use of vacuums equipped with high efficiency particle air (HEPA) filters, (b) proper disposal of abatement waste, (c) the use of engineering and work practice controls to minimize and contain lead-dust produced during abatement, and (d) upgraded work practices to protect workers, occupants, their belongings, and the environment; 3) post-abatement clearance testing of lead in interior surface dust prior to reoccupancy; and 4) evaluation of costs and health and environmental outcomes of alternative abatement methodologies. The findings of this study have provided the basis for studies now in progress based upon the principles just cited. This new approach also provides for temporary relocation of families and their belongings to safe housing during abatement; worker education, training and certification; and takes into account the overall condition of dwellings, the conditions of specific painted surfaces and substrates, and other needed repairs as adopted in part in recent state regulations.[26,27] Post-abatement clearance testing for interior dust-lead prior to reoccupancy has been introduced recently in Maryland and Massachusetts using interim state standards.[26,27]

***Unpublished data, Baltimore City Health Department, Kennedy Institute

The role of lead-dust and hand-to-mouth activity in childhood lead poisoning was first recognized by Gibson in 1904.[42] In applying this knowledge to a new approach to abatement, we would do well to consider the following which Benjamin Franklin wrote in a letter to Benjamin Vaughan in 1786:

> "You will see by it that the Opinion of this mischievous Effect from Lead, is at least above Sixty Years old; and you will observe with Concern how long a useful Truth may be known, and exist, before it is generally receiv'd and practic'd on."

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APPENDIX A

Comparison of Traditional and Modified Abatement Practices

	Traditional Abatement	Modified Abatement
Extent of abatement	Up to 4 feet from floor	Up to 4 feet from floor[a]
Methods	Torch and/or sander	Heat gun
Repainting	No	Yes
Clean-up	Minimal[b]	Thorough[c]
Disposal of debris	Haphazard[d]	Off-site
Worker protection	Minimal[e]	Adequate[f]
Occupant Protection	Minimal	Some[g]
Protection of belongings	Minimal	Some[h]
Supervision of workers	No	Yes[i]

[a] Interior side trim of windows was also abated above four feet from floor.
[b] Typically dry sweeping, if done at all.
[c] Wet cleaning with high-phosphate detergent together with dry vacuuming using standard shop vacuums.
[d] Abatement debris was often left uncontained inside or outside of the home.
[e] No or inadequate respiratory protection; no blood-lead monitoring; often no protective clothing and gear.
[f] Workers were trained in use of respirators, fit tested, and provided with half mask air purifying respirators with high efficiency particle air (HEPA) filters. Overalls were provided and laundered regularly; blood lead monitoring was done.
[g] City crews had better clean up practices, and were more likely to warn parents of hazards and provide instructions on dust control. However, children in both abatement groups had some contact with the home during abatement.
[h] Furnishings and some floors were covered with tarps and kitchen items and clothes were stored in sealed closets and cabinets.
[i] City crews were supervised on-site during abatement.

APPENDIX B

Efficiency of Collection and Analysis of Lead in House Dust

Sampling was done on abated and non-abated surfaces by wiping with alcohol impregnated towelettes (14.6 cm × 20.3 cm). Dust was collected from 0.09 m^2 (one square foot) of floor surfaces using a template. Window sills and window wells were measured and alternate halves were sampled at each visit. Surfaces were wiped back and forth twice in each direction and the procedure was repeated after folding the towelette in half.

Sampling efficiency was assessed by rewiping the same surfaces up to 10 times. Sampling efficiency of a single towelette appeared to be positively related to the degree of smoothness of the surface and inversely related to the loading (total mass) on a surface. When the PbD level was less than 1.0 mg/m^2 on an initial wipe from smooth surfaces with no visible chips of paint, then non-detectable levels (<0.5 ppm lead in 20 ml extractant), or levels just above them, were usually found on the second or third towelette. When the PbD level was greater than 100 mg/m^2 on an initial wipe, and when chips and/or particulates were visible, then PbD levels greater than 10 mg/m^2 were usually detected through at least the tenth towelette.

Efficiency of elution was inversely related to the ratio of mass of sample to mass of extractant (s/e ratio). Elution efficiency at the Trace Metals Laboratory was as follows for the following standard reference materials (SRM) of the National Bureau of Standards: Urban particulate SRM #1648 (0.655 percent lead by weight): 69 to 100 percent as s/e ratio ranged from 1:67 to 1:6667; Paint SRM #1570 (11.87 percent lead by weight): 24 to 93 percent as s/e ratio ranged from 1:76 to 1:208; Orchard leaves (use due to high silica content) SRM #1571 (45 μg Pb/g): 32 to 58 percent as s/e ratio ranged from 1:14 to 1:21.

Placing Patients in the Queue for Coronary Revascularization: Evidence for Practice Variations from an Expert Panel Process

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Abstract: A panel of 16 cardiologists and cardiac surgeons rated 438 case scenarios for the maximum acceptable delay prior to revascularization, using a scale with seven interventional time frames and two nodes for designating dubious or inappropriate cases. If consensus was defined as agreement by 12 or more panelists, only 1.4 percent of the case scenarios showed consensus on a single rating. Dividing the scale into three broad clinical categories (revascularize promptly, place on a waiting list, or no intervention), 11.4 percent of scenarios showed all 16 panelists agreeing on a single category, rising to 59.4 percent of scenarios if agreement by 12 panelists was accepted as a consensus. The mean difference between the panelists' highest and lowest urgency ratings yielded waiting time differences of two weeks for scenarios of very unstable angina, and more than three months for those with stable angina. However, in a regression model, individual panelist factors on average had less effect than clinical features such as severity and stability of angina, or stenosis of major coronary arteries. These findings strongly support the need for consensus criteria to ensure that triage practices are consistent and fair, and also suggest that widespread adoption of a standardized approach to revascularization priorities may be feasible (*Am J Public Health* 1990; 80:1246–1252.)

Introduction

In many countries, limits on medical facilities and manpower make it difficult to offer rapid coronary revascularization procedures to all patients with coronary heart disease who might benefit. Cardiac surgeons and cardiologists currently assess patients according to various informal criteria in an attempt to ensure that persons with more symptoms or a higher time-dependent risk of ischemic events are revascularized expeditiously—a sorting process generally termed triage. However, no formal schema for revascularization triage has been accepted or even proposed in such a fashion as to generate widespread debate.

In the Canadian province of Ontario, waiting lists for elective coronary artery bypass surgery [CABS] during 1988 ranged between two and twelve months in five of nine hospitals with open heart surgery facilities, and the three cardiac surgery centers in Toronto had a combined waiting list of 848 patients as of January 1989.[1] These waiting times have created an ethical imperative to ensure that patients with symptomatic coronary disease at least receive attention according to perceived urgency of clinical need. Providers and the provincial government therefore agreed that formal triage criteria should be developed and applied in a pilot project for Toronto-area hospitals. To arrive at a consensus about guiding clinical principles in this area of practice, an expert panel of cardiologists and cardiac surgeons was convened. We postulated that the lack of accepted criteria for triage might be associated with considerable interpractitioner variation in whether—and where—patients were placed in the waiting lists for coronary revascularization. The formal group process followed by the panelists generated evidence about the nature and extent of inter-practitioner agreement and disagreement. We herein present this evidence and discuss its implications for promotion of a uniform approach to triage practices.

Methods

Panel Process

The group process followed by the revascularization panel was a variant on techniques used by researchers at the RAND Corporation[2–4] and the Canadian National Consensus Conference on Aspects of Cesarean Birth.[5,6] The 16-man panel included five cardiovascular surgeons from academic centers; six cardiologists practicing in academic centers, three of whom had extensive experience with percutaneous transluminal coronary angioplasty [PTCA]; and five cardiologists practicing in community hospitals that refer patients to the Toronto academic centers offering CABS or PTCA (Appendix A). Drawing on pre-circulated background materials, the panelists met for a day in October 1988 and agreed on factors that would be most important in determining urgency of need for revascularization; levels within each factor were also set out (Appendix B). An urgency rating scale was adopted, with several time-frames for revascularization and two appropriateness ratings for indicating those instances where triage to revascularization seemed questionable or contraindicated (Appendix C).

After the first panel meeting, an Urgency Rating Questionnaire was compiled, permuting the factors agreed upon by the panelists into patient case scenarios. Implausible scenarios were eliminated, and levels of given factors deemed unlikely to affect decision-making were eliminated or combined, leaving 438 separate case scenarios (sample materials available on request). Case scenarios were oriented primarily to urgency ratings, in that all patients had at least significant single vessel disease and all were presumed to have clinically unequivocal angina. The questionnaire was organized into five chapters according to the level of what was *a priori* believed to be the most important factor—the pain syndrome, defined as the severity or stability of anginal symptoms, and

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response of those symptoms to medical therapy (Appendix B).

The panel had agreed that a single set of criteria would be developed for revascularization in general, pending the later generation of a separate set of triage criteria for PTCA. Panelists were therefore to rank scenarios with a view to employing either PTCA or CABS, depending on the clinical features. The majority of scenarios, however, were anatomically appropriate for CABS rather than PTCA (Appendix B). Panelists were instructed that the waiting period should be that deemed reasonable given: the presumed risk of death from ischemic events due to delayed revascularization, as suggested by clinical experience and evidence from a precirculated literature review; the presumed ongoing morbidity as a result of anginal symptoms; and other relevant factors as determined by the individual panelist. They were specifically enjoined to approach the questionnaire with an understanding that resources for coronary revascularization were in competition with other worthy programs. The waiting period was presumed to start from the time that anatomical data from angiography were available in the presence of appropriate symptoms of angina.

To help ensure a shared body of knowledge, a literature review was compiled to accompany the Urgency Rating Questionnaire. This review focused on any known temporal differences in risk of death from ischemic events for patients receiving medical rather than surgical therapy, and was organized according to each of the agreed-upon factors, singly and/or in combination with other factors. Panelists were urged to read the document prior to completing the questionnaire, and were also supplied with an executive summary of it for ease of reference.

Our process differed from that developed by the RAND researchers in two key respects. The Ontario triage panelists met initially to agree on the scale and the factors to be incorporated into the Urgency Rating Questionnaire, completed the questionnaire, received by mail their individual ratings as compared to all other panelists, and finally met again to arrive at consensus criteria for triage practices. This process actually reduced one potential source of interpractitioner disagreement—rejection of the plausibility of either the factors used to construct the case scenarios, or the combinations of factors into particular case scenarios. In contrast, Park, *et al*, report that indications lists were substantially revised by the RAND panelists when they met to review their appropriateness ratings for various case scenarios.[2] The other difference in process was re-rating of scenarios. The RAND panelists rated their scenarios twice: first independently, and again at a panel meeting, taking into account their results as compared to other panelists. The Ontario panelists rated scenarios once, without knowledge of their peers' ratings.

Statistical Analysis

With 16 panelists rating 438 case scenarios, there were only eight missing ratings out of a possible 7,008; appropriate allowance was made for them in all analysis. Descriptive measures of agreement and disagreement are explained with the relevant results. More formal statistical analysis involved two methods: an index of agreement for multiple raters and intraclass correlation coefficients.

The agreement index (Appendix D) was constructed to estimate the probability that two randomly chosen panelists, drawn without replacement, would agree on the classification of a randomly chosen scenario. This index—termed P_a—was calculated for the entire set of scenarios, and for each level of angina symptomatology, hereafter termed, "pain syndrome." Agreement was calculated both for exact scale ratings and within three sets of scale ratings (raings 1–4, 5–7, and 8–9) representing similar clinical decisions. The agreement ratings are reported both as overall levels of agreement and corrected for chance agreement between panelists, i.e., a kappa correction.[7] This correction reduces the agreement probability by accounting for the overall likelihood of any two raters agreeing by chance, given the overall pattern of responses across the entire set of 438 scenarios.

The indices of agreement do not penalize panelists for greater scale disagreement. That is, be it with respect to exact agreement or agreement by approximate clinical approach, two randomly drawn raters scoring a scenario for intervention within 24 hours ("extremely urgent") versus intervention within 3–6 months ("marked delay") are both recorded simply as disagreeing, but the extent of this discrepancy is not captured. To reflect the ordinal nature of the rating scale, intraclass correlation coefficients [ICCs] were calculated.[8] Analogous to weighted kappa statistics, the ICCs show the expected numerical correlation between the ratings of two randomly chosen panelists rating the same randomly chosen scenario. For ease of analysis, we assume that the two non-interventional ratings ("questionable" and "inappropriate") follow after the final time-node, "marked delay." This simplifying assumption tends to increase the correlation between panelists' ratings in case scenarios with scores reflecting low urgency, and reduces correlation in scenarios with high urgency. However, differences between "inappropriate" and "marked delay" are arguably much less important than between the former and "extremely urgent."

Linear regression analysis[9] was also performed to estimate the relative impact of practitioner versus clinical factors. To facilitate analysis, the entire scale was again treated as ordered (see above for justification).

Results

Descriptive Grouping of Ratings

For each scenario, one may either consider the number or proportion of panelists agreeing on a given set of contiguous rating categories (scale agreement) or the number of contiguous ratings required to encompass the ratings of a given number of panelists (panel agreement). Thus, consensus consists of panel or scale agreement, or both. Complete agreement on a single rating by all panelists never occurred. If the definition of panel agreement was relaxed to include as few as 12/16 panelists, i.e., agreement by at least 75 percent of the panel, then only 6/438 (1.4 percent) of the case scenarios showed consensus on a single rating. With a further relaxation of the definition of panel agreement, to a simple majority or ≥ nine panelists, agreement on a single rating rose to 102/438 scenarios (23.2 percent). Relaxed definitions of scale agreement were also applied, with the entire nine-point scale taken as contiguous. To encompass the responses of all panelists 50 percent of the time, it was necessary to relax the definition of scale agreement to five contiguous points. On the other hand, by allowing some latitude in definitions of both scale and panel agreement, frequent clustering of responses was evident. For example, at least three-fourths of panelists agreed within a three-point range in over 80 percent of cases.

Indices of Exact Scale Agreement

The probabilities that any two randomly drawn panelists would agree on a particular rating for a given scenario are

shown in Table 1 according to each pain syndrome and overall. The exact agreement is consistently at the 25–30 percent level, falling somewhat only for the highest urgency scenarios—unstable angina with persisting symptomatology requiring parenteral therapy for control. The degree of agreement corrected for chance is poor.

Because our primary concern was with urgency rather than appropriateness, these indices were recalculated including only the time-related ratings between "emergency" (node 1) and "marked delay" (node 7), i.e., eliminating appropriateness as a source of disagreement. The resulting agreement on urgency ratings ran 30–40 percent, and was only moderately higher than in the total scale case (data available on request to author).

Indices of Approximate Scale Agreement

Clinical divisions: The first four ratings on the scale (Appendix A) imply that patients will be revascularized on the same admission if hospitalized for medical therapy or catheterization, or readmitted within two weeks; whereas the next three ratings will be on outpatient waiting lists. We term these two categories respectively "DO" and "WAIT", and the last two non-interventional ratings, "NOT".

Only 11.4 percent of scenarios showed all 16 panelists agreeing within one of these three clinically pertinent categories. However, if panel agreement was re-defined as congruence among ≥12 clinicians, categorical agreement was found for 59.4 percent of scenarios. Moreover, as shown in Table 1, the overall P_a increased greatly, rising to about 65 percent. Thus, there was evidence of a general agreement on a general approach to these patients. The probabilities of agreement between two randomly drawn panelists were even higher (82 percent) when appropriateness ratings were eliminated, leaving only the clinical dichotomy of "DO" versus "WAIT" (data on request to author).

When divided by pain syndrome, the indices reveal a significantly higher degree of agreement for the most unstable patients—the opposite of the trend when exact agreement was sought (Table 1). This enhanced agreement for an overall approach was attributable to the fact that patients still on parenteral therapy obviously could not be discharged from hospital, would almost invariably fall into the "DO" category, and only occasionally be rated as "WAIT". The lower index for the exact ratings was due to poor agreement among panelists within the first four ratings; only four panelists rated any scenarios at node 1 ("emergency").

It should be noted that there was considerable difference among panelists in frequency of use of "NOT" ratings—"questionable" or "inappropriate". While 10 panelists rated between 49 and 105 scenarios in "NOT" categories, the extent of disagreement about appropriateness is suggested by the fact that the other six applied "NOT" ratings to 1, 20, 37, 135, 172, and 200 scenarios respectively. One way of examining divisions of opinion about appropriateness is to consider the proportions of scenarios where ≥4 panelists rate "DO" and ≥4 rate "NOT". Broken down by pain syndrome, the percentages of scenarios with this pattern of disagreement were as follows: mild to moderate stable angina, 4.5 percent; severe stable angina, 1.6 percent; unstable angina with adequate restabilization on oral medical therapy, 4.4 percent; unstable angina with incomplete stabilization on oral medical therapy, 24.2 percent; and unstable angina requiring ongoing parenteral medication, 16.7 percent. Note that the latter two sections of the Urgency Rating Questionnaire were actually identical apart from the pain syndrome. The reduced frequency of "NOT" versus "DO" disagreement was accordingly due entirely to the fact that patients requiring parenteral therapy were believed so ill that the risk-benefit ratio of revascularization was deemed more uniformly acceptable, leading to greater consistency in application of "DO" ratings.

Numerical Scale Association: Intraclass correlation coefficients are also displayed in Table 1. When separated by the five levels of pain syndrome, the intraclass correlations sharply decrease with more severe or unstable angina. This is similar to the situation for exact scale agreement using the non-ordinal index of agreement, and is due to clustering of ratings (e.g. 88 percent of panelists' ratings are between time-nodes 1 and 4 for the most severe pain syndrome). Hence, while there is relatively low variability between scenarios, there is also little overall agreement on the exact timing of revascularization. As well, any disagreement about appropriateness further reduces the correlation coefficient, because of numerical distances between scale points under an assumption of ordinality (e.g., time-nodes 1-4 versus non-intervention nodes 8-9).

Agreement and Disagreement by Individuals and Subgroups

There were no significant differences in total absolute distance from the median scenario ratings or frequency of

TABLE 1—Agreement on Urgency/Appropriateness Rating

	Stable Angina Symptoms on Medical Therapy		Unstable Angina Symptoms on Medical Therapy			
	Mild-moderate	Severe	Resolved	Partly Resolved	Persisting	Overall
Exact						
P_a*	0.30	0.31	0.29	0.30	0.25	0.29
SE (P_a)	0.01	0.01	0.01	0.01	0.01	0.01
Kappa	0.18	0.19	0.17	0.18	0.12	0.16
Clinical Category#						
P_a	0.58	0.65	0.56	0.56	0.81	0.65
SE (P_a)	0.02	0.02	0.02	0.01	0.01	0.01
Kappa	0.33	0.44	0.30	0.29	0.70	0.43
Scale Correlation ICC+ (random effects)	0.49	0.51	0.40	0.16	0.13	0.51

*P_a = probability of agreement for two randomly drawn raters on a randomly chosen scenario; the kappa correction is unweighted. #Scale collapsed into three clinical categories, "DO" within two weeks (time-nodes 1–4); "WAIT" up to six months (time-nodes 5–7); and "NOT" triaged (ratings 8 for "questionable" or 9 for "inappropriate"). +ICC = intraclass correlation coefficient, analogous to a weighted kappa.

outlier status (defined as being greater than one scale point from the median rating) according to practitioner experience, academic or non-academic center for practice, and surgical versus cardiological orientation.

The Practitioner Factor in Context

We used regression techniques to determine, within each pain syndrome and for each practitioner, the influence of practitioner variability as opposed to that stemming from the clinical factors incorporated into the questionnaire. Results for the three panelists with largest and smallest absolute coefficients are shown in Table 2, showing respectively systematic departure from, and congruence with, the mean ratings of the group for any given scenario.

Table 3 presents the regression coefficients for various clinical factors, as well as the median absolute value of the panelist coefficient for each pain syndrome. As suggested by the change *across* chapters due to the pain syndrome alone, this was the dominant determinant of ratings. The influence of the panelists is suggested by comparing the magnitude of the regression coefficients for clinical factors *within* any given pain syndrome to the regression coefficients for the median panelist. The latter is significantly smaller than key clinical variables such as pain syndrome or stenosis of major coronary arteries (standard errors are approximately 0.10 for all coefficients, including the panelist factor.) However, as indicated in Table 2, some panelists did systematically differ from the mean panel rating to a much greater extent.

Urgency Rating Discrepancies in Isolation

Beyond the indices of agreement on urgency ratings calculated above, we sought also to quantify the extent of discrepancies among panelists rating a given scenario between 1 and 7. Two measures were calculated: the maximum discrepancy of ratings within each scenario, and the discrepancy between the extreme ratings when the highest and lowest 25 percent of ratings were eliminated for each scenario. (The number of ratings deleted at each extreme of the sorted ratings was the largest integer less than or equal to the total number of ratings/4). All urgency ratings could, on average, be contained within 3.5 points on the scale, while the majority could be contained in less than two points on average. These results are displayed in Table 4.

TABLE 2—Influence of Panelists on Ratings: Regression Coefficients for Panelists with Least and Greatest Systematic Departure from Group Mean

	Level of pain syndrome*				
	1	2	3	4	5
Greatest Influence					
	−1.14	−0.84	−1.14	1.32	1.79
	0.66	0.82	1.31	1.79	0.84
	−0.93	−0.87	−0.47	−1.71	−1.42
Smallest Influence					
	−0.32	0.35	0.30	−0.31	−0.20
	0.07	0.27	0.14	0.24	0.11
	0.19	−0.04	−0.11	−0.31	−0.14
Standard Errors	0.12	0.11	0.13	0.11	0.14

*Levels of pain syndrome are in the same order as Appendix B. Note that a positive coefficient implies higher numerical score, i.e., less aggressive management; a negative coefficient has the opposite implication. Coefficients reflect influence within a model where, theoretically, the minimum value possible is 1.0 and the maximum is 9.0. However, no mean scenario rating was ever <2.0 and only a few within the mildest pain syndrome exceeded 8.5.

TABLE 3—Impact of Various Factors on Mean Ratings: Regression Coefficients

Chapter of questionnaire	1	2	3	4	5
Pain Syndrome (base case for other factors)*	8.01	7.09	6.98	4.19	2.83
Univariate Departures from Base Case					
Anatomy					
Left main disease	−2.57	−2.34	−2.06	−1.33	−1.05
3 vessel disease	−1.09	−0.53	−1.06	−0.57	−0.54
Left Ventricular Function					
Grade IV	0.08	0.10	0.28	0.77	0.73
Non-invasive Tests					
High risk	−1.34	−1.07	−1.20	NA	NA
Recent MI	NA	NA	NA	0.16	0.26
Previous CABS	NA	NA	NA	0.44	0.49
Median Panelist (absolute value)	0.55	0.33	0.49	0.40	0.31

Notes: Definitions for clinical factors are explained in Appendix B. The model presupposes a baseline case incorporating the pain syndrome. For pain syndromes corresponding to angina classes I-II, III, and IVA, the baseline anatomic factor is single or double vessel disease without left anterior descending arterial stenosis; left ventricular function is graded I-II; non-invasive tests suggest a low or average ischemic risk; and the expected procedural morbidity and mortality is low. For unstable angina without adequate symptomatic stabilization (classes IVB and IVC) neither non-invasive tests nor the distinction between left ventricular grades I and II apply, and the relevant base case is "No recent myocardial infarction" and "No previous CABS." All standard errors are approximately 0.10 (ranging between 0.05 and 0.14) except for Left ventricular grades I and II, where the standard error within angina class IVA is 0.19 in both cases. The coefficients for univariate changes in baseline factors illustrate their influence within a given pain syndrome.

TABLE 4—Widths of Ranges Encompassing Urgency Ratings in Isolation

Minimum number raters rating 1–7 for inclusion of the scenario	No Minimum	≥8 Raters	≥12 Raters
Number scenarios	438	392	327
Mean number rating "1"–"7"	12.85	13.84	14.65
Range (all raters)	3.55	3.69	3.72
Range (trimmed of high and low quartiles)	1.92	1.93	1.90
Mean residual number of ratings in trimmed result	7.12	7.59	7.95

Discussion

Although American[10] and British[11] RAND panels have previously graded the appropriateness of CABS, those evaluations were based upon 1981 indications, and involved ratings on a nine-point appropriateness scale—clearly distinct from our urgency scale with seven different time intervals and two appropriateness categories. It is nonetheless illuminating to compare similar agreement indices for our 16-person panel to those reported for the nine-person RAND panel. If unanimity is sought, the inter-practitioner agreement of the triage panel is about on a par with the first ratings of the RAND panel, and significantly lower than occurs with a second rating by the RAND panel once they knew their colleagues' ratings. However, in a comparison excluding extreme high and low ratings, agreement among the Ontario triage panel is superior to the first ratings and about on a par with the second ratings of the RAND panel (data available on request). This suggests an influence of outliers in the Toronto panel.

We have two suggested explanations for the outlier phenomenon. The first is that the structure of the scale may have been contributory. That is, outliers will be created when some panelists rate a scenario as relatively urgent (time-nodes 1 to 4) while others question the risk-benefit ratio and need for intervention (ratings 8 and 9). The second possibility is that a larger panel may increase the likelihood that one or more panelists will produce idiosyncratic ratings for any given scenario. We could not detect effects according to practitioner experience, site of practice (academic versus non-academic), or surgical versus cardiological orientation. However, the actual number of raters in any identifiable subgroup was small enough to limit our statistical power for detecting such effects.

Because our ratings reflect the initial clinical judgments of cardiologists and cardiac surgeons in the absence of knowledge about their peers' ratings, they offer an indication of the extent of inter-practitioner variation that might occur in actual practice. Moreover, inter-practitioner variation in ratings would be minimized by several features of the exercise followed here. All panelists practiced in urban centers, and were Canadian-certified specialists in cardiology or cardiac surgery. The majority did some specialty training at the University of Toronto, and practiced in or near Metropolitan Toronto. All received the same literature review summarizing how various factors influenced any time-dependent risk of death or myocardial infarction stemming from delay of mechanical revascularization. As already noted, all had agreed to the factors that were to be considered in rating case scenarios, and rated patients according to a standardized scale. All reviewed exactly the same case material, with none of the discrepancies that might occur in actual practice when different doctors assess patients and their test results. It is therefore very unlikely that the results of this exercise over-estimate the extent of inter-practitioner variation in managing this patient population. We suspect that larger discrepancies would be demonstrable on review of actual triage practices in Canada and other countries.

The magnitude of those discrepancies, as demonstrated here, is striking. Consider the following three examples from our results. First, even if agreement among a simple majority of panelists was accepted as reasonable, convergence on a single rating occurred in only 23 percent of scenarios. Second, if we divided the scale into three broad clinical categories—i.e. revascularize promptly, place on a waiting list, or do not plan intervention—only 11.4 percent of scenarios showed all 16 panelists agreeing within one of the three clinically pertinent categories. Third, even after eliminating disagreement about appropriateness of revascularization, the average difference between highest and lowest urgency ratings for the scenarios was 3.5 scale points out of a possible 7—corresponding to time differences ranging from two weeks for the most unstable patients, and on out to more than three months for those with stable angina.

In our view, these discrepancies are at odds with the aims of Canada's universal health care system. The architects of the Canadian health insurance system emphasized the concept of fairness, with the goal being that citizens should receive health services according to their needs.[12] A potential drawback to national health care systems is centrally constrained resources, with the substitution of rationing by queue for the rationing by price or income that occurs in settings of private prepayment for medical services; this criticism has been leveled against the British National Health Service.[13] However, one off-setting advantage of rationing by queue is that physicians are able to rank order patients in the queue according to judgments about the urgency of clinical need—arguably a fairer approach than priorities based on incomes or influence. Our findings raise the concern that this potential advantage is not being fully realized for coronary revascularization in the Canadian system.

While appropriateness issues were not the focus of this exercise, one logical line of attack on waiting lists is to ensure that all those waiting would indeed benefit from planned procedures. The CABS rates in Ontario have plateaued over the last several years. Anderson and Lomas have observed that the rates are being maintained largely by increasing application of CABS to patients over 65 years of age.[14] It is unclear whether the increasing use of CABS in the elderly represents an extension of utilization patterns to a group with a less favorable risk-benefit ratio, or an appropriate shift in utilization as the procedure becomes safer for elderly patients. The inter-rater disagreement on appropriateness ratings observed here suggests that a formal appropriateness panel may also prove worthwhile. In any event, creation of a fairer and more rational queue is a logical complement to any strategy that seeks to optimize resource utilization.

Despite the above-noted variations, at least three-fourths of panelists did agree within a three-point range on the urgency rating scale in over 80 percent of cases. When clinically pertinent categories were considered, any two randomly drawn panelists would rate scenarios in the same category two out of three times. Furthermore, weighed against the clinical variables, the panelists had much less importance as a determinant of ratings than such factors as severity and stability of anginal symptoms, non-invasive test results, or stenosis of major coronary arteries. In contrast, when Goyert, *et al*, examined practices of 11 obstetricians at a single hospital, only nulliparity exceeded practitioner identity as a risk factor for operative delivery.[15]

The revascularization panel's underlying agreement about determinants of urgency proved sufficient to permit a consensus on general principles governing triage of this patient population when the panelists convened in January 1989.[16] These guidelines are already being applied in a pilot Toronto program, were presented to a province-wide conference in November 1989, and will be undergoing further review by a multi-center working party during 1990. The ultimate goal is to standardize triage criteria voluntarily across the province so that inter-practitioner and inter-institutional discrepancies in waiting times can be reduced in the interests of fairness and efficiency.

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APPENDIX A

Expert Panel of Cardiologists and Surgeons

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APPENDIX B

Factors and Levels Adopted by the Cardiology Panel for Patient Triage

A. Presenting pain syndrome and therapeutic response

1. Stable angina on reasonable medical therapy: mild to moderate [Canadian Cardiovascular Society classes I-II][17]
2. Stable angina on reasonable medical therapy: severe [Canadian Cardiovascular Society class III.][17]
3. Unstable angina, pain resolved with intensified medical therapy, and now on oral medication.
4. Unstable angina, symptoms improved but angina with minimal provocation.
5. Symptoms persisting or hemodynamically unstable, requiring coronary care monitoring and parenteral medication.

B. Coronary anatomy defined by angiography

1. Left main coronary artery stenosis with or without other stenoses.
2. Two or three vessel disease, *including* the proximal left anterior descending [LAD] artery or equivalent.
3. Three vessel disease *without* involvement of the proximal LAD artery.
4. Single vessel disease involving the LAD or equivalent vessel.
5. Single or two vessel disease *without* a proximal lesion of LAD artery or an equivalent vessel.

C. Expected procedural morbidity and mortality

1. High (e.g., renal failure, diabetes, peripheral vascular disease, chronic obstructive airways disease).
2. Average/Low

D. Reversible ischemia on non-invasive tests

1. *High risk:* e.g. poor performance on treadmill testing, with early and unequivocal ST-segment changes on electrocardiogram; thallium defects on exercise or dipyridamole stress testing involving either an extensive portion of the anterior wall or multiple areas.
2. *Low* risk: any test failing to meet the above criteria.

E. Left ventricular ejection fraction

1. >50%, approximately grade 1
2. 30/35%–50%, approximately grade 2
3. 20%–30/35%, approximately grade 3
4. <20%, approximately grade 4

F. Previous CABS: Yes/No

G. Myocardial infarction within previous 4 weeks: Yes/No

APPENDIX C

Urgency/Appropriateness Rating Scale Accepted by the Expert Cardiology Panel

1.	Emergency	Immediate revascularization
2.	Extremely urgent	Within 24 hours
3.	Urgent	24–72 hours
4.	Semi-urgent	72 hours to 14 days (same admission)
5.	Short list	2 weeks to 6 weeks
6.	Delayed	6 weeks to 3 months
7.	Marked Delay	3 months to 6 months
8.	Questionable if revascularization should be undertaken	
9.	Inappropriate for revascularization to be undertaken	

Note: Each urgency rating level represents the period within which revascularization is expected to be performed; the outer time limit for each limit represents the maximum acceptable waiting period for patients assigned that urgency rating.

APPENDIX D

Calculation of Agreement Indices

The agreement index was determined as in Davies and Fleiss[7] to calculate the probability that two panelists chosen randomly (without replacement) would agree on the rating of any scenario chosen with equal probability from all possible scenarios.

Thus, the $\binom{16}{2}$ pairings of raters were assessed for agreement on each scenario. Agreement was defined as either strict agreement on urgency scale, or agreement on the clinical categories "DO", "WAIT", and "NOT" (see text for definitions).

Hence, if $I_n(r_1, r_2) = 1$ for r_1 and r_2 agreeing on scenario "n" and 0 otherwise, then:

$$P_a = \frac{\sum_n \sum_{r_1<r_2} I_n(r_1, r_2)}{n\binom{16}{2}}$$

Note that "chance agreement" between two panelists is possible. This is calculated as

$$P_c = \frac{\sum_j \sum_{r_1<r_2} P_j(r_1)\, P_j(r_2)}{\binom{16}{2}}$$

where $P_j(r)$ is the marginal probability that the rating of panelist "r" is "j".

Thus, $P_k = \frac{Pa - P_c}{1 - P_c}$ is the chance corrected, or "kappa" agreement.

Note that kappa may be calculated either for each chapter or over all the scenarios, irrespective of chapter. The variance of the agreement indices is readily calculated and computed, but is unwieldy. (Available on request to author.)

Patient, Provider and Hospital Characteristics Associated with Inappropriate Hospitalization

ALBERT L. SIU, MD, MSPH, WILLARD G. MANNING, PHD, AND BERNADETTE BENJAMIN, BS

Abstract: To determine the relation between patient and provider characteristics and inappropriate hospital use, we examined adult nonpregnancy hospitalizations from a randomized trial of health insurance conducted in six sites in the United States. Appropriateness of inpatient treatment was based on medical record review; patient characteristics on sociodemographic, economic, and health status; and provider characteristics on descriptors of physician practice and hospital facilities.

Twenty-seven percent of admissions attended by physicians licensed for more than 15 years were judged inappropriate, compared to 20 percent for younger physicians. Admissions were more likely to be inappropriate if the patient was female (27 percent compared with 18 percent). Controlling for patient and provider characteristics reduces but does not eliminate the differences in the appropriateness of inpatient care across the study's six sites. Differences in available provider and patient characteristics do not account for geographic differences in inappropriate hospitalization in this study. (*Am J Public Health* 1990; 80:1253–1256.)

Introduction

As part of a wider concern for health care cost containment, recent attention has focused on the inappropriate use of hospital care. Recent studies have documented substantial inappropriate hospital use for adults, as well as children.[1–5] In the RAND Health Insurance Experiment, we reported previously that 23 percent of the admissions made inappropriate use of the acute hospital setting or level of care, while an additional 17 percent of admissions were avoidable by use of ambulatory surgery.[2] In this report, we examined the effect of patient, physician, and hospital variables on inappropriate hospitalizations and days. Our data provide some of the most detailed and comprehensive information available on the health status of patients.

Methods

The health insurance experiment was a randomized trial of cost sharing in health insurance conducted in six United States sites. Families entered the experiment between 1974–77 and were assigned to one of 14 different fee-for-service health insurance plans with different levels of cost sharing. The experiment excluded persons 62 years of age and older at the time of enrollment, those with incomes in excess of $25,000 in 1973 dollars, those eligible for Medicare disability, those in the military and their dependents, veterans with service-connected disabilities, and individuals imprisoned or institutionalized. Additional details on the design of the Health Insurance Experiment are available elsewhere.[6,7]

The sample of 3,413 individuals used as a basis for this analysis includes all enrollees who were 18 years of age or older at enrollment into the study. Medical records were obtained from 90 percent of the hospitalizations for these individuals. We excluded admissions for labor and delivery, psychiatry, and alcohol rehabilitation. This resulted in a sample of medical records from 1,132 admissions.[2]

To determine the appropriateness of hospital admissions or hospital days, two board-certified physicians reviewed medical records using the Appropriateness Evaluation Protocol.[3,8] They classified each admission or randomly selected hospital day into one of three categories: appropriate, inappropriate, or avoidable by ambulatory surgery. In this report, however, we considered admissions avoidable by ambulatory surgery as appropriate for two reasons. Ambulatory surgery centers were not available in all sites during the study period. Further, in Siu, *et al*,[2] the findings were not sensitive to whether ambulatory surgery was considered appropriate or inappropriate. Additional details on the review of hospital records for appropriateness are available elsewhere[2] or on request to the authors.

We use four groups of variables to explain the variation in inappropriate inpatient care: insurance plan, patient health status, patient sociodemographic and economic measures, and provider characteristics. We group the insurance plans into three groups: 1) the free plan (no out-of-pocket cost to the family); 2) the family cost sharing or pay plan, where the coinsurance rate is 25–95 percent for all services up to an upper limit on out-of-pocket expenses; and 3) the individual deductible plan with a 95 percent coinsurance rate for outpatient services (subject to an out-of-pocket limit) and free inpatient care. We use three measures of health status: 1) general health perceptions; 2) physical or role limitations; and 3) chronic disease status. Each measure is based on the self-administered Medical History Questionnaire completed at the beginning of the study. The General Health Index measures perceptions of overall health, as well as resistance to illness and health worry.[9,10] The physical or role limitations measure is scored dichotomously (1 = limited, 0 = otherwise) to indicate the presence of one or more limitations in self-care, mobility, physical activity, and usual role activities; the chronic disease measure is a simple count of the number of diseases or health problems.[11–14] The model used in our analysis also includes covariates for study site, patient age, sex, race, education, and family income and size. With the exception of family income, the data were collected before or at enrollment in the study (1974–77). Family income data are from 1975 in Dayton, 1978 for the three-year group in South Carolina, and 1976 for all other participants.

Variables describing the hospitals were obtained from the American Hospital Association files. This included information regarding accreditation, the presence of a residency program, medical school affiliation, ownership, number of beds, number of admissions, occupancy rate, and available facilities. Information on attending physicians was

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obtained from the American Medical Association Directory of Physicians' Master File. For osteopaths, we obtained information from the Directory of the American Osteopathic Association. We located 95 physicians missing from these two sources by way of the computerized National Yellow Pages. For this latter sample of physicians, information on their characteristics was obtained from their practices by phone interview. These sources yielded information on physician sex, years from licensing, type of practice, practice arrangement, self-designated specialties, and board certification.

Although we obtained patient and hospital descriptors on all 1,132 admissions for whom medical records were available, some information is missing: nine percent for specialty, 12 percent for licensing, and 19 percent for solo/group practice data. Missing data were replaced by the mean value for that variable in regressions, and dummy variables were included to indicate cases with data missing for a given variable. The Appendix lists the independent variables used in this analysis and their means.

Statistical Methods

We used a logistic regression to estimate the probability that a particular admission or hospital day was medically inappropriate. The dependent variable in these regressions is whether the admission or day is inappropriate. For different groups (e.g., site, sex, specialty), we make both unstandardized and standardized comparisons. For the unstandardized comparisons, we predict the mean probability (and calculate its standard error) without correcting for the other differences between the contrast groups. For example, in comparing Seattle with one of the rural sites, we do not adjust for the differences in the mix of physicians or hospital types. We also estimate standardized predictions that remove the effects of factors confounded with the contrast of interest. For example, we want to purge the Seattle-rural site comparison of differences in patient and provider mix. To do this, we predict the probability that each admission or day is inappropriate if that case were in Seattle, then as if it were in Dayton, etc. This procedure is like the usual direct adjustment, except that it is based on the estimated model that controls for several factors simultaneously. All inference statistics are corrected for intratemporal correlation, as well as correlation among providers and family members.[15] We report standardized predictions and 95 percent confidence intervals (CI) for differences between categories of independent variables; p-values are reported in the text for the overall effect of statistically significant independent variables with multiple categories.

Results

As previously reported, we found that 23 percent of the adult hospital admissions were inappropriate and 35 percent of the days were inappropriate.[2] Logistic regression with hospital admission inappropriateness as the dependent variable showed that cost-sharing had no effect on the proportion of admissions that were inappropriate. However, there were differences by site ($p = 6.2 \times 10^{-5}$), gender, education ($p = 0.03$), hospital accreditation and bed size, and physician specialty ($p = <10^{-8}$) and length of time since licensing.

Unstandardized estimates showed that a greater likelihood of an inappropriate hospital admission was associated with a number of variables, but these differences could be due to the intrinsic differences among the groups compared or to other factors that differ among the groups. The standardized comparisons control for these other differences. The standardized predictions in Table 1 demonstrate that the proportion of admissions considered inappropriate are increased substantially if the physician was licensed more than 15 years ago or if the patient was female, and increased moderately if the attending physician was either an internist or a general/family practitioner.

Logistic regression showed that cost sharing had no effect on the appropriateness of a hospital day. Site differences persisted ($p = 0.03$) although they were smaller. Among the patient variables, only sex remained associated, and none of the hospital variables remained associated; years from licensing remained associated among the set of physician variables.

For unstandardized comparisons, the likelihood of a hospital day being inappropriate is increased if the physician was not board certified or was licensed more than 15 years ago, if the hospital was nonpublic, and if the patient was female. The standardized estimates (Table 2) are similar to those for inappropriate admissions although years from licensing and patient gender are more strongly associated and physician specialty less strongly associated.

There were substantial unstandardized differences across sites in the inappropriate use of inpatient care. In the case of both hospital admissions and days, the lowest proportion of inappropriate use is found in Seattle. Standardizing for patient, physician, and hospital variables reduced but did not eliminate the variation among sites in inappropriate use (Table 3). Addition of these explanatory variables reduced the variation resulting from site differences from 5.1 percent to 2.6 percent for hospital admissions and from 2.4 percent to 1.6 percent for inappropriate days. This is most

TABLE 1—Inappropriate Admissions by Provider and Patient Characteristics

Characteristics	Standardized Percentage	Difference (95 % CI) versus Initial Category
Specialty		
General/family	33.8	—
Internal Medicine	34.0	0.2 (−10.1, 10.5)
Surgery	17.3	−16.5 (−25.7, −7.3)
Obstetrics/gynecology	8.1	−25.7 (−34.6, −16.8)
Solo Practice		
Yes	23.2	—
No	23.2	0.0 (−6.3, 6.3)
Board-Certified		
Yes	21.4	—
No	27.5	6.1 (−1.1, 13.2)
Years from Licensing		
≥15	26.8	—
<15	20.0	−6.8 (−12.8, −0.8)
JCAH Accredited		
Yes	21.9	—
No	32.4	10.5 (−0.4, 21.4)
Teaching Status		
Yes	24.0	—
No	22.9	−1.1 (−11.5, 9.3)
Public Hospital		
Yes	20.9	—
No	23.3	2.4 (−9.4, 14.2)
Sex		
Male	17.8	—
Female	26.8	9.0 (4.6, 13.4)
Education		
High School	36.7	—
<High School	36.5	−0.2 (−8.4, 8.1)
Some College	29.4	−7.3 (−17.9, 3.3)
College	27.7	−9.0 (−21.2, 3.3)

TABLE 2—Inappropriate Days by Provider and Patient Characteristics

Characteristics	Standardized Percentage	Difference (95% CI) versus Initial Category
Specialty		
General/family	39.7	—
Internal Medicine	36.6	−3.1 (−14.9, 8.7)
Surgery	30.0	−9.7 (−20.3, 1.0)
Obstetrics/gynecology	33.3	−6.4 (−19.8, 7.1)
Solo Practice		
Yes	34.4	—
No	32.4	−2.0 (−10.6, 6.5)
Board-Certified		
Yes	31.6	—
No	39.2	7.6 (−1.0, 16.3)
Years from Licensing		
≥15	39.0	—
<15	29.9	−9.1 (−17.0, −1.2)
JCAH Accredited		
Yes	33.7	—
No	41.2	7.5 (−6.6, 21.6)
Teaching Status		
Yes	36.5	—
No	33.9	−2.6 (−16.3, 11.0)
Public Hospital		
Yes	30.3	—
No	34.9	4.6 (−10.9, 20.0)
Sex		
Male	23.5	—
Female	42.0	18.5 (10.9, 26.1)

TABLE 3—Inappropriate Hospital Use by Site

	Admissions		Days	
Site	Standardized Percentage	Difference (95% CI) vs Initial Category	Standardized Percentage	Difference (95% CI) vs Initial Category
Seattle	11.4	—	22.1	—
Dayton	20.3	8.9 (0.1, 17.7)	45.7	23.7 (9.2, 38.1)
Fitchburg	17.4	6.0 (−2.2, 14.3)	33.6	11.5 (−2.3, 25.3)
Franklin	24.2	12.8 (3.7, 22.1)	39.2	17.1 (4.5, 29.8)
Charleston	30.8	19.4 (8.5, 30.5)	34.4	12.3 (−2.3, 27.0)
Georgetown	34.4	23.0 (13.7, 32.4)	34.0	11.9 (0.2, 23.7)

striking in the comparison of Georgetown and Seattle for hospital days, where a twofold unstandardized difference (42 compared with 21 percent; not shown) in inappropriate days was reduced by half (34 compared with 22 percent). Thus, although addition of patient, physician, and hospital variables decreased the extent of variation, significant geographic differences in the proportion of inappropriate hospital use remain even after controlling for these sources of variation with available variables.

Discussion

The sites in this study differed in patient characteristics and in provider types and availability, and we found that some of these characteristics were associated with inappropriate use. Nevertheless, we found that site differences in the proportion of inappropriate hospital use remain, even after controlling for most measurable aspects of patient, provider, and hospital characteristics. This finding confirms, for inappropriate hospital use, Wennberg's observation that practice patterns differ by geographic region and cannot be fully explained by geographic differences in patient or provider characteristics.[16]

The magnitude of these site differences was reduced by controlling for these patient and provider variables, some of which were strongly associated with inappropriate use. For both hospital admissions and days, we found that older physicians and female patients were more likely to be associated with inappropriate use. Although certain physician specialties were less likely to result in an inappropriate admission, these specialty differences in inappropriate use were most likely related to the performance of procedures. Many patients admitted by surgeons and gynecologists underwent a procedure within the first day of admission, leading the hospitalization to have been more likely designated appropriate for acute level care.

We cannot determine whether the effect of years from licensing is related either to declining clinical skills or to a cohort effect. Similarly, we are unable to explain the gender differences in inappropriate hospitalization. Gender effects have been observed in most studies attempting to explain overall patterns of medical care utilization.[17] The differences observed in our study could not have been due to child bearing, because pregnancy and related admissions were excluded from this analysis. This leaves a number of possible explanations, including differences in (a) how physicians perceive women's health needs, (b) the level of support available to women at home, and (c) the types of nonobstetric medical problems encountered by women.

Our results reaffirm the importance and policy significance of geographic variations in medical practice. The geographic variations observed in this study could not be attributed solely to patient and provider characteristics, many of which are difficult to modify. Future research should focus on the identification of modifiable variables and methods to reduce inappropriate hospital use.

Current cost containment policies are not likely to selectively reduce inappropriate hospital use,[2] and other approaches to do so are necessary. Retrospective review with feedback of results to physicians[18] may be one such approach. Conducting retrospective audits and making these results available to major buyers (e.g., major employers) is another approach to this problem. While it would be more efficient to target such reviews on those hospitalizations most likely to be inappropriate, our results suggest that targeting to specific physicians or hospitals on the basis of various characteristics would be very difficult. To reduce inappropriate hospital use by mechanisms involving targeted utilization review, other approaches to targeting will need to be developed.

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APPENDIX
Sample Characteristics

Variable	Mean (standard deviation) or percent
Patient Characteristics	
Age (years)	42.2 (12.2)
% Female	60
% Black	15
% receiving AFDC	5
General health index	62.9 (16.7)
Mental health index	72.8 (14.6)
% with role or physical limitation	36
% on cost sharing plan	39
% on individual deductible plan	22
Log (family size)	1.05 (0.55)
Log (family income)	8.77 (0.70)
% with <12 years of education	35
% with 12 years of education	39
% with >12 years of education	26
Physician Characteristics	
% solo practitioner	45
% board-certified	66
% in internal medicine	21
% in surgery	43
% in obstetrics/gynecology	13
% in general or family practice	18
% in licensed for >15 years	51
Hospital Characteristics	
% JCAH approved	89
% belonging to Council of Teaching Hospitals or having an AMA approved residency	26
% public hospitals	9
Log (bedsize)	5.41 (0.63)

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AIDS Conference Proceedings

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Public Health Briefs

Surveillance of Occupational Lung Disease: Comparison of Hospital Discharge Data to Physician Reporting

KENNETH D. ROSENMAN, MD, LINDA TRIMBATH, AND MARTHA STANBURY, MPH

Abstract: A survey of 762 New Jersey physicians showed that 35% reported seeing patients with either asbestosis, coal worker's pneumoconiosis, occupational asthma or silicosis. Three to four times as many patients with these diagnoses were seen as outpatients as were hospitalized. The implications of these results in using hospital discharge data for occupational disease surveillance are discussed. (*Am J Public Health* 1990; 80:1257–1258.)

Introduction

It has been proposed that hospital discharge data be used as part of an occupational disease surveillance system.[1,2] One of the limitations of hospital discharge data is the lack of information on non-hospitalized individuals. We conducted a survey of physicians in New Jersey to obtain estimates of how many patients they saw in their practice with asbestosis, coal workers' pneumoconiosis, occupational asthma, or silicosis in comparison to the number of patients they admitted to the hospital with the same diagnosis.

Methods

The names and addresses of physicians were obtained either from the state hospital discharge system or directly from the hospitals under state occupational disease reporting regulations (New Jersey Administration Code 8:57–1.13). Names and addresses of physicians were obtained by linking medical license numbers in the hospital discharge record to a data set of all licensed physicians in the state. The physicians surveyed had discharged at least one patient for the year 1985, 1986 or 1987 from one of New Jersey's 105 acute care (non-federal) hospitals with the diagnosis of asbestosis (ICD 501), coal workers' pneumoconiosis (ICD 500), pneumoconiosis due to other inorganic dust (ICD 503), pneumonopathy due to inhalation of other dust (ICD 504), pneumoconiosis unspecified (ICD 505), silicosis (ICD 502).

In the spring of 1988, a letter was mailed to these physicians requesting information about their medical specialty, an estimate of the total number of their patients newly diagnosed with asbestosis, coal workers' pneumoconiosis, occupational asthma, and silicosis in the calendar year 1987, and an estimate of the proportion of these patients admitted to the hospital during that same year. Non-respondents to a second and third mailing were contacted by telephone.

Results

A total of 847 physicians were included in the survey, of whom 762 (90.0 percent) completed the questionnaire. The reasons for non-response were: 29 (3.4 percent) letters were undeliverable; 20 (2.4 percent) refused to participate when telephoned; 11 (1.3 percent) were unreachable despite five telephone calls; nine (1.1 percent) had unlisted telephone numbers; six (.7 percent) retired; five (.6 percent) moved out of state; five (.6 percent) were deceased.

Table 1 shows the number of respondents, by medical specialty, who saw patients in 1987 with any of the lung conditions under consideration. The percentage of physicians reporting patients in 1987 is less than 100 percent, because many physicians may have had no patients with these diagnoses in 1987 or because they did not report all such patients.

Table 2 shows the proportion of patients hospitalized: for asbestosis 34 percent (95% CI = 31, 37); for coal workers' pneumoconiosis 26 percent (95% CI = 18, 35); for occupational asthma 23 percent (95% CI = 19, 27); for silicosis 27 percent (95% CI = 20, 35) were hospitalized. For all diseases combined, 548 (30 percent) of 1,814 patients (95% CI = 28, 32) were hospitalized.

Discussion

For the four diseases surveyed, physicians stated that for every patient hospitalized with the condition they had two to three additional patients with the same diagnosis who had not been hospitalized. This selected group of physicians reported nearly as many newly diagnosed cases of pneumoconiosis in New Jersey as are reported annually in the supplementary data system of the Bureau of Labor Statistics.[4] The number of additional patients not hospitalized varied both by disease as well as by the specialty of the reporting physician. The variation of hospitalization by specialty may be a reflection of the severity of disease seen by different physicians. For example, surgeons reported that 79 percent of their patients with asbestosis were hospitalized. The variation, however, may also reflect the relative amount of time physicians see hospitalized versus ambulatory patients.

None of the physicians reported their specialty as occupational medicine. Hospitalizations were not limited to a few specialty groups such as pulmonary physicians. Percentages of physicians reporting patients with lung conditions in 1987 by specialty varied from 8–79 percent although all physicians had admitted patients with these lung conditions

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TABLE 1—Total Number of Physicians Responding and Percent by Specialty who Reported Patients for the year 1987 with Either Asbestosis, Coal Workers' Pneumoconiosis, Occupational Asthma, or Silicosis

Medical Specialty	Number Physicians Responding	Percent Physicians Reporting Patients with Any of the Lung Conditions	Percent Physicians Reporting Asbestosis	Percent Physicians Reporting Silicosis	Percent Physicians Reporting Coal Workers' Pneumoconiosis	Percent Physicians Reporting Occupational Asthma
Internal Medicine	205	43.9	33.2	8.8	6.3	18.5
Pulmonary	65	78.5	70.8	29.2	30.8	61.5
Family/General Medicine	106	41.5	30.2	7.5	9.4	20.9
Surgery	110	17.3	16.4	3.6	0.9	2.7
Cardiology	80	35.0	31.3	3.8	6.3	10.0
Urology	49	8.2	8.2	0.0	0.0	0.0
All Other Specialties	126	18.3	14.3	1.6	4.0	16.7
No Specialty	21	19.0	14.3	4.8	0.0	4.8
Total	762	34.5	28.1	7.2	7.1	17.6

TABLE 2—Numbers of Reported Patients and Percent Hospitalized in 1987 with each Occupational Lung Disease by Physician Specialty

	Asbestosis		Silicosis		CWP		Occup Asthma		All Lung Diseases	
Physician Specialty	No. pts	% hosp	No. pts	% hosp	No. pts	% hosp	No. pts	% hosp	No. pts	% hosp
Internal Medicine	162	45	33	46	27	37	138	24	360	36
Pulmonary	592	17	66	26	46	15	188	20	892	19
Fam/Gen Medicine	78	32	15	7	11	36	61	13	165	23
Surgery	77	79	15	0	1	0	16	19	109	59
Cardiology	140	61	16	4	12	25	21	62	189	57
Urology	5	20	0	—	0	—	0	—	5	20
All other specialties	53	55	4	25	6	50	21	29	84	46
No specialty given	8	25	1	0	0	—	1	0	10	20
Total	1115	34	150	27	103	26	446	23	1814	30

in 1985, 1986 or 1987. Efforts are underway to follow up this survey by encouraging the physicians to report the names of patients they estimated on the questionnaire. Similar surveys in other states will be useful to examine if there are differences in care patterns in different states.

The physicians response rate to the questionnaire was excellent. We attribute this to its briefness and to our only asking for estimates. This is also its major limitation as we did not evaluate the accuracy of the physicians estimates. According to statewide hospital discharge data for 1986, there were 881 patients discharged with asbestosis, 97 patients with silicosis, and 235 patients with coal workers' pneumoconiosis. For the first two diagnoses, the hospital discharges were less than those reported by the physicians; for coal workers' pneumoconiosis, the hospital discharges were more than those reported by physicians. No data on occupational asthma is available from discharge data.

If the percentages of underreporting of 67 to 75 percent for different diseases found in this survey is generalizable and reassessed by periodic surveys of physicians then hospital discharge data because of its relative ease of collection and low cost would be a useful method to examine trends and estimate the number of occupational pneumoconioses.

Recently, the National Institute for Occupational Safety and Health (NIOSH) has been promoting the concept of sentinel provider-based occupational disease surveillance systems.[3] This survey shows that hospital discharge data can be used directly or indirectly to identify names, addresses and medical specialties of potential sentinel providers, and that, with sufficient follow-up, these physicians are willing to report to a state health department information that can be relied on for occupational disease surveillance.

ACKNOWLEDGMENTS

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Food-borne Outbreak of *Giardia Lamblia*

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Abstract: An outbreak of giardiasis occurred following a family party for 25 persons. Nine who had eaten fruit salad became ill, compared with one who had not eaten the salad (Relative Risk = 7.4, 95% CI = 1.4, 169.3). The fruit salad preparer had a diapered child and a pet rabbit at home who were both positive for *Giardia lamblia*. This outbreak emphasizes the importance of good hygienic practices in food preparation and the possibility of domestic-animal-to-person transmission in *Giardia* outbreaks. (*Am J Public Health* 1990; 80:1259–1260.)

Introduction

Giardia lamblia infection is often acquired by drinking contaminated water[1,2] or by person-to-person transmission among preschool children.[3] Transmission by food was first reported in 1981 when an outbreak of giardiasis was caused by eating contaminated home-prepared salmon.[4] We report an outbreak of giardiasis which most likely occurred due to the contamination of a fruit salad with *Giardia* cysts. The investigation also raises the question of whether domestic animals can play a part in transmission.

Background

On March 30, 1986, a party for 25 persons from seven separate family groups was held at a private home in southern New Jersey. Within seven days of the party, several attendees developed giardiasis. The outbreak was identified by the county health department from seven stool specimens received from ill attendees which were positive for *G. lamblia*.

Methods

All party attendees were contacted by telephone, and a questionnaire was administered. Guests were asked whether they had had a diarrheal illness, which of 30 possible food and drink items they had consumed at the party, and whether they had been in contact with day care centers or institutions for the handicapped. To determine if there was a widespread outbreak of giardiasis in the area, four local hospitals were contacted.

A case of giardiasis was defined as a person who had diarrhea (>3 stools/day) for >5 days with or without fever, *or* intermittent diarrhea >5 days (with or without fever) with two or more of the following symptoms: flatulence, nausea, malaise, or abdominal cramps.

Stool specimens were collected from all party attendees and from the children and staff at the local babysitting service which was attended by a diapered child who had been at the party. Specimens were examined for *Giardia* microscopically and enteric bacterial cultures were performed for *Salmonella*, *Shigella*, and *Campylobacter* on all persons with clinical symptoms. A stool specimen was also obtained from a pet rabbit in the home of the diapered child and examined for *Giardia* cysts.

The environmental investigation included an account of the preparation of the food and an investigation of the household water supplies using filters.

Results

Epidemiology

Ten cases of outbreak-associated giardiasis were identified. Nine (eight stool positive) of the cases had onset of symptoms during the period six to 12 days following the party and one case (stool negative) occurred 18 days after the party. Three community giardiasis cases were reported from the local hospitals for the previous five months.

Attack rates by gender and age groups were similar; each household had at least one member who was a case (Table 1). The attack rate for 20 persons who had eaten some food at the gathering was 50 percent compared to zero for those not eating. The five persons who had not eaten any food were excluded from further consumption analysis. Among those eating food at the party, those eating fruit salad were seven times more likely to have a case of illness than those who did not eat the salad (82 percent, 9/11 vs 11 percent, 1/9, RR = 7.4, 95% CI = 1.4, 169.3) (Table 2); nine of the 10 case-patients and all of the stool-positive cases had eaten fruit salad.

Laboratory

Stool specimens from eight of the 10 case-patients were positive for *G. lamblia;* all were negative for enteric bacterial pathogens. One of the two cases with negative studies for *Giardia* had been on metronidazole for 10 days.

Environmental

The fruit salad was prepared in the home of a woman who became ill nine days after the party. She had a two-year-old diapered child who, with four other children, was cared for by a babysitter during the day. Her child, who had eaten the fruit salad, had had no symptoms of gastroenteritis but had a positive stool for *Giardia* on the first specimen. The

TABLE 1—Family Attack Rates among Party Attendees, *Giardia* Outbreak, New Jersey, 1986

Family	Number Persons	Number Cases	Number Stool Positive
A	4	2	2
B	2	2	2
C	4	1	2
D	5	1	0
E	5	1	1
F	2	2	2
G	3	1	0
Total	25	10	9

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TABLE 2—Food-Specific Attack Rates (AR), *Giardia* Outbreak, New Jersey, 1986

	Ate Food	Did Not Eat Food	
Food Item*	Case/Total AR (%)	Case/Total AR (%)	RR (95% CI)
Cold Foods			
Ham	7/13 (54)	3/7 (43)	1.3 (0.5,5.2)
Corned Beef	6/9 (67)	4/11 (37)	1.8 (0.6,4.7)
Deviled Eggs	8/15 (53)	2/5 (40)	1.3 (0.5,9.7)
Potato Salad	6/8 (75)	4/12 (33)	2.3 (0.8,4.8)
Coleslaw	7/11 (64)	3/9 (33)	1.9 (0.6,7.0)
Bread	6/8 (75)	4/12 (33)	2.3 (0.8,4.8)
Rolls	7/13 (54)	3/7 (43)	1.3 (0.5,5.2)
Drinks			
Sodas	7/12 (58)	3/8 (38)	1.5 (0.5,6.2)
Deserts			
Fruit Salad	9/11 (82)	1/9 (11)	7.4 (1.4,169.3)

*Only foods eaten by >50% of the case-patients are reported.

four other children attending the babysitter's home and the babysitter's family were negative for *Giardia*.

Two days before the party, this diapered child had been given a pet rabbit as a present. The animal had been present in the kitchen when the fruit salad was being prepared. The rabbit was positive for *Giardia* (unspecified).

The salad was made two to three hours before being served. The fruit was either peeled or cut on a lucite chopping board and then placed in a plastic bowl with a lid.

No *Giardia* cysts were found in the filters from the homes.

Discussion

This outbreak of giardiasis represents a point-source outbreak with the fruit salad as the most likely vehicle of transmission. The high attack rate for those persons who ate the salad (82 percent, 9/11) implies that *Giardia* was effectively transmitted by this food.

The outbreak had some similarities with the salmon outbreak previously reported.[4] Both had high attack rates for persons who ate the contaminated food—48 percent for salmon and 82 percent for fruit salad—and, in both outbreaks, food was probably prepared without prior hand washing. Diapered infected children were present in both homes where the contaminated food was prepared.

There are two likely hypotheses for transmission of *Giardia* and both postulate that the fruit salad was contaminated by the food preparer who had failed to wash her hands before food preparation. The first hypothesis is that changing of the infected child's diaper would have been an effective way of transmitting cysts to a persons' hands and then to the fruit salad. Fruit salad contains a liquid acid medium which would maintain the viability of *Giardia* cysts which are known to be resistant to acid.[5] It is also known that cysts are rapidly destroyed when dried, but remain viable for weeks under moist conditions.[6]

The second hypothesis for transmission is that the rabbit was positive for *Giardia* when it entered the home and was excreting cysts. These cysts were transferred to the hands of the food preparer who contaminated the fruit salad by failing to wash her hands. There is one report of an outbreak of giardiasis in which transmission occurred between animals and humans in a zoo.[6] In addition, there has been a report that suggests that cats may be responsible for introducing giardiasis into households[7] but little is known about the importance of animals as reservoirs of the human *Giardia*[6] or the susceptibility of humans to animal *Giardia* (other than *G. lamblia*). Traditionally it has been believed that *Giardia* is highly host-specific.[8]

We recommend that when health department personnel investigate *Giardia* outbreaks, they should consider the possibility of food-borne transmission and when giardiasis occurs in a household that contains domestic animals, stool specimens should be collected from the animals for cyst microscopy. If possible, the human and animal species of *Giardia* should be determined. In this way, more information will be collected to determine if *Giardia* cysts are transmitted between domestic animals and humans.

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Leukemia Incidence and Radioactivity in Drinking Water in 59 Iowa Towns

LAURENCE FUORTES, MD, LOUISE ANN MCNUTT, MS, AND CHARLES LYNCH, MD, PHD

Abstract: Fifty-nine towns in Iowa with single source drinking water supplies were stratified on the basis of radium content in finished non-softened water to test the hypothesis of an association with total or acute myeloid leukemia. Fourteen towns had radium concentrations in drinking water exceeding the EPA safety limit of 5 picocuries per liter (pCi/L). A small increasing trend existed for total leukemia with increased radium content in drinking water that is in accordance with either the hypothesis of no effect or of a small effect. (*Am J Public Health* 1990; 80:1261–1262.)

Introduction

Several studies have documented increased cancer incidence or mortality associated with radioactivity of drinking water. The US Environmental Protection Agency (EPA) has set a safety limit of 5 picocuries per liter (pCi/L) for total radium in public water supplies. There are a number of communities nationwide whose water supplies exceed this limit. In a study reported in 1966 on low level radium 226 exposure, death rates from bone cancer were found to be higher in municipalities exposed to higher levels of radium 226 in drinking water. The numbers of cases were small, however.[1]

Ingested radium (Ra^{226} and Ra^{228}), an alpha particle emitter, is a well documented cause of bone and nasal cancers in studies of radium dial painters and follow-up of patients treated with Ra^{224}. In the radium dial painters, the risk of developing bone cancer was impressive in magnitude, (RR = 50), and specific to bone.[2] In the studies of human subjects treated with Ra^{224} (a shorter lived isotope), the risk of both bone cancers and leukemias was increased.[3]

A recent study from Florida revealed an association of acute myelogenous and total leukemia incidence rates with radium content in private wells. In that study, 27 counties were chosen based on their proximity to deposits of phosphate ore.[4] Based on a survey of 50 private wells in each county, 10 counties had high exposure and 17 had low exposure. Counties with greater than 5 pCi/L of radium in 10 percent or more of the sampled wells were included in the high exposure group. The age-standardized incidence density ratio for high exposure vs low exposure was 1.49 for total leukemia and 2.00 for acute myelogenous leukemia.

In a previous Iowa study, associations of radioactivity in drinking water were reported with lung, breast, and bladder cancer.[5] The Iowa data were reexamined in an attempt to determine if radioactivity in drinking water was associated with leukemia incidence.

Methods

Fifty-nine Iowa towns, each of less than 10,000 population, with a single source water supply and without water softening in their treatment process were identified from an Iowa Geologic Survey Bureau data base. The towns were stratified into three groups according to radium^{-226} content in finished water: *high* = Ra^{-226} greater than 5 pCi/L, *medium* = Ra^{-226} between 2–5 pCi/L, and *low* = Ra^{-226} less than 2 pCi/L. For each of the towns studied, there were greater than 2 radium measurements in finished water available. By this schema there are 812,616 persons years in the high, 2,055,060 in the middle, and 1,562,652 person years in the low exposure categories.

Identification of leukemia cases was performed by the State Health Registry of Iowa. Cancer incidence data were utilized for the years 1969–84, excluding 1972. There was no statewide cancer surveillance in 1972. Total and myelogenous leukemia rates are reported.

Age-adjusted, sex-specific incidence rates and 95 percent confidence intervals were calculated for each of the three groups using direct standardization as described by Fleiss.[6] The standard population utilized was the 1970 US population. Statistical testing for linear trend in incidence rates across the three groups was performed using a poisson trend statistic.[7]

Results

The age-adjusted incidence rates for total leukemia and acute granulocytic leukemia are shown in Tables 1 and 2. There was a weak positive relation between total leukemia rates and radioactivity in water, but a weak negative relation between acute granulocytic leukemia rates and radioactivity level consistent with either the hypothesis of no effect or of a small effect. As shown by the wide confidence intervals, the estimates of radioactivity-specific rates were rather unstable.

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TABLE 1—Age-adjusted Incidence of All Leukemias per/100,000 Person Years for 59 Iowa Towns (all ages), 1969–84

	Male		Female		Male & Female		Total Cases	
	Rate	(95% CI)	Rate	(95% CI)	Rate	(95% CI)	Male	Female
Group 1 (N=14)	17.2	(13.4, 21.0)	8.6	(6.3, 11.0)	12.0	(7.5, 16.5)	82	65
Group 2 (N=18)	16.3	(13.7, 18.8)	8.9	(7.2, 10.6)	11.9	(8.7, 14.9)	158	120
Group 3 (N=27)	16.1	(13.6, 18.7)	7.4	(5.8, 8.9)	10.9	(7.9, 13.9)	164	111

Group 1 = High, RA-226, > SPCi/L, 14 towns with an average population = 4,145 for a total of 812,616 person years.
Group 2 = Medium, 2.0 < RA-226 < 5.0 PCi/L, 18 towns with an average population = 8,155 for a total of 2,055,060 person years.
Group 3 = Low, RA-226 < 2.0 PCi/L, 27 towns with an average population = 4,154 for a total of 1,562,652 person years.

TABLE 2—Incidence of Acute Myeloid Leukemia per/100,000 Population for 59 Iowa Towns (all ages) 1969–84, excluding 1972

	Male		Female		Male & Female		Total Cases	
	Direct Rate	(95% CI)	Direct Rate	(95% CI)	Direct Rate	(95% CI)	Male	Female
Group 1 (N=14)	2.5	(1.1, 4.0)	1.2	(0.2, 2.1)	1.6	(0.5, 3.4)	12	8
Group 2 (N=18)	2.6	(1.6, 3.7)	1.3	(0.7, 2.0)	1.8	(0.6, 3.1)	24	17
Group 3 (N=27)	2.5	(1.5, 3.4)	1.8	(0.9, 2.6)	2.0	(0.7, 3.3)	25	22

Group 1 = High, RA-226, > SPCi/L, 14 towns with an average population = 4,145 for a total of 812,616 person years.
Group 2 = Medium, 2.0 < RA-226 < 5.0 PCi/L, 18 towns with an average population = 8,155 for a total of 2,055,060 person years.
Group 3 = Low, RA-226 < 2.0 PCi/L, 27 towns with an average population = 4,154 for a total of 1,562,652 person years.

Discussion

These data do not support the strong relation between radioactivity in drinking water and leukemia incidence suggested by the Florida study.

Published studies on the association of radium in water with cancer are aggregate studies and subject to the biases associated with such designs. The results of the Iowa and Florida studies may to some extent be explained by misclassification and confounding.

In the case of water studies, a more accurate measure of exposure would be based on the amount of radium^{-226} water consumed by each individual and the examination and adjustment for confounding etiologic factors. The amount of radium^{-226} in drinking water in these communities is subject to a variety of factors including home water softening, varying tap water consumption, duration of residence in the community, and so forth.

In the Florida study, a county was defined as high exposure on the basis of 10 percent of the sampled wells exceeding the EPA limit for radium. The Iowa study has the benefit of accurately defined exposure categories in a demographically stable area. Despite similar study designs, this study does not yield the strong association previously reported with leukemia. Future study of cancer incidence and radium concentration in drinking water in these and other communities is needed to clarify the risk of leukemia secondary to naturally occurring Ra^{-226}.

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Survey of Occupational Exposure of Waste Industry Workers to Infectious Waste in Washington State

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Abstract: We surveyed 940 Washington state waste industry workers to evaluate occupational exposure to potentially infectious materials (response rate 47 percent). Only 26 percent were trained specifically to deal with safety hazards associated with medical waste. For the year preceding the survey, 50 percent of respondents reported having received cuts and scratches on the job, 22 percent reported direct contact with waste blood on their clothing or shoes, 8 percent had blood exposure on their skin, 3 percent blood exposure on their face or eyes, and 6 percent occupational hypodermic needlestick injuries (10 percent among waste collectors). (*Am J Public Health* 1990; 80:1262–1264.)

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Introduction

Medical waste disposal has recently emerged as a concern for waste industry workers. The AIDS (acquired immunodeficiency syndrome) epidemic has elevated waste worker concern regarding the potential for disease transmission from medical waste stream sources. Such transmission of AIDS or hepatitis B has never been epidemiologically demonstrated.[1]

Few studies have been conducted to examine waste industry worker exposure to potentially infectious agents in the waste stream. Gellin and Mitchell examined 97 waste workers employed by the City of Cincinnati during January and February of 1968 for skin disorders.[2] Forty-one cases of bacterial, viral, or fungal dermatitis were observed in this group, but all were classified as non-occupational in origin.

Cimino examined New York City Sanitation Department health records of waste workers employed between 1968–69.[3] Needlestick injuries were reported due to the presence of uncontained needles in waste collected from hospitals, physician's and dentist's offices, and discarded needles from drug addicts. All workers reporting needlestick injuries were given

gamma globulin prophylaxis and no cases of hepatitis were reported.

A 1979 report by Clark, *et al*,[4] examined the incidence of viral infection among 43 waste collection workers. Sera antibody levels for 18 viruses were examined from blood samples collected during the spring and fall. The authors found no evidence of increased occupational risk to blood-borne viral infections.

This study was conducted to examine occupational exposure of Washington state waste industry workers to blood-contaminated waste and discarded hypodermic needles in the municipal waste stream.

Methods

The survey was conducted during February and March of 1989. Questionnaire packets were prepared and delivered to seven private sector waste industry firms and six public sector waste collection/disposal agencies. Selection focused on the state's population centers, although rural participation was included. Industry names were obtained through the Washington Utilities and Transportation Commission records and public sector solid waste utility listings. A total of 940 questionnaires were distributed to waste workers directly involved with waste collection or disposal within these firms or agencies. Based on the number of waste workers identified in the 1980 census which was adjusted for population growth and a review of non-participating firms, the surveyed workers account for approximately 70 percent of the waste industry workforce in Washington state.

Participant responses were confidential and not individually identified. Questionnaires were returned directly to the research group by the participants in stamped return envelopes without review by their respective waste industry employers.

Participants included those involved with residential waste collection, commercial collection, and landfill/transfer station operations. Waste worker job categories included heavy equipment operators, truck drivers, collectors, equipment maintenance personnel, laborers, recyclers/sorters, gatekeepers/scalehouse operators, and field supervisors.

Survey variables included the respondent's job category, job safety training, occupational observance of medical waste, exposure to blood-contaminated waste, and occurrence of hypodermic needlestick injuries. Questions regarding occupational exposures or injuries requested that the respondent include the month and year of his/her most recent event to discourage a positive response unless a specific occurrence could be recalled.

Results

Of 940 surveys distributed to waste workers, 438 were returned (47 percent). Of these, 52 percent of respondents conducted all or part of their job activities in residential waste collection, 41 percent in commercial waste collection, and 38 percent in landfill/transfer station operations.

Sixty-nine percent of the respondents reported receiving job safety training. Twenty-six percent indicated that this training included medical waste safety practices. Ninety percent of the respondents reported observing medical waste (e.g. needles, blood, bandages) in the waste stream. Of this group, the frequency of observation of medical waste was identified as daily by 27 percent of this group, weekly by 40 percent, monthly by 17 percent, yearly by 8 percent and less than once a year by 8 percent.

Injury/exposure information for occupational cuts and scratches, blood exposures, and needlestick injuries is based on the respondent's recall for events occurring on at least one occasion. Such information reported for the year preceding the survey required the respondent to include both the month and year of their most recent event.

Cuts and scratches received on the job were reported by 74 percent of the respondents. For the year preceding the survey, such injuries were reported by 50 percent of respondents.

Thirty-two percent of the respondents reported having experienced direct contact with blood on their clothing or shoes, 13 percent on their skin, and 5 percent on their face or eyes. For the year preceding the survey, 22 percent reported having experienced direct contact with waste blood on their clothing or shoes, 8 percent had blood exposure on their skin, and 3 percent had blood exposure on their face or eyes.

Twenty-one percent of responding waste workers reported having been stuck or scratched by a waste hypodermic needle. Six percent reported that their most recent injury occurred within the year preceding the survey. Such injuries were reported by 10 percent of waste collectors within the same time period. Needlestick injuries were reported by 24 percent of respondents for residential waste collection, 21 percent for commercial waste collection, and 11 percent for landfill/transfer station operations. Needlestick events by individual job category are presented in Table 1.

Discussion

In this survey, the low response rate (47 percent) may have resulted in a higher estimated incidence of exposure; exposed individuals should be more likely to respond if an exposure is a memorable experience. If one assumes that none of the non-responders had experienced an exposure and only respondents who could give both the month and year of the event actually experienced an exposure, the estimated exposure incidence among the 940 persons sent a questionnaire for the year preceding the survey would be 10 percent (blood exposure to clothing or shoes), 4 percent (blood exposure to skin), 1.5 percent (blood exposure to face or eyes), and 3 percent (needlestick injuries).

Of greatest potential significance to waste worker health was the reporting of needlestick injuries. Written survey comments from workers described loose needles originating from uncontained disposal or ruptured sharp containers in the waste stream. Needles were identified originating from both residential and medical facility sources. Accounts of uncontained

TABLE 1—Needlestick Injury by Waste Worker Job Category for the Year Preceding the Survey

Primary Job Category	Number (%) of Respondents	Number and Proportion (%) of Respondents Reporting Needlestick Injury
Heavy Equipment Operator	27 (6)	1 (4)
Truck Driver	79 (18)	2 (3)
Collector	241 (55)	23 (10)
Equipment Maintenance	19 (4)	1 (5)
Laborer	23 (5)	1 (4)
Recycler/Sorter	2 (0.5)	0 (0)
Gatekeeper	23 (5)	0 (0)
Field Supervisor	18 (4)	0 (0)
Total	432	28 (6)

*Needlestick response requires respondent noting month and year of occurrence.

needles and the reporting of needlestick injuries sustained by waste workers indicate the need for proper handling, containment and/or destruction of needles by users prior to disposal.

Observation of medical waste (e.g. needles, blood, bandages) in the general waste stream was common (90 percent of respondents). Further study to identify frequency of occupational exposure to blood is recommended, especially in relation to proposed standards for bloodborne pathogens by the US Department of Labor, Occupational Safety and Health Administration.[5]

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Fear of AIDS and Attrition among Medical Technologists

RONALD R. GAUCH, PHD, KAREN B. FEENEY, MPA, AND JAMES W. BROWN, PHD

Abstract: Attitudes toward AIDS were measured by a survey of 212 attendees at the annual meeting of the New Jersey Society for Medical Technology. Twenty five percent of the respondents were considering leaving the profession because of a fear of AIDS. In addition, almost half would not have chosen the field knowing they would be handling HIV-positive samples. This high degree of concern may be an important factor contributing to the shortage of medical technologists. (*Am J Public Health* 1990; 80:1264–1265.)

Introduction

A 1988 survey by the American Society of Clinical Pathologists reported that 9 percent of medical technologists positions are vacant and more than 75 percent of laboratory managers perceive a shortage of qualified laboratory personnel.[1] The shortage among medical technologists has been attributed to a wide spectrum of problems: low pay with a compressed salary structure, lack of upward mobility, job stress, poor working conditions, lack of professional recognition, and increased opportunities in other professions that have commensurate educational requirements.[2,3]

A survey conducted in 1987 among New England laboratories, reported that 39 percent of the respondents mentioned fear of AIDS (acquired immunodeficiency syndrome) as a reason laboratory workers leave the profession.[4] A 1987 study, confined to medical technologists, also found a high fear of acquiring AIDS.[5] The purpose of our study was to describe attitudes of medical technologists toward working with AIDS-infected samples.

Methods

A questionnaire was designed which included 15 questions using Likert rating scales with the response options ranging from strongly agree to strongly disagree. Independent variables included demographic and institutional factors. Three additional questions dealt with an individual's knowledge of AIDS.

The questionnaire was distributed to registrants at the 1988 annual meeting of the New Jersey Society for Medical Technology. Of the 295 registrants, 283 were given forms to be filled out anonymously and 212 were returned (75 percent).

Results

The results showed that fear of AIDS among laboratory workers is common (Table 1). For example, 52 percent agreed with the statement that, despite precautions, it was likely that they could become infected with HIV because of laboratory exposure and 41 percent would like to transfer to a position that required less blood handling. Only 43 percent indicated that they would have chosen the profession knowing they would be handling samples from AIDS patients and one out of four respondents is considering leaving the profession because of a fear of AIDS.

Medical technologists are clearly experiencing pressure to seek a different career. In 86 percent of those queried, respondents reported that friends and family members expressed concern about their working with HIV samples and 53 percent knew of colleagues who had left the profession because of the possibility that they could acquire the human immunodeficiency virus (HIV).

On the other hand, there was general satisfaction with employer safety policies. Three out of four felt their employer provided adequate education concerning HIV risk and that adequate safety measures, which lower the risk of HIV transmission, were available. However, less than half felt that their employer offered adequate counseling in the event of an accident.

It was apparent that the AIDS problem had resulted in improved safety practices within the profession. For example, increased reporting of needlestick injuries and the wearing of gloves were reported by 87 percent and 94 percent of the respondents, respectively.

Not surprisingly, those currently working were more inclined to contemplate a career change than people about to enter the field. One in three of employed medical technologists was thinking about departing the profession because of their fear of AIDS. In contrast, only 13 percent of the students surveyed indicated they were planning to take such a step.

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TABLE 1—Percent of Respondents in Agreement with Survey Questions

Questions	Percent Agreement*
Fear of Acquiring AIDS Questions	
I feel I have the right to be informed of HIV + samples.	96
I believe that fear of acquiring HIV is a major reason for the low enrollments in the MLT and MT schools.	77
Despite precautions, I believe it is likely I could become infected with HIV because of laboratory exposure.	52
In retrospect, I would have chosen the field of medical technology knowing that I would be handling samples from AIDS patients.	43
I would like to remain in the field but would like to transfer to a position that requires less blood handling because of my fear of acquiring AIDS.	41
I am considering leaving the profession because of my fear (and/or my "significant other's" fear) of acquiring HIV in the laboratory.	25
Pressure to Leave the Profession Questions	
I have had friends or family express concern about my working with HIV positive samples.	86
I know of colleagues who have left the clinical laboratory profession because of their fear (and/or their "significant other's" fear) of acquiring HIV in the laboratory.	53
Employer Safety Policies Questions	
I feel that infection control practices in my lab have improved because of fear of acquiring HIV.	79
I feel my employer provides adequate education on HIV risk.	77
I feel my employer has provided adequate safety measures that lower the risk of HIV transmission in the laboratory.	75
I feel my employer offers adequate counseling in the event of an accident with HIV positive samples.	41
Employee Safety Practices Questions	
Because of the risk of contracting AIDS I am more likely to wear gloves today than in the past.	94
Because of the risk of contracting AIDS, I am more likely to report needlestick injuries than in the past.	87
I feel that universal blood precautions will change the way I currently handle samples.	72

*Percent based on a response of strongly agree or agree.

Further analysis was carried out on the group working in the field to determine if there were any important differences between those considering and those not planning a career change. No important differences were present in respect to age, sex, salary level, marital status, responsibility for dependent children, education level, or working environment (hospital or nonhospital work). Furthermore, the two groups were similar in respect to their knowledge of AIDS (i.e. awareness of the transmission, etiology, and diagnosis of AIDS). The only demographic variable that distinguished the two groups was position in the organization. Of those with supervisory responsibilities, 18 percent were considering a job change versus 38 percent of those without supervisory responsibilities (data available upon request to authors).

Discussion

These findings suggest that fear of acquiring AIDS contributes to the attrition rate among medical technologists. On a positive note, it was found that medical technologists tend to be satisfied with employer AIDS policies. There was, nevertheless, concern on the part of a majority of workers about the lack of a counseling program in the event of a laboratory accident. Employers can help reduce this anxiety by having counseling programs in place and making sure workers know they exist.

The survey found that because of AIDS, glove use increased substantially. However, when asked how often they wear gloves, only 46 percent responded that they wore gloves all the time. It therefore appears that laboratory management needs to be more active in motivating their staffs to wear gloves on a regular basis. The Occupational Safety and Health Administration's mandate for routine glove use, which came out after the survey was completed, places an even greater responsibility on the part of management to see to it that employees use safe practices.

It is also important to note that universal blood precautions, where all samples are to be considered infectious, were in effect when the survey was taken. Still, the overwhelming majority of the respondents (96 percent) felt they had the right to be informed of HIV positive samples. This situation illustrates a common dilemma facing the health care community—a patient's right to privacy and an employee's "right to know."

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Are Women Using Postmenopausal Estrogens? A Community Survey

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Abstract: Self-reported estrogen and progestin use in a California community was determined in 1986–87 from a telephone survey of postmenopausal women (n = 954) ages 50–65 years. Current use of hormones was reported by 32 percent; 26 percent took estrogens alone while 6 percent used estrogen + progestin. Comparisons pointed to significant social network and medical care utilization differences. Women who used estrogen therapy were younger, thinner, lived in smaller household units, and were less likely to be widowed. (*Am J Public Health* 1990; 80:1266–1268.)

Introduction

Postmenopausal estrogen use has fluctuated in the past three decades, based on changing perceptions of its associated risks and benefits.[1–11] Currently, estrogens are advocated[12] because of their beneficial effect on serum lipids[13–16] and their protective effect against osteoporosis[10,11] and cardiovascular disease,[17–22] although the risk of cancer with prolonged use remains a concern.[3,8,9,23] Several studies have examined the effect of these changing perceptions on clinical practice.[24–26] Physicians in the mid-1980s report prescribing postmenopausal estrogen to far more of their patients than in the 1970s, although they are prescribing lower doses with an added progestin.[25,26]

We report postmenopausal hormone use in a defined community in 1986.

Methods

Between October 1986–April 1987, a random-digit dial telephone survey was completed of persons ages 50–65 years residing in Sunnyvale, California, a relatively affluent community located at the southern end of the San Francisco peninsula. Only one individual per household was interviewed, with decisions as to whom to interview made using a random allocation procedure. At least five calls were attempted to the household. The phone company estimates that over 97 percent of the households have telephones. The response rate among age-eligible women was over 72 percent.

Postmenopausal women were asked whether they were currently using any prescribed medications and to name the specific medications and dose by reading from the prescription labels. Hormone use was classified by major type of therapy: use of estrogens alone versus use of estrogens plus a progestin. Other questions, adapted from the 1985 National Health Interview Survey,[27] determined social and demographic characteristics, use of other medications and alcohol, exercise and smoking behavior, a brief medical history, and perceptions of current health status. Body mass index (kg/m^2) was calculated from self-reported height and weight.

This report is based on 954 postmenopausal women between ages 50–65 years. Only 45 women were excluded because of missing information on menopausal status or medication use.

Bivariate contingency tables were constructed to assess association between use of estrogens and the interview questions. Multivariate analyses used the logistic model to assess independence of the variables. Adjusted odds ratios and their 95% confidence intervals for estrogen use were calculated from the logistic coefficients and their standard errors[28] and to test for statistical significance of age interaction terms.[29] The dependent variable was coded as use or nonuse of estrogen hormones. Age and body mass index were modeled as continuous variables. Number of members in the household was an ordinal variable coded 0–6+; other independent variables were coded as presence or absence of the history or event.

Results

In this population, 32.2 percent (n = 307) of the women reported current use of postmenopausal estrogens. Of the 307 users, 18 percent (5.9 percent of the total population) reported also taking a progestin. Use was highest in the younger women, with 40.6 percent of women ages 50–54 reporting use compared to 23.1 percent of women ages 60–65 years. The use of progestins also decreased with age, from 9.4 percent of women ages 50–54 years to 2.8 percent of women 60–65 years.

Eighty-three percent of the hormone users identified a specific medication, or a dose if the estrogen was a generic conjugated estrogen. Premarin was the predominately named estrogen (65 percent), followed by the generic conjugated equine estrogens (27 percent). Forty percent of conjugated estrogen users named the .625 mg dosage. Provera accounted for 63 percent of progestin use, with 46 percent of these users naming the 10 mg dose.

Self-reported demographic and health behavior characteristics are shown in Table 1. Users were thinner, reported more regular aerobic exercise, had fewer people living in their households, and were less likely to be a widow than were the nonusers.

There were no differences between users and nonusers in their history of high cholesterol, diabetes, or heart problems (Table 2). Users were less likely to have a history of high blood pressure or stroke, but more frequently reported being under the care of a physician for other medical conditions.

Table 3 shows the results of the multivariate analyses. Age of the woman, body mass index, number of individuals in the household, and widowhood remained independent correlates of postmenopausal hormone use. Other logistic regression models showed history of high blood pressure, aerobic exercise, and presence of other medical conditions were not independently related to the hormone use. In addition, no significant age interactions were demonstrated. Similar analyses were performed to compare estrogen-only use with estrogen + progestin use; age was the only variable independently related to estrogen + progestin use.

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TABLE 1—Descriptive Information on Sociodemographic and Health Behavior Variables for Users and Nonusers of Postmenopausal Hormones in the Sunnyvale Population, 1986

Characteristics	Postmenopausal Hormone	
	Users n = 307*	Nonusers n = 647*
Marital status		
% Married	59.6	59.8
% Widowed	9.1	14.9
% Currently employed	61.2 (304)	58.6 (642)
Education, mean ±sd	13.7 ± 2.6	13.6 ± 2.8
Ethnicity: % White	91.9	88.4
Household size, mean number ± sd	2.0 ± 0.8 (295)	2.4 ± 1.2 (628)
% Current smoker	24.6	26.7
% Aerobic exercisers	23.5 (302)	17.5 (642)
Body Mass Index, mean kg/m^2 ± sd	24.0 ± 3.9 (306)	25.4 ± 4.9 (631)
Alcohol use, mean drinks/2 weeks ± sd	10.4 ± 14.1 (240)	8.5 ± 10.9 (428)

*Total number of users and nonusers, if number different than given in parentheses.

TABLE 2—Self-reported Medical Conditions of Users and Nonusers of Postmenopausal Hormones

Characteristics	Postmenopausal Hormone			
	Users		Nonusers	
	%	N	%	N
History of high blood pressure	28.0	(304)	37.2	(645)
History of stroke	00.3	(307)	3.1	(646)
History of high cholesterol	21.8	(294)	20.6	(622)
History of diabetes	3.2	(285)	5.5	(599)
History of heart problems	6.2	(307)	6.2	(638)
Other medical conditions	23.7	(304)	18.1	(643)
Hospitalizations in past year, mean ± sd	0.2 ± 0.5	(298)	0.2 ± 0.6	(630)
Perceived health rating* mean ± sd	1.8 ± 0.7	(307)	1.9 ± 0.8	(646)

*Health rated as Excellent (1), Good (2), Fair (3), Poor (4).

TABLE 3—Adjusted Relative Odds for Postmenopausal Estrogen Use by Behavioral, Sociodemographic and Reported Medical Conditions

Characteristic	Adjusted* Odds Ratio	(95% CI)
Age trend per year	.92	(.89, .95)
Body mass index trend (kg/m^2)	.93	(.90, .97)
Household size trend (no./household)	.66	(.55, .78)
Widowed	.49	(.30, .82)

*Adjusted using a logistic regression model that included the variables listed on this table, n = 294 users and 612 nonusers.

Discussion

This 1986 community survey shows self-reported current use of PMH (32 percent) is less than expected when compared to recent physician surveys indicating 75–95 percent of gynecologists would prescribe estrogen therapy to most of their patients[25,26] and to estimates from pharmaceutical marketing surveys of 43 percent use among women living in the western part of the United States.[7] The use rate is similar to a recent report from another California community.[30] The use of progestin combined with the estrogen is also less than expected and less than previously reported,[30] with 18 percent of the users reporting a progestin component.

While response bias, i.e., women not knowing or remembering what medications they are currently taking, could have contributed to the low postmenopausal hormone use, it is unlikely to be the major explanation of these discrepancies. Over 80 percent of the estrogen users were able to name their specific medication and dose. Since hysterectomy status was not ascertained in this survey, it is impossible to know what percent of women with an intact uterus were using unopposed estrogens. However, this limitation would not have contributed to an underestimate of total use. A high hysterectomy rate in Sunnyvale could explain the lower use of progestins than seen in the Rancho Bernardo study.[30]

The taking of PMH by women is the outcome of several factors, notably: local physician prescribing practices, medical care utilization patterns, drug side-effects, and social network or information sources of the women. Differences between women who use and do not use estrogens can, therefore, be due to underlying social or behavioral differences as well as to actual drug effects.

As in other surveys,[31,32] the youngest women, who were most likely to be recently menopausal, had the highest current estrogen and progestin use. This pattern probably reflects the effort to control menopausal symptoms as well as to decrease bone density loss. Younger women reached menopause at a time physicians were increasing their postmenopausal hormone prescribing.

The findings of thinner women more likely to use hormones was also shown in the earlier surveys.[31,32] This difference may reflect increased menopausal symptoms in thinner women due to their decreased endogenous estrogen levels, or more prescribing by physicians because of increased risk of osteoporosis in thinner women.

Women who were widowed were less likely to be on estrogen therapy than women who were currently married, but women who used hormones had fewer people living in their households. These findings may reflect basic social network differences between users and nonusers: e.g. nonusers may be major caretakers of other family members and less likely to have the time or inclination to care for themselves; a recent study of persons over age 65 reported that people who live with others go to doctors less often.[33]

In summary, this survey shows a discrepancy between published physician enthusiasm for PMH and low reported use by women. Comparisons between users and nonusers reflect differences in physician prescribing patterns, social networks, and health care utilization patterns.

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Estimating the Mortality Cost of AIDS: Do Estimates of Earnings Differ?

CHARLES E. BEGLEY, PHD, MARTIN M. CRANE, PHD, AND GRADY PERDUE, PHD

Abstract: The future earnings of male Texans dying of AIDS in 1987 were estimated using: national earnings profiles; earnings derived from occupations listed on death certificates; and earnings reported by persons with AIDS who responded to a survey. Mortality cost estimates using the two sources of actual earnings differed by a modest amount in comparison to the estimate using national earnings profiles. (*Am J Public Health* 1990; 80:1268–1270.)

Introduction

Because of the relative youthfulness of persons with acquired immunodeficiency syndrome (AIDS), the cost to society from the loss of earnings of those who die prematurely is an important concern in measuring the impact of the disease. Two published studies have attempted to determine this cost using the foregone earnings approach. Hardy and coworkers estimated the present value of the foregone earnings of the first 10,000 AIDS cases at $4.6 billion, over three times their estimate of total hospital expenditures (approximately $1.4 billion) for these cases.[1] Scitovsky and Rice estimated the current value of foregone earnings associated with AIDS deaths in 1986 at $6 billion, approximately six times their estimate of personal medical care costs ($1.0 billion).[2]

These estimates may be criticized because of the absence of actual earnings data in estimating the present value of future earnings lost. The foregone earnings method uses national cross-sectional profiles of earnings and labor force participation rates by age and sex.[3] It is simply assumed that a person with AIDS has the same employment and earnings pattern as other Americans in his or her age and sex cohort.

This paper examines this assumption by comparing estimates of the foregone earnings of males who died of AIDS in Texas in 1987 using national earnings profiles and two sources of actual earnings.

Methods

To derive the first estimate, we followed the standard practice of multiplying the present values of lifetime earnings

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by age category, derived from national earnings profiles, by the corresponding number of AIDS deaths in Texas and summing the products across categories. The present value of future earnings by age, reported by Scitovsky and Rice for 1984,[2] were updated using the ratio of mean earnings for full-time workers in 1987[4] as compared to those in 1984.[5] The age characteristics of males who died of AIDS in 1987 were supplied by the Texas Department of Health.

The occupational status (obtained from death certificates) of a 10 percent random sample (N = 96) of males who died of AIDS in 1987 was the basis for the second estimate of mortality costs. The occupations of these persons were translated into average annual earnings using occupation-specific wage data for the Houston and Dallas-Ft. Worth SMSAs from the Bureau of Labor Statistics, US Department of Labor.[6,7]

The third estimate used earnings derived from a statewide survey of living persons with AIDS.* In the summer of 1988, a convenience sample of 1,083 persons with human immunodeficient virus (HIV) infection being served by community-based AIDS service organizations, physicians' offices, and other service providers were asked to complete an anonymous, self-administered questionnaire. The questionnaire requested information on occupation and income at the time of first HIV-related diagnosis. Forty percent (434) of the questionnaires were returned; 280 respondents (six females, 274 males) had an AIDS diagnosis. These individuals represented about 15 percent of all AIDS cases estimated to be alive in Texas at the time of the survey. Of the male respondents with AIDS, 228 (83 percent) supplied income information. Since the survey excluded persons outside the larger metropolitan areas of the state, illiterate and/or poorly educated individuals, and Spanish-speaking persons, it does not represent the state as a whole.

Mean earnings for males with AIDS in different age groups was derived, based on each source of actual earnings. Comparisons were made with national mean earnings reported by the Bureau of Census.[4] Percentage differences for each group were calculated and used to adjust the present value of expected lifetime earnings for the corresponding group. The adjusted earnings figures were then multiplied by the number of AIDS deaths in Texas in 1987 by age category and summed across categories to obtain mortality cost estimates. The cost estimate effect of each data source was subjected to sensitivity analysis to allow for errors in the estimates of actual earnings.

TABLE 1—Comparison of Age, and Occupational Characteristics: Survey Sample, Death Certificate Sample, and Case Registry

Characteristics	Information Source: Survey (N = 228) %	Death Certificates (N = 96) %	Texas AIDS Case Registry, 9/2/88 (N = 4926) %
Age (years)			
20–29	20.7	24.7	25.9
30–39	53.3	51.5	48.2
40–49	21.6	18.6	17.7
50+	4.3	6.2	7.8
Ethnicity			
Black	2.2	No	11.6
White	86.7	Data	77.0
Hispanic	8.8		11.1
Other	1.8		0.3
General Occupation			
Professional/ Technical	37.4	43.3	No Data
Clerical/Sales	23.3	19.6	
Service	27.8	16.5	
Other	11.5	20.6	
Agriculture/Fishing	0.9	1.0	
Processing	0.4	—	
Machine Trades	1.8	—	
Benchwork	1.8	4.1	
Structural	4.8	13.4	
Miscellaneous	1.8	2.1	

TABLE 2—Comparison of Current Earnings: National Profiles, Survey Sample, and Death Certificate Sample[a]

National[b]		Survey			Death Certificate		
Age Group	$	#	$	%Diff.	#	$	%Diff.
20–24[c]	9,009	5	11,100	23.2	5	18,252	103.0
25–29	19,570	43	15,692	−19.8	18	18,694	−4.5
30–34	24,485	70	23,840	−2.6	33	22,393	−8.5
35–39	29,019	52	28,574	−1.5	17	20,891	−28.0
40–44	32,314	31	29,107	−9.9	9	27,716	−14.2
45–49	33,655	18	25,388	−24.6	8	23,289	−30.8
50–54	32,581	6	28,600	−12.2			
55–59	28,422	2	33,250	17.0	4	25,207	−11.3
60–64	26,800	1	20,000	−25.3			
>64	15,460				2	0	−100.0
Total	24,015	228	24,098	0.3	96	21,442	−10.7

[a] Foregone earnings based on national, survey, and death certificate estimates of current earnings: $743.4 million, $697.2 million, $656.4 million. Ten percent variations in the current earnings estimates produced foregone earnings ranging from $627.5 million to $772.4 million.
[b] Source: Reference 4.
[c] The national data include ages 18–24.

Results

Although obtained in different ways, the survey and death certificate samples were not significantly different from the Texas AIDS case registry with respect to age (Table 1). However, Blacks were underrepresented in the survey sample and Whites were overrepresented. There were more individuals with low wage occupations in the survey than in the death certificate sample.

The average annual earnings of male persons with AIDS in Texas, based on the death certificate sample, was 11 percent below the national average (Table 2). However, it was .3 percent above the national average when based on the survey sample.

The present value of lifetime earnings lost for male AIDS deaths in 1987 was $743 million based on national earnings profiles, $697 million based on earnings from the survey, and $656 million based on earnings derived from the death certificate sample. The two estimates using actual earnings were below the national earnings estimate by 6 percent and 12 percent, respectively. Given the possibility of error in the sample estimates of actual earnings, mortality costs were recalculated using earnings of 10 percent above and below the sample estimates for each age group. These variations lead to differences in mortality cost estimates ranging from 4 percent above to 15 percent below the national estimate.

*Crane, MM, Jardine L, Tortolero S, Hines A, Johnson C: Survey of Texans with HIV infection: medical, social service and financial needs. Unpublished report for the Texas Legislative Task Force on AIDS. December 21, 1988.

Discussion

The mean earnings of male Texans with AIDS, based on occupational status, was found to be 12 percent below the national average. However, self-reported earnings from a sample survey averaged less than 1 percent above the national average. It is possible that bias in the survey sample could account for the difference. As noted earlier, it is likely that the survey sampling procedure reduced participation by individuals at the lower end of the income spectrum.

The effect of using the two sources of actual earnings rather than national earnings to estimate future earnings lost was a relatively small reduction. The differences remained relatively small after increasing and decreasing actual earnings estimates by 10 percent. This finding suggests that the additional effort required to obtain actual earnings for persons with AIDS in order to calculate the foregone earnings of premature mortality was not necessary in this case. However, further studies are needed to determine if the findings hold in less biased samples in other areas of the country and for the changing AIDS population.

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Sociodemographic Correlates of Neutralizing Poliovirus and Hepatitis A Virus Antibodies as Markers of Different Modes of Acquiring Immunity

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Abstract: The prevalence and sociodemographic correlates of antibodies against poliovirus and hepatitis A virus (HAV) were compared in a random sample of 457 military recruits in Israel inducted during 1987. Lower socioeconomic status (SES) was associated with a higher prevalence of anti-HAV antibodies (67.3 vs 32.5 percent), whereas the reverse was true for type I poliovirus (78.4 vs 89.5 percent). While the high prevalence of anti-HAV antibodies observed in the lower SES groups reflects considerable natural exposure to enteroviruses, immunity against poliovirus appears to be determined primarily by compliance with vaccination. (*Am J Public Health* 1990; 80:1270–1271.)

Introduction

Between 1961–83, the annual incidence of poliomyelitis in Israel ranged from three to 35 cases.* It was felt that the population should have high levels of immunity through vaccination, but enhanced immunity acquired through continuous exposure to wild virus in the environment was also considered possible. Both the hepatitis A virus (HAV) and the poliovirus are transmitted by the fecal-oral route. Ethnic and educational differences in HAV infection have been demonstrated in Israel[1,2] and elsewhere.[3] We compared the sociodemographic correlates of antibodies against both HAV infection and poliomyelitis as markers of the modes of acquiring immunity to these diseases.

Methods

Sera stored at −20°C which had been taken from a random sample of 494 military conscripts in Israel in 1987 were used for the serological studies.

Sociodemographic data included age, country of origin, education and socioeconomic status (SES). Country of origin was defined by the father's birthplace (or where this was Israel, the paternal grandfather's birthplace). Those from Europe (excluding Turkey), the Americas, Australasia and Southern Africa were defined as Westerners. All other birthplaces, except Israel, were classified as Easterners. To simplify the classification, those whose father and paternal grandfather were born in Israel were excluded from the analyses, leaving a sample size of 457.

The normal approximation was used to compute 95 percent confidence intervals for difference between percentages. Multiple logistic regression analysis was used to determine correlates of antibodies while controlling for other potentially confounding factors.

Hepatitis Serology

The anti-HAV antibody studies were performed at the central Israel Defense Force laboratories by means of solid

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*Israel Ministry of Health, publications on selected infectious diseases 1961–88.

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phase radioimmunoassay (HAVAB-Abbott Laboratories, N. Chicago, IL).

Polio Serology

The neutralizing poliovirus antibody studies were carried out at the Israeli Central Virology Laboratory. The sera were inactivated before testing in a microneutralization system. Serial four-fold dilutions were prepared in microtiter plates and between 30 and 100 TCD_{50} of wild poliovirus (Mahoney--type 1, MEF—type 2, Saukett—type 3) were added. The mixtures were incubated at 36°C for 20 hours, followed by the addition of Hep-2 cells. Following incubation, fixation, and staining, the plates were examined macroscopically and titers were defined as the highest dilution of serum that completely inhibited cytopathic effect. In this study, antibody titers greater than 1:4 were regarded as positive. Since large inter-laboratory differences have been demonstrated,[4] caution should be exercised in comparing these titers with those obtained in other laboratories.

Results

The prevalence of anti-HAV antibodies was substantially lower for Westerners (30.9 percent of 201) than for Easterners (57.0 percent of 256) (95% CI for the difference was 17.0, 35.4). The prevalence of neutralizing poliovirus antibodies was slightly lower for Easterners for all three types: type 1—82.0 percent vs 89.6 percent, type 2—98.1 percent vs 98.5 percent, type 3—83.2 percent vs 86.6 percent. The prevalence of anti-HAV antibodies was substantially higher for subjects of lower SES (67.3 percent of 171 vs 32.5 percent of 286; 95% CI for the difference 25.3, 44.2) whereas the prevalence of neutralizing poliovirus antibodies was greater for higher SES (89.5 percent of 286 versus 78.4 percent of 171; 95% CI for the difference 4.1, 17.5). In multiple logistic regression analysis (Table 1), low SES and Eastern origin were positively associated with the presence of anti-HAV antibodies after adjusting for education and sex. For type 1 poliovirus, high SES was positively associated with higher antibody titers after adjusting for the other variables.

TABLE 1—Multiple Logistic Regression Adjusted Odds Ratios (with 95% confidence intervals) for the Association between the Presence of Anti-Hepatitis A Antibodies and Neutralizing Poliovirus Type 1 Antibody Titers >1:4 with Sociodemographic Variables in 18–19 year-old Jews in Israel Examined in 1987

	Anti-HAV Antibodies	Poliovirus type 1 antibodies
Variable	Odds ratio (95% CI)	Odds ratio (95% CI)
Sex (Male = 0)	1.22 (0.81–1.83)	1.22 (0.71–2.10)
Ethnic origin (West = 0)	2.29 (1.52–3.46)	0.63 (0.35–1.12)
Years of education (<12 yr = 0)	0.83 (0.46–1.50)	1.42 (0.70–2.89)
Socioeconomic status (High = 0)	3.75 (2.31–6.09)	0.40 (0.22–0.74)

CI = Confidence interval

Discussion

The higher prevalence of anti-HAV antibodies in 18–19 year old Jews of Eastern origin compared with those of Western origin, found in earlier studies,[1] has persisted. The prevalence was also found to be independently higher in those of lower SES. This indicates continuing differential exposure of subpopulations in Israel to viruses transmitted by the fecal-oral route, possibly due to spread among children particularly in large families.[2,5]

The associations of SES and ethnic origin with neutralizing poliovirus antibodies were opposite to those found for HAV. This finding would tend to exclude any significant natural exposure to wild poliovirus. The lower prevalence of immunity against polio among those of Eastern origin and lower SES may be due to variation in immunization coverage in different segments of the population[6] or differences in the uptake of oral poliovaccine due to interference by concurrent gastrointestinal infections.

The findings in the present study suggest that subgroups in this population who are more susceptible to HAV infection are less likely to have high levels of neutralizing poliovirus antibody titers. Thus while HAV appears to be widely transmitted under lower socioeconomic conditions, immunity against poliovirus in Israel over the past 20 years appears to have been determined primarily by compliance with vaccination. If the new HAV vaccine currently being tested becomes routine in childhood, immunity will be determined mainly by vaccine compliance.

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